

ANNUAL REPORT OF THE AUSTRALIAN MENINGOCOCCAL SURVEILLANCE PROGRAMME, 2006

The Australian Meningococcal Surveillance Programme

Abstract

In 2006 there were 271 laboratory-confirmed cases of invasive meningococcal disease analysed by the National Neisseria Network, a nationwide network of reference laboratories. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 166 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 105 cases were confirmed by non-culture-based methods. Nationally, 217 (80%) confirmed cases were infected with serogroup B and 26 (9.6%) with serogroup C meningococci. The total number of confirmed cases was 74 (21%) fewer than the 345 cases identified in 2005. Numbers of cases decreased in all jurisdictions except Queensland. The age group showing the greatest decrease in numbers (by about one-third) was in those aged 25 years or more. A typical primary disease peak was observed in those aged 4 years or less with a lower secondary peak in adolescents and young adults. Serogroup B cases were 93% of all cases in those aged 4 years or less and 77% in those aged 15–24 years. The proportion of invasive disease represented by serogroup C disease was highest in the 20–24 years and 25–44 years age groups. The common phenotypes circulating in Australia were B:15:P1.7, B:4:P1.4, C:2a:P1.4 and C:2a:P1.5, but again with significant jurisdictional differences. No evidence of meningococcal capsular ‘switching’ was detected. About two thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06–0.5 mg/L). All isolates remained susceptible to rifampicin and ciprofloxacin. *Commun Dis Intell* 2007;31:185–194.

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

Introduction

Invasive meningococcal disease (IMD) remains an infection of public health interest in Australia. Additionally, a publicly-funded program of selective vaccination with conjugate serogroup C meningococcal vaccine was completed in 2004 and the prospect of additional vaccines, e.g. porin-based vaccines for serogroup B meningococcal disease, increases the need for precise data on circulating meningococcal subtypes. The National Neisseria

Network (NNN) is a national laboratory-based program for the examination of *Neisseria meningitidis* from cases of IMD and has operated since 1994 through the collaboration of reference laboratories in each jurisdiction. The NNN supplies information on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility. These data are meant to 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.

Annual reports summarising data gathered since the inception of the program were published in *Communicable Diseases Intelligence*. The following report analyses the characteristics of meningococci isolated in the calendar year 2006 and follows the format used first for the 2004 annual report published in *Communicable Diseases Intelligence* where data on all laboratory-confirmed cases were aggregated for analysis. Prior to 2004, data on IMD diagnosed by culture-based and non-culture methods were provided separately.

Methods

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

Isolate-based invasive meningococcal diseases cases

Each case confirmation was based upon isolation of a meningococcus from a normally sterile site and defined as IMD according to Public Health Laboratory Network criteria. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was grown from both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case was classified as one of meningitis. It is recognised that the total number of

cases, and particularly the number of cases of meningitis e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture was sterile, is underestimated. However the above approach has been used since the beginning of this Programme² 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 Programme uses the following parameters to define the various levels of penicillin susceptibility or resistance when determined by a standardised agar plate dilution technique.^{3,4}

Sensitive, MIC \leq 0.03 mg/L.

Less sensitive, MIC 0.06–0.5 mg/L.

Relatively resistant MIC \leq 1 mg/L.

Strains with MICs that place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses. However precise 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 nucleic acid amplification (NAA) and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques⁵ 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 report.⁶ 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.^{7–9} 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 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 271 instances of laboratory-confirmed cases of IMD in 2006 (Table 1) compared with 345 in 2005, 361 in 2004 and 494 in 2003. In 166 cases (61.2%), a positive culture was obtained with or without a positive non-culture-based test and 105 cases were confirmed by a non-culture-based method alone. The total number of all laboratory-confirmed cases decreased in most jurisdictions in 2006 when compared to 2005 data. The largest decreases in numbers were in New South Wales (to 84 from 112) and Western Australia (to 19 from 45). Smaller decreases were noted in other jurisdictions with the exception of Queensland where numbers detected increased

Table 1. Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2006, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	A	Y	W135	NG*	
ACT	1	1					2
NSW	57	16		1	5	5	84
NT	3	0					3
Qld	58	4		1	1	4	68
SA	13	0		1	1		15
Tas	4	1					5
Vic	64	3		1	5	2	75
WA	17	1			1		19
Australia	217	26	0	4	13	11	271

* Not serogrouped.

to 68 from 58 after a fall in 2005. There were 2 less cases in the Australian Capital Territory, 3 less in the Northern Territory, 8 less in South Australia, 3 less in Tasmania and 4 less in Victoria.

There were also 2 instances of isolation of *N. meningitidis* from travellers returning to Australia from overseas—1 of serogroup A, apparently acquired in France, and 1 of serogroup B, acquired in Greece.

Seasonality

Fifty-seven cases occurred between 1 January and 31 March, 60 between 1 April and 30 June, 95 between 1 July and 30 September and 59 between 1 October and 31 December. A winter peak of meningococcal disease is usual.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those 4 years and under (Table 2, Figure 1). Those aged less than 1 year or in the 1–4 age group together accounted for 100 (37% of the total) cases in 2006. There were 110 cases confirmed in these age groups (32% of the total) in 2005. A secondary disease peak is also usual in the adolescent and young adult age group. The total of 49 (18% of all confirmed cases) cases in those aged 15–19 years was much the same as the number and proportion of cases in this age group in 2005 (48, 17%), but remained less than the 61 (17%) cases seen in 2004 and the 89 (18%) cases seen in 2003. Those aged 15–24 years together accounted for 79 (29%) cases in 2006. There were 88 (23.4%) cases in 2005 and 96 (26.7%) cases in these combined age groups in 2004.

Serogroup data

The serogroup of the meningococci causing disease was determined in 259 of the 271 laboratory-confirmed cases of IMD. Of these 259 where a serogroup was determined, 217 (83.8%) were serogroup B and 26 (10%) were serogroup C. In 2005, there were 251 (76.9%) confirmed cases of serogroup B and 50 (15.3%) of serogroup C and in 2004, 243 (73%) serogroup B cases and 71 (21%) serogroup C cases. In 2006, an additional 13 (5%) cases were W135 and 3 (1.2%) were serogroup Y.

The serogroup distribution varied with age (Figure 1) and jurisdiction (Table 2), as in previous years. Traditionally, 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).

Serogroup B meningococci predominated in all age groups in aggregated national data. Ninety-three (93%) of the total of 100 laboratory-confirmed IMD

Figure 1. Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2006, by age group

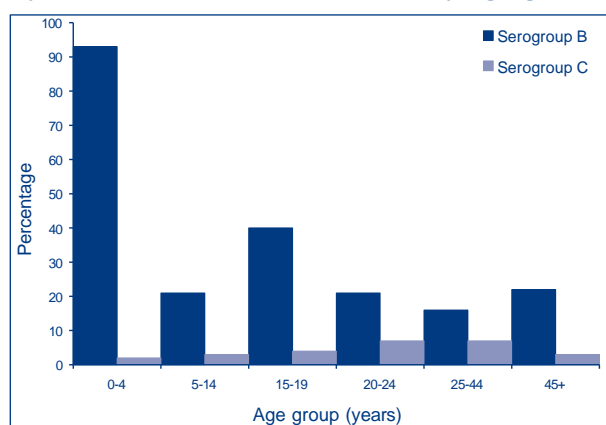
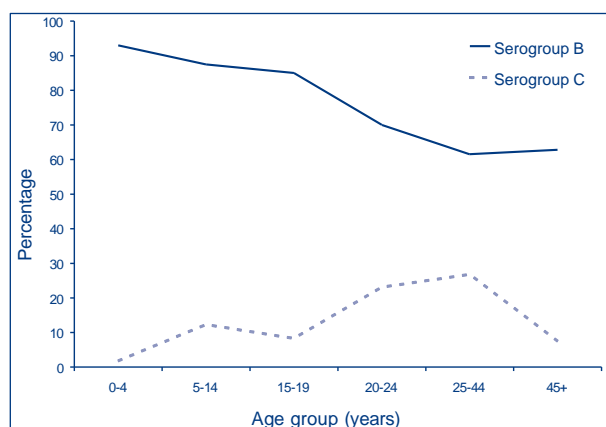


Figure 2. Serogroup B and C meningococcal disease as a percentage of cases of invasive meningococcal disease confirmed by all methods, Australia, 2006, by age group



cases in those aged less than 4 years were serogroup B and 2 (2%) serogroup C. In those aged 5–14 years, 21 serogroup B meningococcal cultures represented 84% of the 25 confirmed cases and the 3 cases of serogroup C represented 12% of cases. There were 49 cases of IMD confirmed nationally in those aged 15–19 years in 2006 compared with 48 in 2005. These 49 cases comprised 40 (82%) serogroup B and 4 (8.2%) serogroup C—virtually identical numbers and proportions were reported in 2005. There were 30 instances of IMD in those aged 20–24 years in 2006. Of these, 21 (70%) were serogroup B and 7 (23%) were serogroup C. These numbers and proportions differ little from 2005 data for this age group when 22 (7%) infections with serogroup B and 8 (24%) with serogroup C meningococci were recorded out of a total of 33 cases. (In 2004, the number of infections, 35, and their distribution, 20 (57%) of serogroup B and 11 (35%) of serogroup C, was not too dissimilar).

Table 2. All laboratory-confirmed cases of invasive meningococcal disease, Australia, 2006, by age group, 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						1					1
	C							1				1
	Total						2					2
NSW	B	14	7	4	3	10	6	4	7	2		57
	C	1		2		2	5	4	1	1		16
	Total	15	9	6	3	14	13	9	11	4		84
NT	B	1	1	1								3
	C											0
	Total	1	1	1								3
Qld	B	16	17		1	15	5	4				58
	C	1				1		2				4
	Total	17	18	1	4	18	5	7	1			68
SA	B	2	2			1		2	2	1	3	13
	C											0
	Total	2	2			1		2	3	1	4	15
Tas	B		1	1				1	1			4
	C					1						1
	Total		1	1		1		1				4
Vic	B	12	12	8	2	13	6	4	6	1		64
	C						1	1		1		3
	Total	13	12	8	2	14	7	7	7	5		75
WA	B	7	1	1		1	3	1	1	1	1	17
	C				1							1
	Total	8	1	1	1	1	3	1	1	1	1	19
Australia	B	52	41	15	6	40	21	16	17	5	4	217
	C	2	0	2	1	4	7	7	1	2		26
	Other	2	3	1	0	5	2	4	6	4	1	28
	Total	56	44	18	7	49	30	27	24	11	5	271
	% of all	20.7	16.2	6.7	2.6	18	11	10	8.9	4	1.9	

NS Not stated. (Totals include cases due to other serogroups (n = 27) and cases where the serogroup was not determined (culture confirmed 0, NAA confirmed 8 and serology confirmed 3).

In older age groups (25 years and above), there were 62 laboratory-confirmed cases of IMD in 2006, of which 38 (61.2%) were serogroup B and 10 (16.1%) were serogroup C. The latter number is less than the 27 serogroup C cases seen in older age groups in 2005 and the 32 seen in 2004.

Table 3 shows a comparison of the number and proportion of serogroup B and C cases by age group from 2004 to 2006. There was no change in the number of serogroup B cases in those aged 4 years or less or in those aged 15–24 years, but a reduction in serogroup B numbers in those aged 5 to 14 years from 38 in 2005 to 21 in 2006. There has also been a substantial decline in the number of cases of IMD

in those aged more than 25 years—from 101 to 61. This occurred for both serogroup B (to 38 from 51) and serogroup C (to 10 from 27) infection from 2005 to 2006.

Some pronounced differences in the jurisdictional distribution of serogroup B and C meningococcal have been noted previously in Australia. In recent years serogroup B infections predominated nationally and in all jurisdictions, but serogroup C disease was more prevalent in the eastern States and uncommonly encountered in South and Western Australia. The number of serogroup C cases nationally in 2006 was about half that for 2005. Serogroup B disease now accounts for more than 80% of IMD nation-

Table 3. A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2006, by age group

Year	Serogroup	Age group									
		< 4 years		5–14 years		15–19 years		20–24 years		25+ years	
			%		%		%		%		%
2006	B	93	93.0	21	84	40	82	21	70	38	61.3
	C	2	2.0	3	12	4	8.2	7	23	10	16.1
	All	100		25		49		30		62	
2005	B	99	90.0	38	75	39	81.0	22	67	51	50.0
	C	6	5.5	5	10	4	8.0	8	24	27	27.0
	All	110		51		48		33		101	
2004	B	97	88.0	27	77	40	65.0	20	57	59	50.0
	C	6	5.5	5	14	17	28.0	11	31	32	27.0
	All	110		35		61		35		117	

ally with little jurisdictional variation in serogroup distribution. Numbers of serogroup C cases in most jurisdictions were low, but 16 of the 26 cases of serogroup C disease nationally occurred in New South Wales. Eleven of these 16 serogroup C cases in New South Wales were in those outside the 'vaccination' age group. In Queensland, while the total number of IMD cases increased, the numbers of serogroup C infections was low. Clusters of serogroup C disease were not uncommonly encountered in past years, but none were reported in 2006.

Phenotypes of invasive meningococcal isolates

Typically there is considerable heterogeneity in serogroup B meningococci and this was again the case in 2006 when the phenotype of invasive isolates, based on a determination of their serogroup, serotype and serosubtype, were analysed. 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 14 isolates of the B:4:P1.4 phenotype were identified in Victoria, New South Wales, Queensland and South Australia

Table 4. Common serotypes and serosubtypes of isolates from culture positive cases of *Neisseria meningitidis* infection, 2005, by state or territory

State or territory	Serogroup B				Serogroup C					
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n		
ACT	4	1	1.7	1	2a	1	1.4	1		
NSW	4	11	1.4	4	2a	8	1.5	7		
			1.7	3			1.5,2	1		
			1.15	1			nt	1	nst	1
			1.5	1	15	6	1	1	1	1
			1.3,6	1						
			nst	1						
			1.5	1						
	1	6	Various	4	1	1	1	1		
									1.7,14,	1
									nst	2
	nt	17	Various	4	1	1	1	1		
									nst	2
									nst	7
									1.4	4
1..14									3	
1.9	2									
1.10	1									

Table 4. Common serotypes and serosubtypes of isolates from culture positive cases of *Neisseria meningitidis* infection, 2005, by state or territory, continued

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
NT	nt	3	nst	3				
Qld	15	7	1.7	5	2a	1	1.5	1
			1.7,16	1	nt	2	1.5	1
			nst	1				
	1	4	Various	4			nst	1
	4	2	1.4	2				
	2a	1	1.5	1				
	nt	20	1.4	9				
			1.9	2				
			1.5	1				
			1.6	1				
			1.14	1				
		nst	6					
Tas	4,,7	2	nst	2	2a	1	1.4	1
	nt	1	nst	1				
Vic	4,7	14	1.19,15	5	2a	1	1.7,4	1
			1.22,14	3	2b	1	1.5,2	1
			1.5	3				
			1.7 (4)	2				
			1.18	1				
	15	5	1.7	5				
	19,1	7	1.18	4				
			1.22,14	3				
	19	3	Various	3				
	7	6	1.19	5				
		1.7,4	1					
WA	14	3	1.2,14	3	2a	1	nst	1
	15	1	1.2,14	1				
	1	1	nst	1				
	nt	2	1.19,15	1				
			nst	1				
SA	15	1	1.4	1				
	nt	7	1.14	3				
			nst	4				

nt Not serotypable.

nst Not serosubtypable.

in 2006. Numbers of isolates of this phenotype, circulating in New Zealand at high rates for many years, have declined in recent years in Australia. Historically, the other common phenotype circulating has been B:15:P1.7 and remained so in 2006. In 2006 nationally, a total of 16 examples were detected from the Australian Capital Territory, Victoria, New South Wales, Queensland and Western Australia. This distribution was also observed in 2005.

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, e.g. serogroup B isolates of serotype 2a or 2b. A single isolate of B:2a:P1.5 was found in New South Wales in 2006. Among serogroup C strains, phenotype C:2a:P1.4 is of particular interest. This phenotype has figured prominently in Victorian data in former years. In 2003 there were 29 cases while in

2004 there were 21 and in 2005 8 serogroup C isolates of this serotype/serosubtype were detected nationally. Eight isolates with this phenotype were seen nationally in 2006 and these were detected in the Australian Capital Territory, Victoria, New South Wales and Queensland, all in low numbers. All except one of the serotypeable serogroup C isolates was of serotype 2a. The most frequently detected 2a serosubtype, 1.5, was present only in New South Wales and Victoria.

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

Outcome data (survived or died) were available for 125 (46%) of the 271 laboratory-confirmed cases (Table 5). Nine deaths were recorded in this group (7.2%). Outcomes were available for 99 of 217 (45.6%) serogroup B infections and 12 of 26 (46%) serogroup C infections. There were 6 (6%) deaths from serogroup B infections and a single death attributable to serogroup C disease. The other 2 fatal cases were with infections due to W135 meningococci.

There were 3 deaths in 44 patients (6.8%) with meningitis, all due to serogroup B meningococci. Six deaths were recorded in 68 bacteraemic patients (8.8%). There were 58 cases of serogroup B meningococcal bacteraemia with 3 deaths (5%). The single fatality with serogroup C disease was in a group of 5 cases where outcomes were recorded and the 2 septicaemic fatalities due to W135 meningococci were recorded in 5 instances of bacteraemia with this serogroup.

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 48 isolates from CSF either alone or with a blood culture isolate and 116 from blood cultures alone. There were 2 other isolates from synovial fluid. The ratio of CSF isolates to blood culture isolates was 0.4:1. For PCR-based diagnoses, this ratio was 0.8:1.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and sixty-seven isolates were available for determination of their susceptibility to penicillin. Using defined criteria, 113 isolates (67.6%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and 55 (32.4%) were fully sensitive

Table 6. Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2006

Specimen type	Isolate of meningococci	PCR positive*	Total
Blood	116	50	166
CSF +/- blood	48	42	90
Other†	2	5	7
Serology alone‡			8
Total	166	97	271

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

† Joint and tissue samples (5) or not stated (3).

‡ Serology positive in the absence of positive culture or polymerase chain reaction.

Table 5. Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, 2006, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	35	3	1	2	0	41
	Died	3	0	0	0	0	3
	Total	38	3	1	2	0	44
Septicaemia	Survived	58	4	1	3	2	68
	Died	3	1	0	2	0	6
	Total	61	5	1	5	2	74
All cases	Survived	93	11	2	5	5	115
	Died	6	1	0	2	0	9
	Total	99	12	2	7	5	125

NG Not groupable.

(\leq MIC 0.03 mg/L). These proportions are similar to those observed in recent years. Six isolates had MICs of 0.5 mg/L. Four of these were serogroup C isolates from New South Wales.

Other antibiotics

All isolates were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to ciprofloxacin. A single serogroup B strain from Queensland had a slightly elevated MIC for rifampicin of 1 mg/L.

Discussion

There has been a continuing decline in the number of laboratory-confirmed cases of IMD in Australia for several years¹⁰ and this continued in 2006. Numbers declined in all states and territories with the exception of Queensland. Cultures were obtained from sterile sites in 166 cases, the lowest number of isolates available over the duration of the program that commenced in 1994. Non-culture-based diagnoses were used to confirm a further 105 (38.7%) cases of IMD. This decrease in IMD has been attributable to all serogroups, but notably in serogroup C infections and the 26 cases of this serogroup identified nationally, accounted for 9.6% of all cases in 2006.

Traditionally, the distribution of cases of IMD in Australia showed major differences when considered by jurisdiction, age and serogroup of the infecting organism. Western and South Australia have long had a preponderance of serogroup B infections whereas Victoria, Tasmania and the Australian Capital Territory tended to have a greater proportion of serogroup C infections than New South Wales or Queensland. Differences between jurisdictions in serogroup distribution have become less in recent years with the relatively greater decline in serogroup C infections. Nationally in 2006, serogroup B infections were 8 times more common than serogroup C of IMD. Only in New South Wales did the number of serogroup C cases detected reach double figures. Most of these 16 cases were in those outside the target age groups for vaccination programs with serogroup C conjugate vaccine. Only small numbers of infections due to serogroups Y and W135 were encountered.

Serogroup B infections were again more frequently encountered in younger age groups where there is a primary peak in IMD infection rates. In the earlier years of this Programme, serogroup C disease figured prominently in a secondary disease peak that occurred in adolescents and young adults in some jurisdictions. However, serogroup C infections were infrequently encountered in this and other age groups in 2006. Also of interest is the overall decline in numbers of IMD in those aged 25 years or more (Table 3).

There has been a decrease in both serogroup B (by about one third) and serogroup C cases (by about two thirds) in this age group since 2004.

The NNN is not as well placed as others to analyse the effect of the national vaccination program with serogroup C conjugate vaccine on trends in IMD for reasons previously discussed.¹⁰ These included differences over time in data collection and laboratory detection methods. Further, fluctuations in the rates of IMD can occur naturally or be influenced by rates of intercurrent viral infection. However, it is still possible to speculate that the decrease in serogroup C disease in older age groups may be the result of a 'herd' immunity effect whereby the number of circulating serogroup C meningococci is reduced by the vaccination program in younger age groups who may otherwise be responsible for the introduction of a pathogenic strain into family groups. This does not explain the accompanying, but lower, decrease in serogroup B disease.

The continuing absence of any substantial numbers of meningococci showing evidence of genetic recombination is reassuring and consistent with findings elsewhere. Some concerns have been expressed that the well established ability of *Neisseria meningitidis* to undergo substantial genetic reconfiguration by a number of mechanisms may pose threats to the longer term efficacy of monovalent capsular vaccines. 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 proportion of cases and must be interpreted with caution. The NNN does not attempt collection of morbidity data associated with IMD.

NNN trend data show no major shifts in penicillin MICs in invasive isolates in Australia and penicillins remain a suitable treatment for IMD. The 4 New South Wales serogroup C isolates with penicillin MICs of 0.5 mg/L were of the same phenotype. However, this was the common serogroup C phenotype in New South Wales in 2006 and no case linkages were established. All isolates were susceptible to the third generation cephalosporins and to the prophylactic agents rifampicin and ciprofloxacin.

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