Annual report of the Australian Meningococcal Surveillance Programme, 2004

The Australian Meningococcal Surveillance Programme

Abstract

This report by the National Neisseria Network, a nationwide collaborative laboratory programme, describes 361 laboratory-confirmed cases of meningococcal disease diagnosed in Australia in 2004. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 245 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 116 cases were confirmed by non-culture based methods. Nationally, the majority of cases were serogroup B (243 isolates, 68%) or serogroup C (71 isolates, 20%) meningococci. The total number of cases was 133 fewer than the 494 cases identified in 2003 and the number of confirmed cases decreased in all jurisdictions except Western Australia where the total was unchanged. There was a 15 per cent decrease in serogroup B infections, but a greater (45%), fall in the number of serogroup C cases. The age distribution of meningococcal disease showed a typical primary peak in those aged four years or less with a secondary peak in adolescents and young adults. Serogroup B cases were 88 per cent of all cases in those aged four years or less and 63 per cent in those aged 15–24 years age range. The proportion of all invasive disease represented by serogroup C disease was highest in the 15–24 years and older age groups. The common phenotypes circulating in Australia were B:4:P1.4 and C:2a:P1.4. However, significant jurisdictional differences in the serogroup and phenotypic distribution of meningococci was again evident and considerable heterogeneity of subtypes was noted. No evidence of sustained transmission of meningococci undergoing capsular 'switching' or genetic recombination was detected. About two thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). A single isolate was penicillin resistant at 1 mg/L. *Commun Dis Intell* 2005;29:149–158.

Keywords: disease surveillance; Neisseria meningitidis

Introduction

The National Neisseria Network (NNN) is a collaborative national programme of reference laboratories in each state and territory of Australia. It examines those aspects of microbiological laboratory medicine relevant to the public health control of invasive meningococcal disease (IMD), namely, diagnosis, antimicrobial resistance surveillance and organism typing, including both isolate-based and non-culture derived methodologies. The first reports from the Meningococcal Surveillance Programme, which began in 1994, relied solely on data derived from examination of isolates from culture-positive cases of IMD, in particular, their phenotype and antibiotic susceptibility. Increasingly, data have been derived from non-culture based methods, notably the genotype and diagnoses based on nucleic acid amplifications assays (NAA), and have been included in reports. The information is provided to supplement that from clinical notification schemes.

In 2003, a publicly funded program of vaccination of children and adolescents with serogroup C conjugate vaccine was commenced and generally was fully operational in 2004. This report analyses information gathered by the NNN on laboratory-confirmed cases of IMD in the calendar year 2004. The format departs from previous annual reports published in *Communicable Diseases Intelligence*[1–10] insofar as aggregated data on all laboratory-confirmed cases are now analysed together.

Methods

The NNN is a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria, *Neisseria meningitidis* and *Neisseria gonorrhoeae*.[1–11] A network of reference laboratories in each state and territory (see acknowledgements) performs and gathers laboratory data on cases of IMD throughout Australia.

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Isolate based invasive meningococcal disease cases

Each case confirmation was based upon isolation of a meningococcus from a normally sterile site and defined as IMD according to the Public Health Laboratory Network definitions. 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 categorises cases on the basis of site of isolation of the organism. Where an isolate is grown from both blood and cerebrospinal fluids (CSF) cultures in the same patient, the case is classified as one of meningitis. It is recognised that the 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 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 has been unified 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:[11]

sensitive	MIC ≤ 0.03 mg/L;
sess 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 including NAA and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques[12] and has been progressively introduced in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 annual report. The serological results are based on results of tests performed using the methods and test criteria of the Manchester PHLS reference laboratory, United Kingdom as assessed for Australian conditions.[13–15] 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 NAA is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

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

Number of laboratory-confirmed cases

There were 361 instances of laboratory-confirmed cases of IMD in 2004 (Table 1) compared with 494 cases in 2003. In 245 cases a positive culture was obtained with or without a positive non-culture based test and 116 cases were confirmed by a non-culture based method alone. The total number of all laboratory-confirmed cases fell in 2004 compared with 2003 in all jurisdictions except in Western Australia where identical numbers were recorded in each year. There were 47 fewer laboratory-confirmed cases in New South Wales in 2004, 38 fewer in Victoria, 21 fewer in Queensland, 17 fewer in South Australia, five fewer in the Northern Territory and three fewer in the Australian Capital Territory and Tasmania.

Seasonality

Eighty-four (23.5%) of cases occurred between 1 January and 31 March 2004, 99 (27.5%) between 1 April and 30 June, 109 (30%) between 1 July and 30 September and 69 (19%) between 1 October and 31 December 2004. A winter peak of meningococcal disease is usual.

Table 1. Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2004, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	A	Y	W135	NG	
ACT	3	8					11
NSW	78	18		3	5	19	123
NT	5	1			1		7
Qld	50	19	1	2	2	1	75
SA	13	1					14
Tas	8	5			1	3	17
Vic	55	13		3	2	3	76
WA	31	6			1		38
Australia	243	71	1	8	12	26	361

NG Not groupable.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those aged four years and under (Table 2, Figure 1). Those aged less than one year or in the 1–4 years age group accounted for 49 (13.7%) and 62 (17.2%) cases respectively. The combined total of cases confirmed by all methods in these two groups (111) is less than that in 2003 (140). However, these two age groups together comprised a similar proportion of all cases in 2004 (30.9%) as in 2003 (28.3%). A secondary disease peak is also usual in the 15–19 years age group. The total of 61 cases (17% of all confirmed cases) in this age group in 2004 was less than the 89 (18%) seen in 2003. Those aged 15–24 years together accounted for 96 cases (26.7%).

Serogroup data

The serogroup of the meningococci causing disease was determined in 335 cases. Two hundred and forty-three (73%) were serogroup B, 71 (21%) serogroup C, one serogroup A, 8 (2%) serogroup Y and 12 (3.3%) serogroup W135. The serogroup was not determined in one culture confirmed case, in 10 of 101 cases confirmed by NAA or in any of the 12 serologically confirmed cases. In 2003, a total of 285 (58%) cases of serogroup B and 155 (31%) cases of serogroup C IMD were identified from a total of 494 laboratory-confirmed cases.

The serogroup distribution varied with age (Figure 2) and jurisdiction (Table 2), as in previous years. Serogroup B disease is concentrated in younger age groups with serogroup C infections increasing as a proportion of all isolates in adolescents and young adults (Figure 2).

Figure 1. Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2004, by age

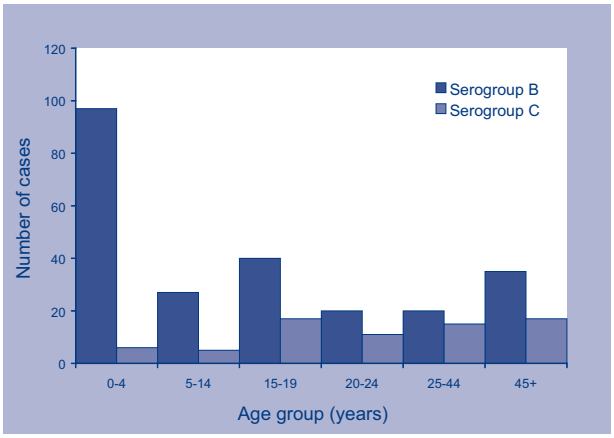


Figure 2. Serogroup B and C meningococcal disease as a percentage of cases of invasive meningococcal disease confirmed by all methods, Australia, 2004, by age

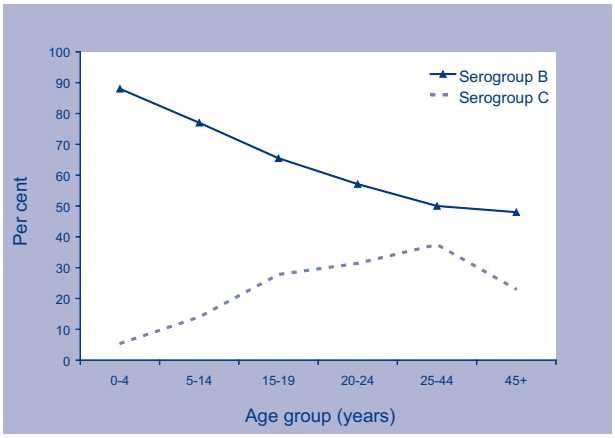


Table 2. All laboratory-confirmed cases of invasive meningococcal disease, Australia 2004, by age, jurisdiction and serogroups

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	1	0	0	0	1	0	0	0	0	1	3
	C	0	1	0	1	0	2	2	2	0	0	8
	Total	1	1	0	1	1	2	2	2	0	1	11
NSW	B	14	19	9	2	8	6	3	11	3	3	78
	C	0	1	0	0	3	3	6	5	0	0	18
	Total	15	24	10	3	13	13	15	23	4	3	123
NT	B	0	3	0	0	0	1	0	1	0	0	5
	C	0	0	0	0	0	0	0	1	0	0	1
	Total	0	3	0	0	0	1	0	3	0	0	7
Qld	B	8	11	3	3	11	4	5	2	3	0	50
	C	1	0	0	1	11	2	3	0	1	0	19
	Total	10	11	3	5	23	6	9	3	5	0	75
SA	B	3	1	0	0	2	0	4	3	0	0	13
	C	1	0	0	0	0	0	0	0	0	0	1
	Total	4	1	0	0	2	0	4	3	0	0	14
Tas	B	3	3	0	0	2	0	0	0	0	0	8
	C	0	0	0	1	2	0	1	1	0	0	5
	Total	3	3	0	1	4	0	1	4	1	0	17
Vic	B	10	8	5	2	8	7	5	5	5	0	55
	C	1	1	0	0	1	1	2	5	2	0	13
	Total	12	10	5	2	10	8	7	13	9	0	76
WA	B	4	9	2	1	8	2	3	1	1	0	31
	C	0	0	1	1	0	3	1	0	0	0	6
	Total	4	9	3	2	8	5	4	1	2	0	38
Australia	B	43	54	19	8	40	20	20	23	12	4	243
	C	3	3	1	4	17	11	15	14	3	0	71
	Other	3	4	1	2	4	4	5	15	6	0	47
	Total	49	62	21	14	61	35	42	52	21	4	361
	%	13.6	17.2	5.8	3.9	16.9	9.7	11.6	14.4	5.8	1.1	100

NS Not stated.

Totals include cases due to other serogroups (n = 21) and cases where the serogroup was not determined (culture confirmed 1, NAA confirmed 13 and serology confirmed 12).

In 2004, 97 (88%) of the total of 110 laboratory-confirmed IMD cases in those aged less than four years were serogroup B and 6 (5.5%) were serogroup C. In those aged 5–14 years, 27 serogroup B meningococcal cultures represented 77 per cent of the 35 confirmed cases and the five cases of serogroup C represented 14 per cent. The 96 confirmed cases in those aged 15–24 years comprised 60 (63%) serogroup B and 28 (29%) serogroup C. Half of the 117 infections in older age groups were serogroup B and a quarter were serogroup C.

When data from 2004 and 2003 are compared, the number of both serogroup B and serogroup C cases in 2004 was lower in all the above age groups (Table 3). The decrease in serogroup C cases in 2004 when compared with 2003 was proportionally greater than the reduction in the number of serogroup B cases. Serogroup B infections thus represented a higher proportion of all cases in 2004 than in 2003.

Jurisdictional differences in the distribution of serogroup B and C meningococcal cases continued in 2004 (Table 1). Serogroup B infections predominated nationally and in all jurisdictions except the Australian Capital Territory where 8 of 11 confirmed cases were with serogroup C. In New South Wales, Queensland, South Australia, Tasmania and Victoria, the number and proportion of cases represented by serogroup C meningococci decreased. Substantial decreases in the number of serogroup C infections were noted in Victoria (from 47 in 2003 to 13 in 2004), New South Wales (44 to 18) and Queensland (37 to 19). Lower numbers of serogroup C cases were also seen in Tasmania (decrease in 2004 by 3), the Australian Capital Territory (by 2) and South Australia (by 1). The number of serogroup C cases increased by one in the Northern Territory and in Western Australia.

Outcome data for all laboratory-confirmed cases of invasive meningococcal disease

Outcome data (survived or died) were available for 225 (63%) of the 358 laboratory-confirmed cases (Table 4). Eighteen deaths were recorded in this group (8%) (Table 4). Outcomes were available for 160 of 243 (66%) serogroup B infections and 34 of 71 (48%) serogroup C infections. There were 13 (8.1%) deaths in serogroup B infections and 3 (8.8%) in serogroup C infections.

There were two deaths in 26 patients (7.7%) with meningitis; both of these patients were infected with a serogroup B strain. Fifteen deaths were recorded in 181 bacteraemic patients (8.3%). There were 135 cases of serogroup B meningococcal bacteraemia with 10 deaths (7.4%) and 30 cases were caused by serogroup C strains among whom three fatalities were recorded (10%). No fatalities were recorded with serogroup Y (5 cases), but there were two fatalities among the seven instances of serogroup W135 bacteraemia.

Table 3. A comparison of the number and proportion of serogroup B and serogroup C confirmed cases, 2004 and 2003, by age

Year	Serogroup	Age							
		< 4 years		5–14 years		15–24 years		25+ years	
		n	%	n	%	n	%	n	%
2004	B	97	88	27	77	60	63	59	50
	C	6	5.5	5	14	28	29	32	27
	All	110		35		96		117	
2003	B	116	83	33	53	69	48	66	47
	C	14	10	21	33	67	44	51	36
	All	140		63		151		140	

Table 4. Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, 2004, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	12	3	1	0	2	24
	Died	2	0	0	0	0	2
	Total	20	3	1	0	2	26
Septicaemia	Survived	125	27	4	5	5	166
	Died	10	3	0	2	0	15
	Total	135	30	4	7	5	181
All cases	Survived	147	31	5	5	19	207
	Died	13	3	0	2	0	18
	Total	160	34	5	7	19	225

NG Not groupable.

Phenotypes of invasive meningococcal isolates

Isolates continue to be phenotyped and considerable heterogeneity amongst invasive isolates was again evident. The predominant serotypes/serosubtypes in each state and territory are shown in Table 5. Serogroup B meningococci are in general more heterogeneous, but also more difficult to characterise by serological methods and a number could not be phenotyped. Nineteen isolates of the B:4:P1.4 phenotype were identified in Victoria, New South Wales, Queensland and Western Australia. Numbers of isolates of this phenotype, circulating in New Zealand at high rates for many years, have declined in recent years in Australia. Forty-one meningococci of this phenotype were detected in 2002 and 25 in 2003. Historically, the other common phenotype circulating has been B:15:P1.7 but only eight strains of this type were seen, five of them in New South Wales.

Table 5. Commonly isolated serotypes and serosubtypes and phenotypes of *Neisseria meningitidis* of interest, Australia, 2004, by state or territory

State/territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT		0		0	2a	7	1.4	2
							1.5,2	1
							nst	4
NSW	4	16	1.4	8	2a	1	1.5	6
			1.14	3			1.5,2	0
			1.15	3			1.2	0
			nst	2			1.4	1
							nst	4
	15	7	1.7	5	14	`	1.14	1
			1.6	1	NT	3	1.5	2
			1.13	1			1.13	1
	nt	28	1.4	4				
			1.7	1				
			1.15	2				
			others	5				
			nst	16				
NT	14	2	nst	2	2a	1	1.5	1
Qld	4	4	1.4,(7)	2	2a	13	1.4	6
			1.14	2			1.5	5
	15	4	1.7,(1)	4			nst	2
	1	3	1.14	2	14	1	1.12,13	1
			1.2	1				
	nt	13	1.4	6				
			1.7	1				
			1.5,2	1				
			1.15	1				
			nst	3				
Tas	4	2	1.19,15	2	2a	4	1.4, 7–2	4
Vic	4	13	1.4	8	2a	8	1.4	7
			1.15	2			1.5,10	1
			1.16	1			1.14	1
	1	1	1.14	1	4	1	1.16	1
	15	6	various		nt	1	1.12,13	1
	2b	1	1.16	1				
	nt	18	1.15	8				
			1.14	4				
			nst	3				
WA	4	2	1.4	1	2a	1	1.4	1
			nst	1	nt	2	1.5	1
	15	1	nst	1			nst	1
	nt	1	1.16	1				

nt Not serotypable.

nst Not serosubtypable.

Of interest were any serogroup B meningococci of serotypes 2a or 2b. These serotypes are more often seen in serogroup C organisms, but in 2004, a single isolate only of phenotype B:2b:P1.16 was detected.

Among serogroup C strains, phenotype C:2a:P1.4 is of particular interest. This phenotype has figured prominently in Victorian data in recent years. In 2004, 21 serogroup C isolates were of this serotype/serosubtype. In 2003, there were 29 isolates with this phenotype. This phenotype was detected in all jurisdictions except the Northern Territory and South Australia. Seven were found in Victoria, six in Queensland, four in Tasmania and two in the Australian Capital Territory with single examples in New South Wales and Western Australia. All except three of the typeable serogroup C isolates was of serotype 2a. Serotype 2b strains were not detected in serogroup C isolates.

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 and those shown as serologically positive were culture and PCR negative. There were 53 isolates from CSF either alone or with a blood culture isolate and 186 from blood cultures alone. There were six other isolates from synovial fluid and tissues. Trends in relative rates of positive isolates have been followed in these reports (Figure 3). The ratio of CSF isolates to blood culture isolates was 0.28:1. For PCR based diagnosis, this ratio was 0.73:1. This probably reflects the capacity of PCR to amplify meningococcal DNA even after antibiotic treatment and/or delayed lumbar puncture.[16]

Table 6. Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2004

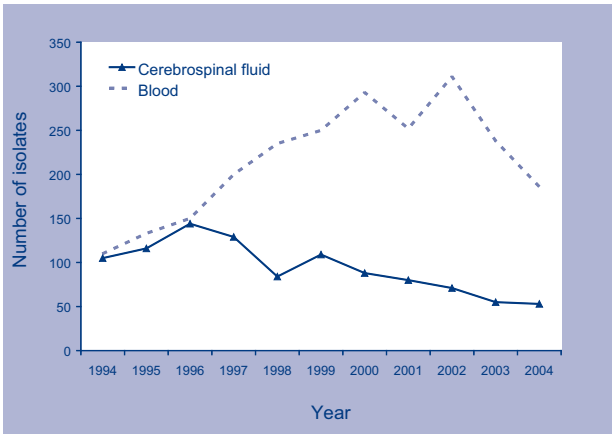
Specimen type	Isolate of MC	PCR positive*	Total
Blood	186	59	245
CSF +/– blood	53	43	96
Other†	6	2	8
Serology alone‡			12
Total	245	104	361

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

† Joint and tissue samples.

‡ Serology positive in the absence of positive culture or PCR.

Figure 3. Numbers of meningococcal isolates from cerebrospinal fluid and blood culture, 1994 to 2004



Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Two hundred and thirty-eight isolates were available for determination of their susceptibility to penicillin. Using defined criteria, 90 strains (38%) were fully sensitive to penicillin and 147 (62%) less sensitive (MIC 0.06 to 0.5 mg/L). These proportions are similar to those observed in recent years. One isolate from a blood culture had an MIC of one mg/L and six isolates, from blood cultures (3), CSF (2) and joint fluids (1), had MICs of 0.5 mg/L.

Other antibiotics

All isolates were susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to the prophylactic agents ciprofloxacin and rifampicin.

Discussion

There were 361 laboratory-confirmed cases of IMD in 2004, 245 (67.9%) by culture of *N. meningitidis* and 116 (32.1%) by non-culture based methods. The 245 isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme (AMSP) in 2004 was the lowest number recorded since the 216 examined in the first year of the program in 1994. The annual numbers of isolates examined from 1997 to 2002 have ranged between 323 and 393 with 303 available in 2003.

This AMSP report is for the first time, based on a combined analysis of all cases of IMD confirmed by any recognised method. In earlier NNN reports, analyses were derived from culture confirmed cases, with NAA based data added from 1999. The increased contribution of non-culture-based

methods to IMD diagnosis means that a more comprehensive picture can be obtained if diagnoses by all test modalities are aggregated.

In 2004, the number of laboratory-confirmed cases of IMD was less in each jurisdiction than in 2003, except in Western Australia where 38 cases were identified in both years. Numerically, larger reductions in numbers were seen in New South Wales, Victoria, Queensland and South Australia. Serogroup B and serogroup C infections again predominated although numbers of both serogroups were less than those recorded in recent years. However the reduction in the number of serogroup C infections from 155 in 2003 to 71 in 2004 (54%) was proportionally greater than the decrease in serogroup B infections from 285 to 243 (15%). This was true for all age groups (Table 4) so that serogroup B infection accounted for a greater percentage of all IMD in 2004. NNN reports have consistently noted that the age distribution of IMD showed a primary peak in those aged four years or less and was predominantly with serogroup B meningococci while in a secondary peak in adolescents and young adults the proportion of serogroup C infections increased. This pattern was again observed in 2004. Serogroup C disease remained an important element of IMD in young adults and older age groups.

Analysis of the effect of the national vaccination program with serogroup C conjugate vaccine is beyond the scope of this report. Specific mention was made in the 2003 report of the caveats placed on AMSP data if used to assess disease rates and effects of vaccination campaigns.[10] These included differences between clinical and laboratory surveillance case definitions, the different rates of introduction and use of non-culture based confirmatory tests over time and the influence of clinical practice on laboratory based diagnosis. These concerns remain and fluctuations in the rates of IMD can occur naturally or be influenced by rates of intercurrent viral infection. Any assessment of the impact of the vaccination program on IMD rates will thus require a continuing and detailed analysis.

Preliminary data since the vaccination campaign indicate that some meningococci isolated show evidence of genetic recombination. Clonal complexes of meningococci responsible for IMD may express different capsular polysaccharides or recombination of *porA* and *porB* and other genes may occur. A number of strains that show these characteristics have been detected in Australia, but only in low numbers to date. However close attention should continue to be paid to analysis of meningococcal subtypes and any evidence of their clonal expansion thoroughly investigated.

Mortality data were assessable in only a proportion of cases and must be interpreted with caution. The NNN does not attempt collection of morbidity data associated with IMD.

A penicillin MIC of one mg/L was detected in a single strain in 2004. NNN trend data show no recent shifts in penicillin MICs of invasive strains. Penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and the prophylactic agents rifampicin and ciprofloxacin.

Acknowledgments

Isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these strains is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and public health personnel.

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References

1. National Neisseria Network. Meningococcal Isolate Surveillance Australia, 1994. *Commun Dis Intell* 1995;19:286–289.

2. National Neisseria Network. Meningococcal Isolate Surveillance Australia, 1995. *Commun Dis Intell* 1996;20:422–424.

3. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1996. *Commun Dis Intell* 1997;21:217–221.

4. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1997. *Commun Dis Intell* 1998;22:205–211.

5. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1998. *Commun Dis Intell* 1999;23:317–323.

6. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1999. *Commun Dis Intell* 2000;24:181–189.

7. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2000. *Commun Dis Intell* 2001;25:113–121.

8. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2001. *Commun Dis Intell* 2002;26:407–418.

9. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2002. *Commun Dis Intell* 2003;27:196–208.

10. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2003. *Commun Dis Intell* 2004;28:194–206.

11. Australian Gonococcal Surveillance Programme. Penicillin sensitivity of gonococci in Australia: development of an Australian gonococcal surveillance programme. *Br J Vener Dis* 1984;60:226–230.

12. Porrit RJ, Mercer JL, Munro R. Detection and serogroup determination of *Neisseria meningitidis* in CSF by polymerase chain reaction (PCR). *Pathology* 2000;32:42–45.

13. Gray SJ, Borrow R, Kaczmarski EB. Meningococcal serology. In: Pollard AJ, Martin MCJ, eds. *Meningococcal Disease Methods and Protocols*. Totawa, New Jersey: Humana Press, 2001. p. 61–87.

14. 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;33:375–378.

15. 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: In press 2005.

16. Bryant PA, Hua YL, Zaia A, Griffith J, Hogg G, Curtis N, Carapetis JR. Prospective study of a Real-Time PCR that is highly sensitive, specific and clinically useful for diagnosis of meningococcal disease in children. *J Clin Microbiol* 2004;42:2919–2925.