Australian Meningococcal Surveillance Programme annual report, 2012

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Abstract

In 2012, there were 208 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, and 222 cases notified to the National Notifiable Diseases Surveillance System, thus laboratory data were available for 93.7% of cases of IMD in Australia in 2012. Isolates of *Neisseria meningitidis* from 116 invasive cases of meningococcal disease were available for testing, and the phenotype (serogroup, serotype and serosubtype) and/or genotype, and antibiotic susceptibility were determined. Molecular typing was performed for the 92 cases confirmed by nucleic acid amplification testing (NAAT). Typing information was available for 194 of the 208 laboratory confirmed cases and 83% (161 cases) were serogroup B infections, 5.7% (11 cases) were serogroup C infections, 3.6% (11 cases) were serogroup W135, and 7.7% (15 cases) were serogroup Y meningococci. The number of laboratory confirmed IMD cases in 2012 was the lowest since laboratory surveillance data have been reported. Primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents (15–19 years) and young adults (20–24 years), respectively. Serogroup B cases predominated in all age groups and jurisdictions. In 2012, the most common porA genotype circulating in Australia was P1.7-2,4. Serogroup C, W135 and Y cases were numerically low, similar to previous years. Decreased susceptibility to the penicillin group of antibiotics was observed in 81.9% of isolates, and 1 isolate exhibited resistance to penicillin. All isolates remained susceptible to ceftriaxone, ciprofloxacin and rifampicin. *Commun Dis Intell* 2013;37(3):E224–E232.

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

Introduction

The National Neisseria Network (NNN) is a long-standing collaborative association for the laboratory surveillance of the pathogenic *Neisseria* species (*N. 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 for the AMSP. The AMSP data supplement the clinical notification data from the National Notifiable Diseases Surveillance System (NNDSS). The NNN receives samples for analysis from about 90% of IMD cases notified to NNDSS.[2] The AMSP annual reports are published in *Communicable Diseases Intelligence*.[3]

The characteristics of the meningococci responsible for IMD are important both for individual patient management, contact 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) has seen 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 *N. meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in Australia in 2012.

Methods

Case confirmation of invasive meningococcal disease cases

Case confirmation was based upon isolation of, or positive nucleic acid amplification testing (NAAT) for, *N. meningitidis* from a normally sterile site and defined as IMD according to Public Health Laboratory Network criteria.[4] Information regarding the site of infection, the age and sex of the patient, and the outcome of the infection (survived/died) was collated.

Cases were categorised on the basis of the site from which *N. meningitidis* was isolated or from which meningococcal DNA was detected. When *N. meningitidis* was grown from both blood and cerebrospinal fluid (CSF) cultures from the same patient, the case was classified as one of meningitis. It is recognised that the total number of IMD cases, and particularly the number of cases of meningitis,

may be underestimated if a lumbar puncture was not performed or was delayed and the culture was sterile. However, the above approach has been used since the beginning of this program and is continued for comparative purposes. Where the diagnosis is made by serology, it is not possible to definitively classify a case as meningitis or septicaemia.

Phenotyping and genotyping of *Neisseria meningitidis*

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. Genotyping of isolates and DNA extracts from NAAT diagnosis is performed by sequencing of products derived from amplification of the porin genes *porA, porB* and *FetA*.

Antibiotic susceptibility testing

Antibiotic susceptibility was assessed by determining the minimum 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 or resistance when determined by a standardised agar plate dilution technique.[5]

Sensitive: MIC ≤ 0.03 mg/L
Less sensitive: MIC 0.06–0.5 mg/L
Resistant: MIC ≥ 1 mg/L

Meningococcal serology

Laboratory diagnosis of suspected cases of IMD can be made serologically based on the demonstration of IgM antibody by enzyme immunoassay to *N. meningitidis* outer membrane protein using the methods and test criteria of the Health Protection Agency, United Kingdom, as assessed for Australian conditions.[6–8]

Results

Aggregated data on laboratory confirmed invasive meningococcal disease cases

In 2012, there were 208 laboratory-confirmed cases of IMD analysed by the National Neisseria Network, and 222 cases notified to the NNDSS, thus laboratory data were available for 93.7% of cases of IMD in Australia in 2012 (Table 1). This was the lowest annual total of IMD cases recorded by the NNDSS and the AMSP since surveillance data was collated (Figure 1).

In 2012, a positive culture was obtained for 116 of 208 (56%) cases of which 92 (44%) cases were confirmed by NAAT testing alone. There were no IMD cases diagnosed serologically in 2012.

The highest number of laboratory-confirmed cases was from New South Wales (62 cases), slightly lower than the 67 cases in 2011. Victoria had 33 cases, markedly lower than the 53 cases in 2011. Numbers for the other states were similar to 2011 (Table 1).

Seasonality and age distribution

As in previous years, the peak incidence for IMD continues to be late winter and early spring (1 July to 30 September) (Table 2).

Nationally, the peak incidence of IMD was in children aged less than 5 years, which was similar to previous years. Between 2007 and 2011, 28% to 36% of cases were in this age group. In 2012, 62 of

Table 1: Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012, by serogroup and state or territory

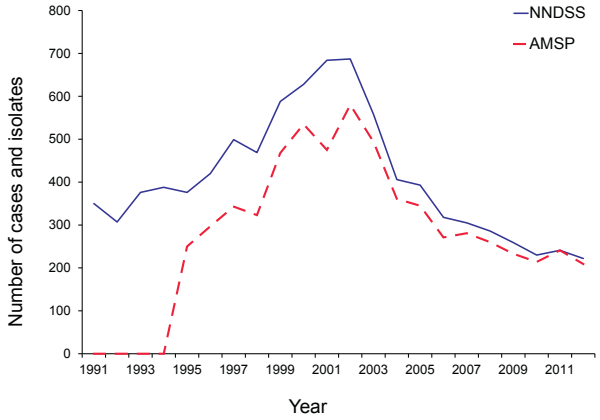
State or territory	Serogroup						Total
	B	C	Y	W135	NG	ND	
ACT	1	0	0	0	0	0	1
NSW	43	2	5	4	7	1	62
NT	2	1	0	0	0	1	4
Qld	45	3	4	3	0	4	59
SA	23	1	0	0	0	0	24
Tas	4	1	1	0	0	1	7
Vic	28	1	4	0	0	3	33
WA	15	2	1	0	0	0	18
Australia	161	11	15	7	7	7	208

NG Non-groupable.

ND Non-determined (samples were examined by nucleic acid amplification test).

208 (30%) IMD cases occurred in this age group, as shown in Table 3. A secondary disease peak has also been observed in previous years amongst adolescents and young adults aged 15 to 24 years. Of all cases 13.5% (28 confirmed cases) in those aged 15 to 19 years in 2012 was lower than the number reported for the years 2007 to 2011 (between 17% and 20%). There were 26 cases of IMD (12.5%) in the 20 to 24 years age group, which was similar to 2011, but lower than the 22% to 31% reported in this age group in the years 2007 to 2010.

Figure 1: Number of invasive meningococcal disease cases reported to the NNDSS compared with laboratory confirmed data from the AMSP, Australia, 2012



Serogroup data

The serogroup was determined for 194 of the 208 laboratory-confirmed cases of IMD in 2012 (Table 1). Of these, 161 (83%) were serogroup B and 11 (5.7%) were serogroup C. The proportion of cases that were serogroup B was similar to the proportion reported between 2006 and 2011 (between 84% and 88%). The number and proportion of cases of serogroup C in 2012 was slightly higher than in 2011 (9 cases; 3.7%). In 2012 there were 7 (3.6%) cases of serogroup W135, which was less than the previous year (11 cases; 5.2%). There were 15 (7.7%) cases of serogroup Y, a slightly higher proportion than in 2011 (6.2%) and higher than the proportions in 2009 and 2010 (3.5% and 3.9% respectively). With the continuing low number of serogroup C infections, serogroup B meningococci predominated in all age groups and jurisdictional differences in serogroup distribution were not evident.

In 2012, total IMD cases, the number of cases due to serogroup B, and the proportion of serogroup B cases

Table 2: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012, by quarter

Serogroup	Qtr 1	Qtr 2	Qtr 3	Qtr 4	Total 2012
B	24	47	56	34	161
C	3	1	3	4	11
Y	1	3	9	2	15
W135	0	1	4	2	7
NG/ND	3	3	8	0	14
Total	31	55	80	42	208

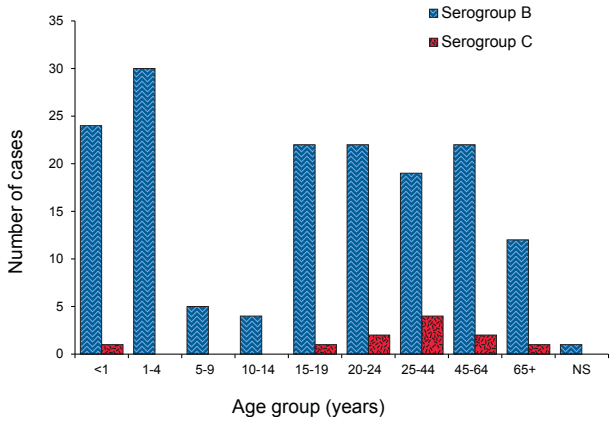
Table 3: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012, by age and serogroup

	Age group										
Serogroup	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	Total
B	24	30	6	4	22	22	19	21	12	1	161
C	1	0	0	0	1	2	4	2	1	0	11
Y	1	0	0	0	1	2	4	3	4	0	15
W135	0	2	0	0	2	0	0	2	1	0	7
NG/ND	1	3	4	3	2	0	0	1	0	0	14
Total	27	35	10	7	28	26	27	29	18	1	208
% of B within age group	88. 9	85.7	60.0	57.1	78.6	84.6	70.4	72.4	66.7	100.0	

NS Age not stated
NG Non-groupable
ND Non-determined (samples were examined by nucleic acid amplification test).

from the total was lower in each of the age categories less than 20 years (Table 2, Figure 2). The proportion of serogroup B cases in the 20 to 24 years age group (84.6%) was higher than the previous year (61%) but similar to 2007 to 2010 (between 80% and 88%). In people aged 25 years or over, there was a modest increase in the proportion of serogroup B cases from 2011. This may in part be explained by an increase in the number of serological IMD diagnoses (and thus serogroup not determined) in this age category for 2011. The peak number of serogroup C cases occurred in the 25 to 44 years age category, as reported for 2011. There were 2 serogroup C cases in those aged less than 20 years in 2012, but no cases in 2011.

Figure 2: Number of serogroup B and C cases of confirmed invasive meningococcal disease, Australia, 2012, by age



NS Not serotyped

Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2012, the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype, were analysed for New South Wales (Table 4). Serogroup B meningococci are in general more difficult to characterise by serological methods and a number could not be phenotyped. All 35 New South Wales IMD isolates were phenotyped, the most common being B:4:P1.4 followed by B:15:P1.7.

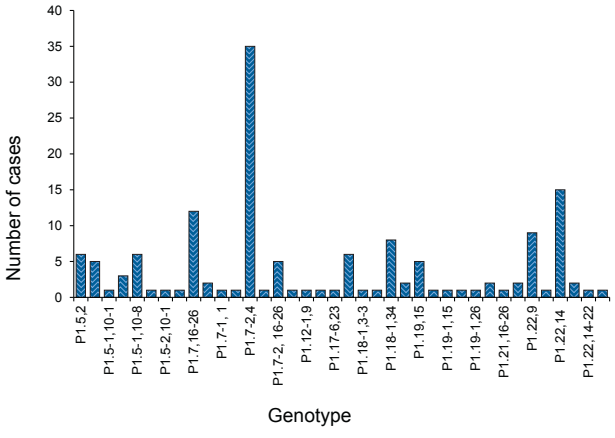
Genotyping data of invasive meningococcal samples (culture or nucleic acid amplification test products)

Sequencing products derived from amplification of the variable region *porA, porB* and *FetA* genes is used in an increasing number of jurisdictions in place of serotyping using monoclonal antibodies. In 2012, genotyping data were available from all states and territories for 146 of 208 (70%) IMD cases (Table 5). The predominant *porA* genotypes for serogroup B isolates were again P1.7-2,4 (35 cases, compared with 21 in 2011), P1.22,14 (15 cases, compared with 7 in 2011) and P1.7,16-26 (12 cases, compared with 19 in 2011 (Table 6 and Figure 3).

Table 4: Laboratory confirmed cases of invasive meningococcal disease, New South Wales and the Australian Capital Territory, 2012, by phenotype

Serotype	Subtype	Serogroup	Total
4	P1.4	B	5
NT	P1.5	Y	3
15	P1.7	B	3
1	P1.14	B	3
NT	NST	B	2
4	P1.14	B	2
15	NST	B	2
NT	P1.4	B	2
1	P1.4	B	1
NT	P1.5,2	B	1
15	P1.7,1	B	1
4	P1.9	B	1
NT	P1.9	B	1
4	NST	B	1
2a	P1.2	W135	1
NT	P1.3	W135	1
NT	P1.5	B	1
NT	P1.6,3	W135	1
NT	P1.7	Y	1
NT	P1.16	W135	1
NT	NST	ND	1

Figure 3: Number of *porA*-genotypes* for serogroup B in cases of invasive meningococcal disease, Australia, 2012



* Where genotype data available.

The predominant *porA* genotype for serogroup C isolates was P1.5-1,10-8 (6 cases, compared with 4 in 2011). The AMSP was not aware of any epidemiological link between any of the cases reported where genotyping was available.

Outcome data for invasive meningococcal disease for laboratory-confirmed cases

For 69% of IMD cases (143/208), outcome data (survived or died) were available from the referring laboratories (Table 7). Nine deaths were recorded amongst the 143 cases for whom outcome data were available. Eight of these deaths were attributable to serogroup B infections, and one to serogroup C infection.

Anatomical source of samples for laboratory confirmed cases

There were 69 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone or with a positive blood sample. There were 133 diagnoses of septicaemia based on cultures or NAAT examination from blood samples alone (Table 8). There were no IMD cases diagnosed by serology in 2012. Sites other than blood, CSF or serum from which diagnoses were made were tissue (1 by polymerase chain reaction), and joint fluid (5 by culture).

Antibiotic susceptibility testing of invasive meningococcal isolates

Penicillins

Susceptibility to penicillin and other antibiotics was determined for 116 of 208 (56%) cases in 2012. Using defined criteria, 95 (82%) isolates were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L; and 19 (16%) isolates were fully sensitive (MIC 0.03 mg/L or less). One isolate was resistant (MIC = 1.0 mg/L). The proportion of less sensitive strains was lower than in 2011 (86.4%) but higher than that reported in 2007 to 2010 (range 67% to 80%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone and by extrapolation to other third generation cephalosporins. All isolates were fully susceptible to ciprofloxacin. There were 2 isolates with altered susceptibility to rifampicin (MIC = 0.5 mg/L).

Table 5: Laboratory confirmed cases of invasive meningococcal disease, Australia (excluding New South Wales and the Australian Capital Territory), 2012, by *porA* genotype

Genotype *porA*	B	C	W135	Y	Total
P1.7-2,4	26	0	0	0	26
P1.22,14	9	1	0	1	11
P1.7,16-26	8	0	0	0	8
P1.22,9	7	0	0	0	7
P1.5-1,10-8	0	6	0	0	6
P1.18-1,34	6	0	0	0	6
P1.5,2	1	0	2	2	5
P1.7-2,16-26	5	0	0	0	5
P1.19,15	5	0	0	0	5
P1.5-1,2-2	3	0	0	1	4
P1.18-1,3	2	0	0	2	4
P1.19-3,15	1	1	0	0	2
P1.22,14-6	2	0	0	0	2
P1.5-1,10-1	0	0	0	1	1
P1.5-1,10-4	0	0	0	1	1
P1.5-2,10-1	0	0	0	1	1
P1.5-8,2-48	1	0	0	0	1
P1.7,30	1	0	0	0	1
P1.7-1,1	1	0	0	0	1
P1.7-1,13-1	0	0	0	1	1
P1.7-2,13-1	1	0	0	0	1
P1.7-11,16-26	1	0	0	0	1
P1.12-1,9	1	0	0	0	1
P1.17-6,23	1	0	0	0	1
P1.18-1,3-3	1	0	0	0	1
P1.18-7,9	1	0	0	0	1
P1.19-1,10-8	1	0	0	0	1
P1.19-1,15	1	0	0	0	1
P1.21,16-26	1	0	0	0	1
P1.22,10-8	1	0	0	0	1
P1.22-1,14	1	0	0	0	1
Total	89	8	2	10	109

Discussion

In 2012 there were 208 IMD cases laboratory-confirmed by the NNN, representing 93.7% of notifications to the NNDSS.[2] This was the lowest number of confirmed IMD cases and notifications since surveillance data began in 1991. It was also one-third of the number of confirmed cases and notifications of IMD in Australia in 2002 (580 confirmed IMD cases of 687 notifications) when the number of cases of IMD peaked. A primary peak in IMD infection rates is evident in children aged less than 5 years, as reported in previous years, with a secondary peak in adolescents and young adults.

The proportion of cases with serogroup B IMD cases is essentially the same as that reported between 2006 and 2011. The proportion of cases

Table 6: Laboratory-confirmed cases of invasive meningococcal disease, Australia (excluding New South Wales and the Australian Capital Territory), 2012, by state or territory

	NT		Qld		SA		Tas		Vic		WA	
Genotype *porA*	Serogroup	n	Serogroup	n	Serogroup	n	Serogroup	n	Serogroup	n	Serogroup	n
P1.7-2,4			B	14	B	12						
P1.22,14			B	1	C	1	B	1	B	5	B	2
P1.22,14			Y	1								
P1.7,16-26			B	4	B	1	B	1	B	2		
P1.22,9			B	1					B	6		
P1.5-1,10-8	C	1	C	3					C	1	C	1
P1.18-1,34			B	2					B	3	B	1
P1.5,2			W135	2					B	1	B	1
P1.5,2									Y	1		
P1.7-2,16-26			B	4							B	1
P1.19,15			B	2					B	3		
P1.5-1,2-2			B	1					B	2	Y	1
P1.18-1,3			B	1			Y	1	B	1		
P1.18-1,3									Y	1		
P1.19-3,15							B	1			C	1
P1.22,14-6			B	1							B	1
P1.5-1,10-1			Y	1								
P1.5-1,10-4									Y	1		
P1.5-2,10-1			Y	1								
P1.5-8,2-48									B	1		
P1.7, 30											B	1
P1.7-1,1					B	1						
P1.7-1,13-1											Y	1
P1.7-2,13-1									B	1		
P1.7-11,16-26											B	1
P1.12-1,9			B	1								
P1.17-6,23			B	1								
P1.18-1,3-3									B	1		
P1.18-7,9			B	1								
P1.19-1,10-8			B	1								
P1.19-1,15			B	1								
P1.21,16-26			B	1								
P1.22,10-8									B	1		
P1.22-1,14			B	1								

Table 7: Outcome data of infection for laboratory confirmed cases of invasive meningococcal disease, Australia, 2012, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	35	0	2	0	2	39
	Died	3	0	0	0	0	3
	Unknown	20	1	2	0	5	28
	Total	58	1	4	0	7	70
Septicaemia	Survived	72	5	8	3	2	90
	Died	5	1	0	0	0	6
	Unknown	24	3	2	3	5	37
	Total	101	9	10	6	7	133
Other	Survived	2	1	1	1	0	5
	Died	0	0	0	0	0	0
	Unknown	0	0	0	0	0	0
	Total	2	1	1	1	0	5
All cases	Survived	109	6	11	4	4	134
	Died	8	1	0	0	0	9
	Unknown	44	4	4	3	10	65
	Total	161	11	15	7	14	208

NG Serogroup not groupable or not determined.

Table 8: Anatomical source of samples positive for laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012

Specimen type	Isolate of meningococci	PCR positive*	Serology alone†	Total
Blood	93	40	0	133
CSF +/– blood	18	51	0	69
Other‡	5	1	0	6
Total	116	92	0	208

* Nucleic acid amplification test (NAAT) positive in the absence of a positive culture.

† Serology positive in the absence of positive culture or NAAT.

‡ Joint fluid (n=5), tissue (n=1).

PCR Polymerase chain reaction.

with serogroup C cases continues to be low across all age groups, following the decline as a result of the introduction of the serogroup C vaccine in 2003. As in previous years, there were only a small number of serogroup C cases in those aged 25 years or over, 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.[9] Low numbers of infections with serogroups Y and W135 is usual for Australia, however there was a proportional increase in serogroup Y disease in 2011 to 2012 compared with previous years. This will continue to be monitored to determine whether it is the beginning of an increasing trend.

As in previous years, phenotypic and genotypic data found no evidence of substantial numbers of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination. There have been concerns that the emergence of new and invasive subtypes following extensive vaccine use would occur given the capacity for genetic reconfiguration within meningococci.[9] Monitoring of meningococcal genotypes will continue as part of the NNN program.

Outcome data were assessable for 69% of the cases reported by laboratories, and thus should be interpreted with caution. Eight of the 9 fatal cases of IMD were associated with serogroup B infection

and one with serogroup C. The NNN does not attempt active collection of morbidity data associated with IMD.

The proportion of IMD isolates with penicillin MICs in the less sensitive category (0.06–0.5 mg/L) for 2012 was 82%. This was lower than that reported in 2011, but higher than in previous years indicating a continuing shift in penicillin MICs of IMD isolates from sensitive to less sensitive category. All isolates were susceptible to the third generation cephalosporins and ciprofloxacin. Strains with decreased susceptibility to quinolone antibiotics have been the subject of ongoing international interest following their first description by the AMSP in 2000.[10–13] There were 2 isolates with altered susceptibility to rifampicin from Queensland.

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