Annual reports

Annual report of the Australian Meningococcal Surveillance Programme, 2008

The Australian Meningococcal Surveillance Programme

Abstract

In 2008, there were 260 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, a nationwide network of reference laboratories. One hundred and forty-nine isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were available for which the phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility were determined. An additional 111 cases were confirmed by non-culture based methods. Nationally, 223 (85%) laboratory-confirmed cases where a serogroup was determined were infected with serogroup B and 17 (6.5%) infected with serogroup C meningococci. Nationally, the total number of confirmed cases has remained relatively stable since 2006, but the number of cases in each jurisdiction may vary from year to year. Queensland had the highest number of recorded cases in 2008. 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 were again B:15:P1.7 and B:4:P1.4. Although serogroup C cases were numerically low, phenotype C:2a:P1.5 predominated in this group. No evidence of meningococcal capsular 'switching' was detected. About three-quarters of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06–0.5 mg/L). All isolates remained susceptible to ceftriaxone. One isolate had reduced susceptibility to rifampicin and two to ciprofloxacin. *Commun Dis Intell* 2009;33(3):259–267.

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, *Neisseria meningitidis* and *N. gonorrhoeae*. NNN has operated since 1994 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 national data on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility, in annual reports published in *Communicable Diseases Intelligence*.[2] These data supplement those from clinical notification schemes.

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. Despite the significant reduction in the number of cases of IMD since 2004 when a publicly-funded program of selective vaccination with conjugate serogroup C meningococcal vaccine was completed, IMD remains an issue of public health concern in Australia. The success of 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 2008.

Methods

Isolate based invasive meningococcal disease cases

Case confirmation was based upon isolation of a meningococcus from a normally sterile site or demonstrated 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 of the infection (survived/died) 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 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* has been used to supplement and supplant serotyping analyses based on the use of monoclonal antibodies. For the purposes of continuity and comparability, the typing data from both approaches have been approximated in the accompanying tables by converting sequence data to the more familiar serotyping/serosubtyping nomenclature.

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 MICs 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 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 260 laboratory confirmed cases of IMD in 2008 (Table 1) compared with 281 in 2007, 271 in 2006, 345 in 2005 and 361 in 2004. In 149 cases (57%), a positive culture was obtained with or without a positive non-culture based test and 111 (43%) cases were confirmed by a non-culture based method alone. The total number of all laboratory confirmed cases increased in Queensland from 75 in 2007 to 83 in 2008 and this jurisdiction had the highest number of laboratory confirmed cases. In New South Wales, numbers

Table 1: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2008, by serogroup and state or territory

State or territory	Serogroup					Total
	B	C	Y	W135	NG*	
ACT	4	1	0	0	0	5
NSW	46	6	4	5	1	62
NT	4	2	0	0	0	6
Qld	73	4	1	2	3	83
SA	18	0	0	1	0	19
Tas	1	0	0	0	0	1
Vic	51	2	2	0	6	61
WA	22	0	0	0	1	23
Australia	219	15	7	8	11	260

* Not serogrouped.

detected decreased to 62 from 101 in 2007. There were 61 cases in Victoria, which was little changed from the 59 cases in 2007. Small or no numerical differences were noted in other jurisdictions.

Seasonality

Thirty-six cases occurred between 1 January and 31 March, 61 between 1 April and 30 June, 98 between 1 July and 30 September and 65 between 1 October and 31 December. A winter peak of meningococcal disease is more usual, but the above pattern was also present in 2007.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those aged 4 years or under (Table 2). Those aged less than one year or in the 1–4 year age group, together accounted for 94 cases (36.1% of the total) in 2008. There were 100 cases confirmed in these age groups (35.5%) in 2007. A secondary disease peak is also usual in the adolescent or young adult age group. The total of 50 cases (19.2% of all confirmed cases) in those aged 15–19 years was a little less than the 56 cases (19.9%) in this age group in 2007. Those aged 15–24 years accounted for 71 cases (27.2%) in 2008 and 87 cases (31%) in 2007.

Table 2: All laboratory confirmed cases of invasive meningococcal disease, Australia, 2008, by age, state or territory and B and C serogroups

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	1	0	0	0	0	0	0	0	4
	C	0	0	0	0	0	0	1	0	0	0	1
	Total	2	1	1	0	0	0	1	0	0	0	5
NSW	B	8	8	1	2	10	2	8	6	0	1	46
	C	0	1	0	0	0	1	0	3	1	0	6
	Total	10	10	1	2	13	4	9	9	3	1	62
NT	B	1	1	0	0	0	0	1	0	0	1	4
	C	0	0	0	0	1	0	1	0	0	0	2
	Total	1	1	0	0	1	0	2	0	0	1	6
Qld	B	20	13	3	7	13	3	5	7	2	0	73
	C	1	1	0	0	0	1	0	1	0	0	4
	Total	23	15	3	8	14	5	5	8	2	0	83
SA	B	0	3	0	0	5	4	5	1	0	0	18
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	4	0	0	5	4	5	1	0	0	19
Tas	B	0	0	1	0	0	0	0	0	0	0	1
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	0	1	0	0	0	0	0	0	0	1
Vic	B	10	6	3	5	12	2	7	5	1	0	51
	C	1	0	0	0	0	0	0	0	1	0	2
	Total	11	6	3	5	15	4	7	8	2	0	61
WA	B	6	3	0	0	2	4	3	3	1	0	22
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	6	4	0	0	2	4	3	3	1	0	23
Australia	B	47	35	9	14	42	15	29	22	4	2	219
	C	2	2	0	0	1	2	2	4	2	0	15
	Total B+C	49	37	9	14	43	17	31	26	6	2	234
	other	4	4	0	1	7	4	1	3	2	0	26
	Total	53	41	9	15	50	21	32	29	8	2	260
	% of all	20.4	15.7	3.5	5.8	19.2	8.1	12.3	11.1	3.1	0.8	

NS Not stated.

Totals include cases due to other serogroups (15) and cases where the serogroup was not determined (11).

Serogroup data

The serogroup of the meningococci causing disease was determined in 249 of the 260 laboratory confirmed cases of IMD. Of these 249 cases where a serogroup was determined, 219 (88%) were serogroup B and 15 (6%) serogroup C. In 2007, 223 (85%) were serogroup B and 17 (6.5%) serogroup C. In 2008, an additional 8 cases (3.2%) were of W135 and 7 cases (2.8%) were of serogroup Y. With the continuing decline in the number of serogroup C infections, serogroup B meningococci predominated in all age groups (Figure) and jurisdictional differences in serogroup distribution were not evident. The 15 serogroup C cases of IMD were distributed in 5 jurisdictions: New South Wales (6), Queensland (4), Victoria and the Northern Territory (2 each) with a single case in the Australian Capital Territory. Eight of the 15 cases of serogroup C disease in 2008 were in those aged 25 years or more, 4 cases were reported in those aged 4 years or less, a single case in those aged 15–19 years and a further two 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 2008. In those aged 14 years or less, there was a decrease in total case numbers and in serogroup B cases in 2007, but there was no further change noted in case numbers in these age groups in 2008. Serogroup C case numbers were always low in these age groups. In those aged 15–19 years and 20–24 years, the number of serogroup B cases has remained relatively unaltered, but the proportion of serogroup B cases increased as serogroup C cases declined. Again, the relative proportion of serogroup B and C IMD cases was unaltered in 2008 from that observed in 2007. In older (25 years or more) age groups there was a further increase in the number and proportion of serogroup B cases in 2008 whereas the number of serogroup C cases in these age groups was unaltered.

Figure: Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2008, by age

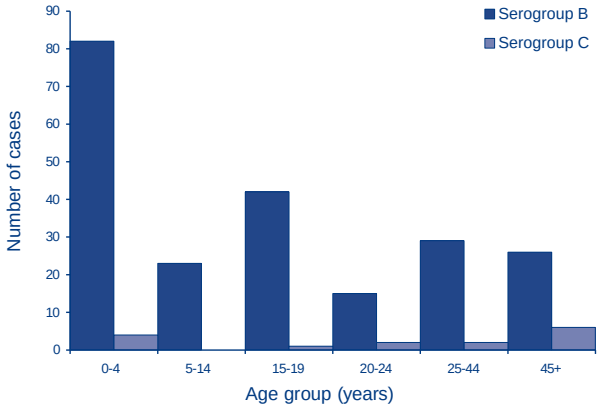

Table 3: A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2008, by known age

Year	Serogroup	Age									
		< 4 years		5-14 years		15 –19 years		20-24 years		25+ years	
		n	%	n	%	n	%	n	%	n	%
2008	B	82	89.1	23	95.8	42	91.3	15	83.3	57	81.4
	C	4	4.4	0	0	1	2.2	2	11.1	8	11.4
	All*	92		24		46		18		67	
2007	B	83	90	19	83	48	91	24	80	49	75
	C	4	4	0	0	2	4	3	10	8	12
	All	92		23		53		30		65	
2006	B	93	93	21	84	40	82	21	70	38	61.3
	C	2	2	3	12	4	8.2	7	23	10	16.1
	All	100		25		49		30		62	
2005	B	99	90	38	75	39	81	22	67	51	50
	C	6	5.5	5	10	4	8	8	24	27	27
	All	110		51		48		33		101	
2004	B	97	88	27	77	40	65	20	57	59	50
	C	6	5.5	5	14	17	28	11	31	32	27
	All	110		35		61		35		117	

* All cases where a serogroup was determined.

Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2008, the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype were analysed and again showed this diversity. The predominant serotypes/serosubtypes in each state and territory are shown in Table 4. Serogroup B meningococci are in general also more difficult to characterise by serological methods and a number could not be phenotyped. A total of 20 isolates were of serotype 4 and nine of these were from New South Wales, five from Victoria and three from Queensland with

Table 4: Common serotypes and sero-subtypes of isolates from culture positive cases of Neisseria meningitidis infection, 2008, by state or territory

State or territory	Serogroup B				Serogroup C			
	serotype	n	serosubtype	n	serotype	n	serosubtype	n
ACT	4	1	1.14	1	2a	1	1.14	1
	15	1	1.7	1				
NSW	4	9	1.15	2	2a	4	1.5	2
			1.4	2			NST	2
			1.6,3	1	NT	2	1.3	1
			nst	4			NST	1
	15	6	1.7	5				
			nst	1				
	14	2	1.4	1				
			1.5,2	1				
	1	2	1.14	1				
			1.6,1.3	1				
	nt	6	nst	2				
			Diverse:1.3/.4/.5/.9	1 ea				
NT	4	1	1.4					
	nt	3	nst	3				
Qld	15	5	1.7	5	2a	2	1.5	1
	1	4	1.14	2			NST	1
			nst	2	15	1	1.9	1
	4	3	1.4	2				
			nst	1				
	14	2	1.4	1				
			nst	1				
	nt	23	1.4	7				
			Diverse:1.13/.14/.15/5	1 ea				
			nst	12				
Tas								
Vic	7	7	1.19	3	2a	2	1.4	2
			1.5	3				
			1.17,9	1				
	15	5	1.7	4				
			1.4	1				
	19.1	6	1.18	3				
			1.22,14	1				
	4,7	5	1.4	3				
			1.5	1				
			1.18	1				
WA	1	4	nst	2				
			1.14/1.6	1 ea				
	14	2	nst	2				
	15	2	1.7	1				
	4	1	1.4	1				
	nt	6	nst	4				
			1.14/1.4	1 ea				

nt Not sero-typable.

nst Not sero-subtypable.

single isolates from the Australian Capital Territory, the Northern Territory and Western Australia. Eight of these 20 were of serosubtype P1.4, which has been circulating in New Zealand at high rates for many years. Another 19 serogroup B isolates were of serotype 15, and 16 of these were of sero-subtype 1.7, which has been circulating in Australia for many years.

The 12 serogroup C strains that were phenotyped were predominantly of serotype 2a (9 strains) and this phenotype has predominated in serogroup C meningococci in Australia for many years. Two strains could not be serotyped and one was of serotype 15, usually found within serogroup B meningococci. 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 previously. For example, in 2003 there were 29 serogroup C isolates of this serotype/serosubtype detected nationally, 21 in 2004, and eight in 2005. Only a 2 isolates with this phenotype were seen in 2008, both in Victoria. Three of the serogroup C:2a isolates were of sero-subtype 1.5 and one of 1.14.

Outcome data for invasive meningococcal disease for laboratory confirmed cases

Outcome data (survived or died) were available for 76 (29%) of the 260 laboratory confirmed cases (Table 5). Four deaths were recorded in this group (5.2%), all with serogroup B infection for which outcomes were available for 64 of 219 cases. Three of the cases were attributable to septicaemia and the fourth to meningitis. No deaths were recorded in 12 infections caused by other serogroups. A single death in 25 patients with meningitic IMD and 3 deaths in 51 bacteraemic patients were recorded.

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, while those shown as PCR positive were culture negative with or without positive serology. There were 85 diagnoses of meningitis based on cultures or PCR examination of CSF either alone or with a positive blood sample (including 2 PCR based diagnoses on post-mortem brain samples and 170 from blood samples (cultures or PCR) alone. There were three other isolates from synovial fluid and in 2 cases the

Table 6: Anatomical source of samples positive for a laboratory confirmed case of invasive meningococcal disease, Australia, 2008

Specimen type	Isolate of MC	PCR positive*	Total
Blood	116	54	170
CSF +/– blood	30	55‡	85
Other†	3	2	5
Total	149	111	260

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

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

‡ 2 diagnosed by PCR of brain tissue.

Table 5: Outcome data (survived, died) for laboratory confirmed cases of invasive meningococcal disease, 2008, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	21	0	2	1	0	24
	Died*	1	0	0	0	0	1
	Total	22	0	2	1	0	25
Septicaemia	Survived	39	2	1	1	5	48
	Died	3	0	0	0	0	3
	Total	42	2	1	1	5	51
All cases	Survived	60	2	3	2	5	72
	Died	4	0	0	0	0	4
	Total	64	2	3	2	5	76

* Clinical sample from post-mortem brain tissue.

NG Not groupable.

source of the clinical sample was not disclosed. No cases that were serologically positive were culture and PCR negative.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and forty-nine isolates were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 108 isolates (72%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and the remainder (21%) fully sensitive (MIC 0.03 mg/L or less). The proportion of less sensitive strains was slightly less than that reported in 2007 (79%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) A single isolate had altered susceptibility to rifampicin and two to ciprofloxacin (MIC, 0.25 mg/L). All three were reported from Queensland.

Discussion

The total number of laboratory-confirmed cases of IMD nationally has remained 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 Queensland recording the highest number of cases in 2008 (83) with a reduction in numbers from New South Wales. These changes in case distribution were essentially attributable to altered numbers of serogroup B cases in 2008 and little change was detected in serogroup C numbers. Cultures were obtained from sterile sites in 149 cases, the lowest number of isolates detected over the duration of the program that commenced in 1994, and a further slight decline from the 154 cases seen in 2007 and the 166 cases from which isolates were obtained in 2006. Non-culture based diagnoses were used to confirm a further 111 (43%) cases as IMD (127 [45%] in 2007). Attention is specifically drawn to earlier AMSP reports that explain differences between the number of clinically notified cases and laboratory confirmed cases.[10] It should also be remembered that surveillance systems rarely capture all cases in any given period so that small differences in numbers of cases should be expected.

Only 15 serogroup C infections were identified nationally in 2008 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, Western Australia or Tasmania with only small numbers present in the other jurisdictions. 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 again evident in younger age groups with a secondary peak in adolescents and young adults. In contrast to data from the earlier years of this program, serogroup C disease was again infrequently encountered in the latter age group in 2008. The reduced and low number of serogroup C cases in those aged 25 years or more (Table 3) was also maintained, and may be attributable to 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 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 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 NNN of penicillin MICs in invasive isolates showed that the proportion with decreased susceptibility to penicillins was 72%, a little less than 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 a small number of isolates from Queensland with decreased susceptibility to rifampicin (1) and ciprofloxacin (2). The latter 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] A single isolate with decreased susceptibility to quinolone antibiotics was detected in 2007 in AMSP data.

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