Annual report

# Australian Meningococcal Surveillance Programme annual report, 2016

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## Abstract

In 2016, there were 243 laboratory-confirmed cases of invasive meningococcal disease analysed by the Australian National Neisseria Network. This number was the highest number of laboratory confirmed cases since 2008. Probable and laboratory confirmed invasive meningococcal disease (IMD) are notifiable in Australia, and there were 252 IMD cases notified to the National Notifiable Diseases Surveillance System in 2016, the highest number reported since 2010. Meningococcal serogrouping was able to be determined for 98% (237/243) of laboratory confirmed IMD cases. Serogroup B infections accounted for 87 cases (37%), the lowest number and proportion reported since inception of the Australian Meningococcal Surveillance Programme (AMSP) in 1997. In contrast, the number and proportion of serogroup W infections (44%, 107 cases) in 2016 was the highest since the AMSP began. In addition, the number and proportion of serogroup Y infections (16%, 40 cases) was also the highest recorded by the AMSP. Molecular typing results were available for 225 of the 243 IMD cases. Of the serogroup W IMD strains that were able to be genotyped, 92% (97/105) have the PorA antigen encoding gene type P1.5,2 and of these, 72% (70/97) were sequence type 11, the same type as the hypervirulent serogroup W strain that has been circulating in the UK and South America since 2009. The primary IMD age peak was observed in adults aged 45 years or more, whilst secondary disease peaks were observed in those aged less than 5 years, and in adolescents aged 15–19 years. Serogroup B infections predominated in the age groups less than 1 year and 20–24 years, whereas serogroup W infections predominated in those aged 45 years or more. For all other age groups, distribution of serogroup B and W infections was roughly equal. Of the IMD isolates tested for antimicrobial susceptibility, 6% (11/189) were resistant to penicillin, and decreased susceptibility to penicillin was observed in a further 90% (170/189) of isolates. One Men W isolate demonstrated an elevated minimum inhibitory concentration (MIC) to ceftriaxone (0.125mg/L), the highest reported in Australia. All isolates tested were susceptible to rifampicin and ciprofloxacin.

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

## Introduction

Australia's National Neisseria Network (NNN) is an established, collaborative network of reference laboratories in each state and territory that contribute to the laboratory surveillance system of the pathogenic *Neisseria* species *(N. meningitidis* and *N. gonorrhoeae).* Since 1994 the NNN has coordinated laboratory data from the examination of *N. meningitidis* cases of invasive meningococcal disease (IMD) for the Australian Meningococcal Surveillance Programme (AMSP).[1] The AMSP is funded by the Australian Government Department of Health. The NNN laboratories supply phenotypic and genotypic data of invasive meningococci for the AMSP. These data supplement the notification data from the National Notifiable Diseases Surveillance System (NNDSS), which includes cases of probable and laboratory confirmed IMD. The characteristics of meningococci responsible for IMD, and the associated demographic information, are important considerations for management of individual patients and their contacts; and

to inform public health responses for outbreaks or case clusters, locally and nationally. The introduction of the publicly funded conjugate serogroup C meningococcal vaccine onto the National Immunisation Program in 2003 has seen a significant and sustained reduction in the number of cases of serogroup C IMD after 2003.[2] However, IMD remains an issue of public health concern in Australia and continued monitoring of phenotypic and genotypic features of IMD strains is critical to monitor, plan and inform clinical management and public health interventions.

## Methods

### Case confirmation of invasive meningococcal disease

Case confirmation is based on isolation of *N. meningitidis*, or a positive nucleic acid amplification testing (NAAT) from a normally sterile site, defined as laboratory definitive evidence of IMD by the Communicable Diseases Network Australia (CDNA) criteria [3]. Information regarding the site of infection, age and sex of the patients is collated by the NNN for the AMSP.

IMD cases are categorised on the basis of the site from which *N. meningitidis* was isolated, or from which meningococcal DNA was detected (blood, joint fluid, vitreous fluid). When *N. meningitidis* is detected from both blood and cerebrospinal fluid (CSF) from the same patient, the case is classified as one of meningitis.

### Phenotyping and genotyping of *Neisseria meningitidis*

Phenotyping is limited to the determination of the serogroup by detection of soluble polysaccharide antigens. Genotyping of both isolates and DNA extracts is performed by sequencing of products derived from amplification of the porin genes *por*A, *por*B and *fet*A.

### Antibiotic susceptibility testing

Isolates were tested to determine their minimum inhibitory concentration (MIC) values to antibiotics used for therapeutic and prophylactic purposes: ceftriaxone, ciprofloxacin; rifampicin. This program defines the penicillin categories as: sensitive (MIC ≤ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L) and resistant (MIC ≥ 1 mg/L).

## Results

In 2016, there were 243 laboratory-confirmed cases of IMD analysed by the NNN, and 252 cases notified to the NNDSS. Thus, laboratory data were available for 96% of notified cases of IMD in Australia in 2016 (Figure 1). This number of laboratory-confirmed cases of IMD was the highest reported since 2008, with an increase of 40% from the previous year (n=174). The number of cases notified to the NNDSS was the highest reported since 2010 (n=226), with an increase of 39% from the previous year (n=182). In 2016 the peak incidence for IMD occurred in mid-spring and early summer (1 October to 31 December 2016) (Table 1). This was different to previous years where the peak incidence occurred in mid-winter and early spring.

Victoria reported the highest number of cases (76 cases) in 2016, an increase from 54 cases in 2015 and the highest number of cases reported from this state since 2005 (n=80) (Table 2). New South Wales had the second highest number of IMD cases in 2016 (69 cases) and this was the highest number of cases reported in this state since 2010 (n=76). All jurisdictions, with the exception of South Australia and the Australian Capital Territory, recorded a rise in IMD cases in 2016 compared with 2015.

### Age distribution

The peak incidence of IMD in 2016, as in 2015, occurred in adults aged 45 years or more. This age group represented 35% (86/243) of IMD cases in 2016 (Table 3). Within this age group, 50 cases were in those aged 65 years or more, which was the highest number and proportion of cases for this age group reported by the AMSP. Prior to 2015, the primary peak incidence of IMD was in children less than 5 years of age, however in 2016, they represented 21% of IMD cases, the lowest proportion of cases noted by the AMSP

**Figure 1: Number of invasive meningococcal disease cases reported to the National Notifiable Diseases Surveillance System compared with laboratory confirmed data from the Australian Meningococcal Surveillance Programme, Australia, 2016**

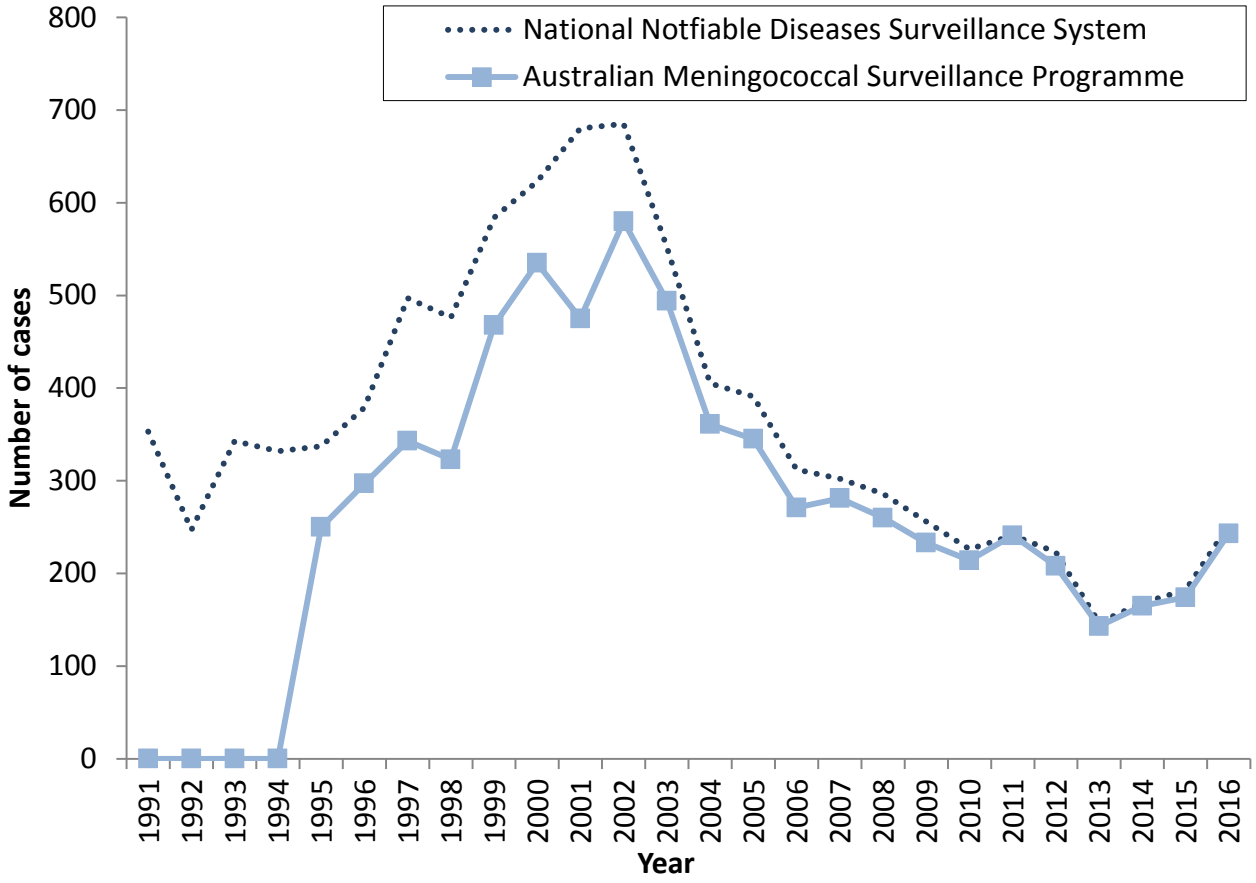



**Table 1: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2016, by quarter**

| Serogroup | 01 January - 31 March | 01 April - 30 June | 01 July – 30 September | 01 October - 31 December | 2016 Total |
|---|---|---|---|---|---|
| B | 19 | 18 | 28 | 22 | 87 |
| C | 2 | 0 | 0 | 1 | 3 |
| Y | 4 | 8 | 10 | 18 | 40 |
| W | 14 | 17 | 36 | 40 | 107 |
| NG | 2 | 0 | 1 | 0 | 3 |
| ND | 1 | 0 | 1 | 1 | 3 |
| Total | 42 | 43 | 76 | 82 | 243 |

NG: non groupable.
ND: not determined.

**Table 2: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2016, by state or territory and serogroup**

| State or territory | Serogroup | | | | | | Total |
|---|---|---|---|---|---|---|---|
| | B | C | Y | W | NG | ND | |
| ACT | 1 | 0 | 0 | 1 | 0 | 0 | 2 |
| NSW | 25 | 2 | 15 | 23 | 3 | 1 | 69 |
| NT | 2 | 0 | 0 | 0 | 0 | 0 | 2 |
| Qld | 15 | 0 | 13 | 14 | 0 | 0 | 42 |
| SA | 22 | 0 | 0 | 4 | 0 | 0 | 26 |
| Tas. | 0 | 0 | 1 | 4 | 0 | 0 | 5 |
| Vic. | 17 | 1 | 9 | 47 | 0 | 2 | 76 |
| WA | 5 | 0 | 2 | 14 | 0 | 0 | 21 |
| **Australia** | **87** | **3** | **40** | **107** | **3** | **3** | **243** |
| Proportion of all IMD cases | 35.8% | 1.2% | 16.5% | 44.0% | 1.2% | 1.2% | |

NG: non groupable
ND: not determined

**Table 3: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by age and serogroup**

| Serogroup | Age group | | | | | | | | | Total |
|---|---|---|---|---|---|---|---|---|---|---|
| | <1 | 1 to 4 | 5 to 9 | 10-14 | 15-19 | 20-24 | 25-44 | 45-64 | 65+ | |
| **B** | 14 | 11 | 3 | 1 | 14 | 18 | 15 | 6 | 5 | 87 |
| **C** | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 1 | 0 | 3 |
| **Y** | 2 | 1 | 1 | 0 | 5 | 4 | 2 | 10 | 15 | 40 |
| **W** | 8 | 11 | 3 | 0 | 13 | 7 | 16 | 19 | 30 | 107 |
| **NG** | 0 | 2 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 3 |
| **ND** | 0 | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 3 |
| **Total** | 24 | 26 | 7 | 1 | 34 | 30 | 35 | 36 | 50 | 243 |
| **%B of within age group** | 58.3 | 42.3 | 42.9 | 100.0 | 41.2 | 60.0 | 42.9 | 16.7 | 10.0 | |

NG: non groupable
ND: not determined

for this age group in any year. Between 2003 and 2014, the proportion of IMD that occurred in children aged less than 5 years ranged from 28% to 36% of cases. A secondary disease peak has also been observed in previous years amongst adolescents aged 15–19 years. Of the total cases of IMD in 2016, 14% (34/243) were in those aged 15–19 years, which was less than the proportion reported in the period 2006 to 2011 (16–20%), and 2013–2015 (17–20%).

## Anatomical site of samples for laboratory confirmed cases

In 2016, diagnosis was made by a positive culture in 78% (189/243) of cases and 22% (54/243) of cases were confirmed by NAAT testing alone (Table 4).

There were 45 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone or with a positive blood sample. There

**Table 4: Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by anatomical source and method of confirmation**

| Specimen type | Isolate of NM* | PCR positive** | Total |
|---|---|---|---|
| Blood | 162 | 26 | 188 |
| CSF +/- Blood | 17 | 28 | 45 |
| Other | 10 | 0 | 10 |
| Total | 189 | 54 | 243 |

* NM: *Neisseria meningitidis*
** NAAT: nucleic acid amplification testing.

were 188 diagnoses of septicaemia based on cultures or NAAT examination from blood samples alone (Table 4). There were 8 IMD diagnoses by positive joint fluid culture, 1 IMD diagnosis by positive tissue culture, and 1 IMD diagnosis by positive abscess culture.

## Serogroup data

*Number and proportions of cases of serogroup B, C, Y, W invasive meningococcal disease*

The serogroup was able to be determined for 237 of 243 laboratory-confirmed cases of IMD (98%) in 2016 (Tables 2 and 3). The overall decrease in IMD cases since 2002 was initially predominantly due to a reduction in the number of cases of IMD caused by serogroup C from 2003 to 2007 following the introduction of the serogroup C vaccine. After 2009, a decline in the numbers IMD cases caused by serogroup B was reported, from 194 cases in 2009 to 104 cases in 2013. In 2014, there was an increase in the numbers of IMD cases caused by serogroup B (n=129), however, in 2015 the numbers of IMD cases caused by serogroup B was similar to 2013. In the years 2006–2012 the proportion of IMD cases caused by serogroup B was 84%–88%, in 2013–2014 it was 75–80%, and in 2015 it was 64%. The number and proportion of IMD cases caused by serogroup B declined further in 2016 to the lowest number (n=87) and proportion of total IMD (36%) reported by the AMSP. The number of IMD cases caused by serogroup C (3 cases) in 2016 was similar to the previous year 2015 (2 cases), which was the lowest total reported by the AMSP. Since 2014, the rise in the total number of IMD cases has been due to a rise in the number of cases of IMD caused by serogroup W and serogroup Y (Figure 2).

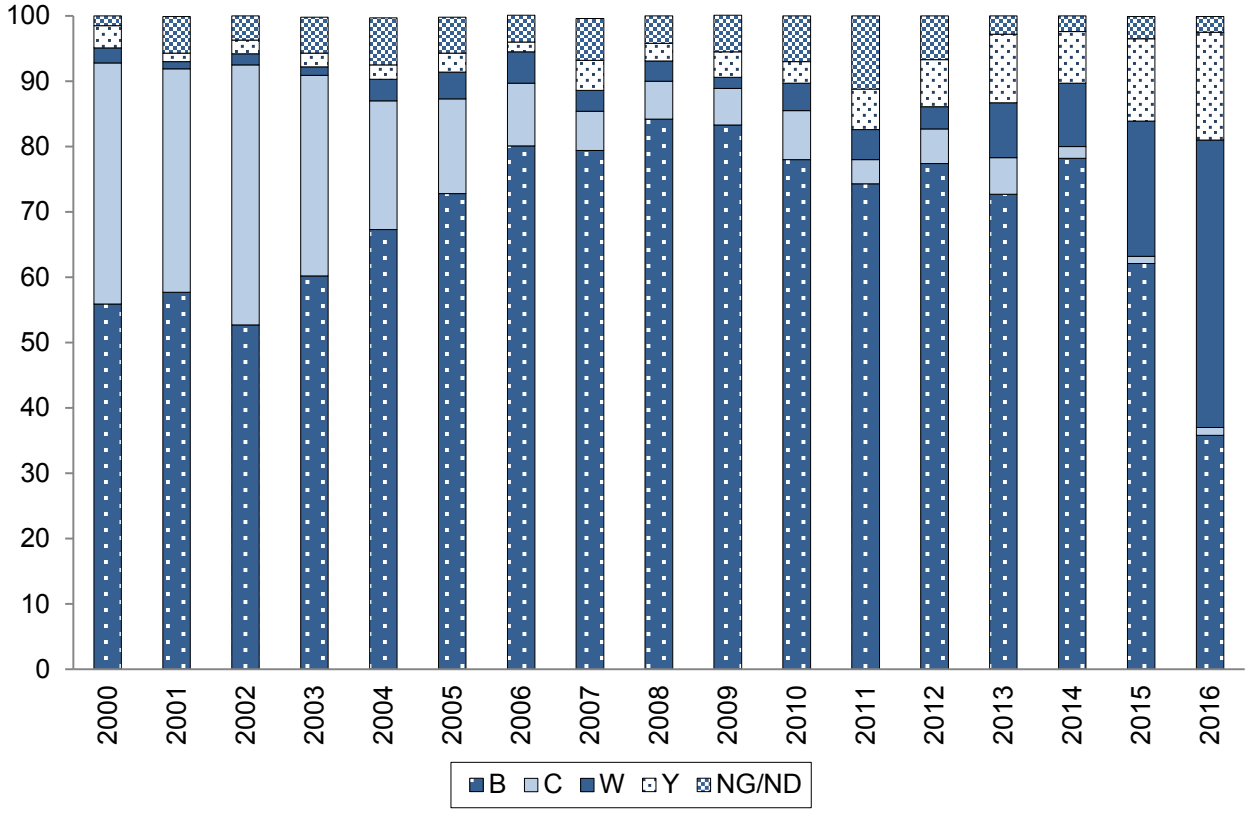
The number and proportion of cases of IMD caused by serogroup W in 2016 (107 cases, 44% of total IMD) was the highest reported by the AMSP, and was almost triple the number of cases reported in 2015 (36 cases), and a ten-fold increase in the average number of annual IMD cases caused by serogroup W reported before 2015. Prior to 2016, the proportion of cases of IMD caused by serogroup W ranged from 1–5% in the period 1997–2012, 8–10% in 2013–2014, to 21% of the total cases of IMD in 2015.

The number and proportion of cases of IMD caused by serogroup Y in 2016 (n=40, 17% of total IMD) was also the highest reported by the AMSP, almost double the number of IMD cases reported in the previous year (22 cases), and a four-fold increase in the average number of annual serogroup Y cases reported before 2015. Prior to 2016, the proportion of cases of IMD caused by serogroup Y ranged from 1–5% in the period 1997–2010, 6–11% in 2011–2014, and was 13% of the total cases of IMD in 2015.

Of the 107 cases of IMD cases caused by serogroup W in 2016, 47 cases (44%) were reported from Victoria, where serogroup W represented 62% (47/76) of cases, 23 cases (22%) were reported in New South Wales, representing 33% (23/69) cases and 14 cases (13%) were reported in Western Australia, representing 67% (14/21) cases. This is in contrast to previous years where, serogroup B has been predominant in these states. Serogroup W was reported in all jurisdictions, except in the Northern Territory in 2016.

Of the 40 cases of IMD caused by serogroup Y in 2016, 15 cases (38%) were reported from New South Wales, where this serogroup represented 22% (15/69 cases) of cases. Thirteencases (33%) were reported in Queensland, representing 31%

**Figure 2: Proportion of serogroups of laboratory-confirmed invasive meningococcal disease, Australia, by year**




(13/42 cases) of cases. Serogroup Y was reported in all jurisdictions except in the Northern Territory, Australian Capital Territory, and South Australia. Serogroup B was reported in all jurisdictions except in Tasmania, and continued to be the predominant serogroup amongst IMD cases reported in South Australia.

In 2016, the predominant serogroup for children less than 5 years continued to be serogroup B, however the proportion was the lowest since 2000. (Table 3, Figure 3). In young adults aged 20–24 years, IMD caused by serogroup B was also the predominant serogroup, however this proportion was lower than in 2014 (83%), 2007–2010 and 2012 (72-88%), but similar to 2011, 2013, and 2015 (62-67%). The proportion of IMD caused by serogroup B in the all other age groups was less compared with previous years, due to the large increase in the number of IMD cases caused by serogroup W and Y in these age groups.

In 2016, there was an increase in the number of IMD caused by serogroup W across all age groups except those aged 10–14 years. For those aged more than 45 years, IMD caused by serogroup W was the predominant serogroup (49/83 cases or 57%). This is in contrast with previous years, where serogroup Y was predominant for this age group. There was also a large increase in the number and proportion of IMD cases caused by serogroup W (16/35 cases, 46%) in those aged 25–44 years, compared with previous years, where serogroup B was predominant (68–87% since 2007).

## Genotyping

In 2016, genotyping results were available for 93% (225/243) of IMD cases (Tables 5 and 6). The predominant *por*A genotype for IMD cases caused by serogroup B was P1.7-2,4 (25 cases, 33% of serogroup B that were typeable), which is similar to 2015 (Figure 4). The predominant *por*A genotype for serogroup Y IMD cases was P1.5-1,10-1 (34 cases, 85% of serogroup Y IMD cases that were typeable).

**Figure 3: Number of serogroups B, Y and W cases of laboratory-confirmed invasive meningococcal disease, Australia, 2016, by age**

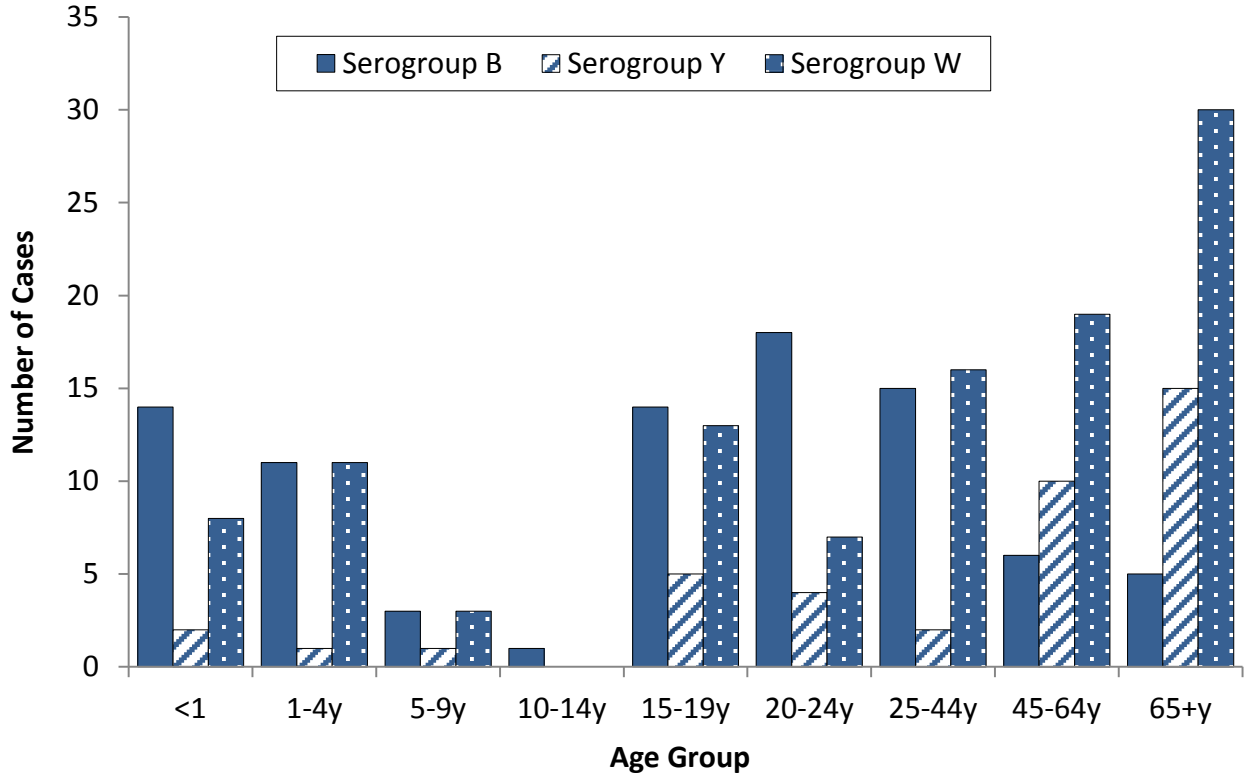



**Figure 4: Number of porA genotypes for serogroup B in laboratory- confirmed cases of invasive meningococcal disease Australia, 2016**

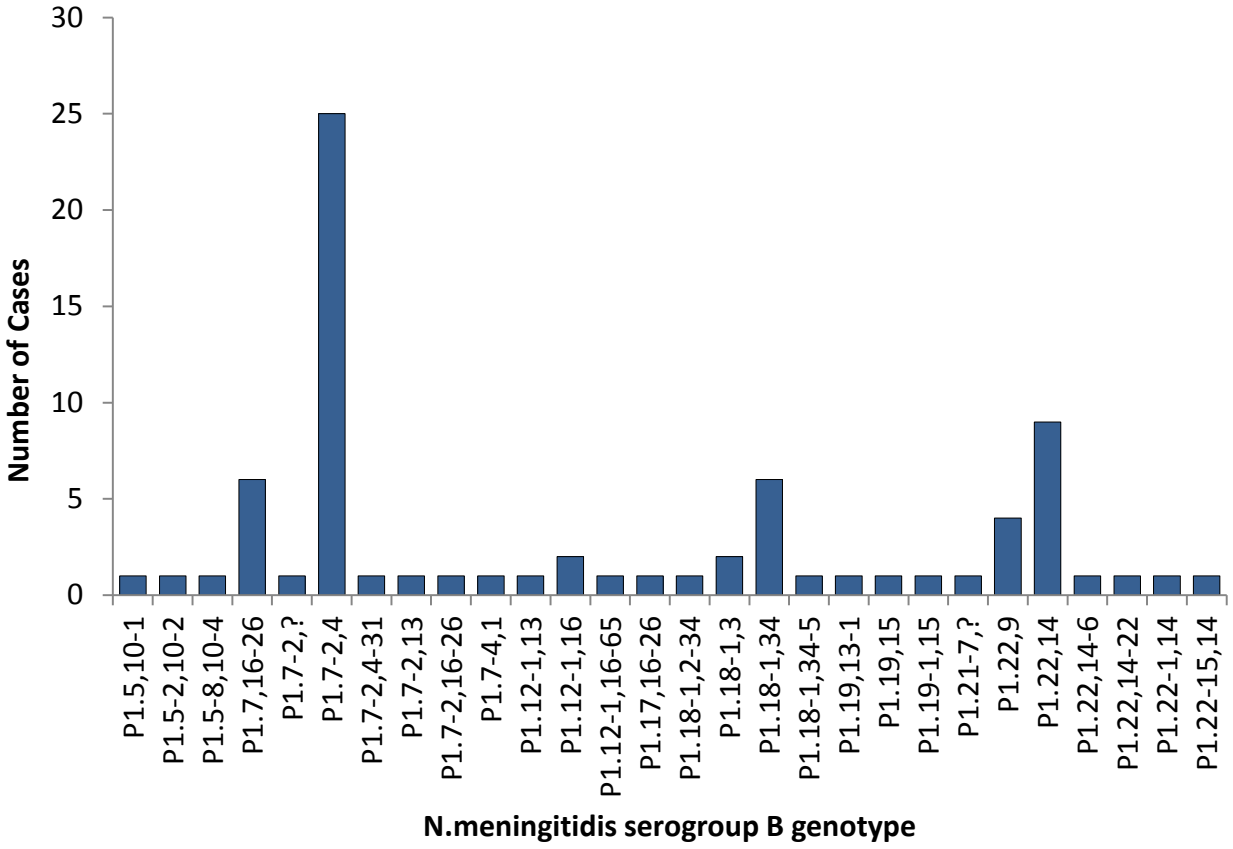


**Table 5: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by *por*A genotype**

| 2016 AMSP | No. PER SEROGROUP | | | | | |
|---|---|---|---|---|---|---|
| **GenotypeporA** | **B** | **C** | **Y** | **W135** | **ND** | **TOTAL** |
| P1.5,2 | 0 | 2 | 0 | 96 | 2 | **100** |
| P1.5,2-59 | 0 | 0 | 0 | 1 | 0 | **1** |
| P1.5,10-1 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.5-1,2-2 | 0 | 0 | 2 | 0 | 0 | **2** |
| P1.5-1,10-1 | 0 | 0 | 34 | 0 | 0 | **34** |
| P1.5-1,10-4 | 0 | 0 | 0 | 2 | 0 | **2** |
| P1.5-2,10-1 | 0 | 0 | 3 | 0 | 0 | **3** |
| P1.5-2,10-2 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.5-2,10-29 | 0 | 0 | 1 | 0 | 0 | **1** |
| P1.5-8,10-4 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.5-9,2 | 0 | 0 | 0 | 1 | 0 | **1** |
| P1.5-11,2 | 0 | 0 | 0 | 1 | 0 | **1** |
| P1.7,16-26 | 6 | 0 | 0 | 0 | 1 | **7** |
| P1.7-2,? | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.7-2,4 | 25 | 0 | 0 | 0 | 0 | **25** |
| P1.7-2,4-31 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.7-2,13 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.7-2,16-26 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.7-4,1 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.12-1,13 | 1 | 1 | 0 | 0 | 0 | **2** |
| P1.12-1,16 | 2 | 0 | 0 | 0 | 0 | **2** |
| P1.12-1,16-65 | 1 | 0 | 0 | 0 | 0 | **1** |
| | | | | | | |
| P1.17,16-26 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.18-1,2-34 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.18-1,3 | 2 | 0 | 0 | 3 | 0 | **5** |
| P1.18-1,34 | 6 | 0 | 0 | 0 | 0 | **6** |
| P1.18-1,34-5 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.19,13-1 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.19,15 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.19-1,15 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.21-7,? | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.22,9 | 4 | 0 | 0 | 0 | 0 | **4** |
| P1.22,14 | 9 | 0 | 0 | 0 | 0 | **9** |
| P1.22,14-6 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.22,14-22 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.22-1,14 | 1 | 0 | 0 | 0 | 0 | **1** |
| P1.22-15,14 | 1 | 0 | 0 | 0 | 0 | **1** |
| TOTAL | 75 | 3 | 40 | 104 | 3 | **225** |

**Table 6: Distribution of *porA* genotype laboratory-confirmed cases of invasive meningococcal disease, Australia, 2016, by state or territory**

| Genotype *porA* | No. PER SEROGROUP PER STATE | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | NSW | QLD | VIC | SA | WA | ACT | TAS | NT |
| P1.5,2 | 21W, 1C | 12W | 44W, 1C, 1NG | 4W | 14W | 1W | 1W | |
| P1.5,2-59 | | | 1W | | | | | |
| P1.5,10-1 | | | | 1B | | | | |
| P1.5-1,2-2 | | 1Y | | 1Y | | | | |
| P1.5-1,10-1 | 15Y | 10Y | 8Y | | 1Y | | | |
| P1.5-1,10-4 | | 2W | | | | | | |
| P1.5-2,10-1 | | 2Y | 1Y | | | | | |
| P1.5-2,10-2 | | 1B | | | | | | |
| P1.5-2,10-29 | | | | | | | 1Y | |
| P1.5-8,10-4 | | | 1B | | | | | |
| P1.5-9,2 | 1W | | | | | | | |
| P1.5-11,2 | | | 1W | | | | | |
| P1.7,16-26 | | 3B | 4B | | | | | |
| P1.7-2,? | 1B | | | | | | | |
| P1.7-2,4 | 3B | 3B | 3B | 15B | 1B | | | |
| P1.7-2,4-31 | | | 1B | | | | | |
| P1.7-2,13 | | | | | 1B | | | |
| P1.7-2,16-26 | | 1B | | | | | | |
| P1.7-4,1 | | | | | 1B | | | |
| P1.12-1,13 | 1C | | | | | | | 1B |
| P1.12-1,16 | 2B | | | | | | | |
| P1.12-1,16-65 | | 1B | | | | | | |
| P1.17,16-26 | 1B | | | | | | | |
| P1.18-1,2-34 | | | 1B | | | | | |
| P1.18-1,3 | | | 1B | | | | 3W | 1B |
| P1.18-1,34 | 4B | 1B | | | 1B | | | |
| P1.18-1,34-5 | | | 1B | | | | | |
| P1.19,13-1 | | | | 1B | | | | |
| P1.19,15 | | 1B | | | | | | |
| P1.19-1,15 | 1B | | | | | | | |
| P1.21-7,? | 1B | | | | | | | |
| P1.22,9 | 2B | | 1B | 1B | | | | |
| P1.22,14 | 4B | 2B | 3B | | | | | |
| P1.22,14-6 | | 1B | | | | | | |
| P1.22,14-22 | 1B | | | | | | | |
| P1.22-1,14 | 1B | | | | | | | |
| P1.22-15,14 | | | 1B | | | | | |

There were 105 serogroup W IMD cases that were able to be genotyped, 97 of these (92%), had the *por*A antigen encoding gene type P1.5,2. Multilocus sequence typing showed that, of these 72% (70/97) were sequence type (ST)-11 — the same strain type as the hypervirulent serogroup W strain reported in the UK and South America since 2009[4, 5] (Table 7).

### Antibiotic susceptibility testing

Antimicrobial susceptibility testing was able to be performed for 77% (189/243) of the IMD cases in 2016. Of these, 6% (11/189) were resistant to penicillin (MIC ≥ 1 mg/L), the highest number and proportion of isolates with penicillin resistance reported by the AMSP. Only 4% of isolates tested were fully susceptible to penicillin (MIC ≤0.03 mg/L), and 90% (170/189) of isolates were less sensitive to penicillin (MIC=0.06–0.5 mg/L). Of the isolates that were resistant to penicillin, all were serogroup W, and 9/11 were P1.5,2: ST-11, one was P.1.5,2: ST-12351, and one was P.1.5,2-59: ST-11. This represented 11% penicillin resistance in all serogroup W isolates available for testing (n=99). There was one isolate that had an elevated MIC to ceftriaxone (MIC=0.125 mg/L), the highest recorded by the AMSP. This isolate was W:P1.5,2, ST-11 and was less sensitive to penicillin. All isolates tested were susceptible to rifampicin and ciprofloxacin.

## Discussion

In 2016, there were 243 cases of laboratory confirmed IMD, representing 96% of the number of notifications to the NNDSS.[2] The number of laboratory-confirmed IMD in 2016 represents a 40% increase cases compared with the previous year. The number of IMD cases has been steadily rising since the nadir in 2013, however the number of cases reported this year remains less than half of the peak number of IMD cases reported in Australia in 2002 (n=684). The introduction of the serogroup C vaccine to the national immunisation schedule in 2003 has resulted in a very large and sustained reduction in the number and proportion of serogroup C IMD cases in this country, and in 2015 the number and proportion of IMD cases caused by serogroup C was the lowest ever reported by the AMSP. In early 2014, a recombinant multicomponent meningococcal B vaccine became available in Australia. [6] This vaccine is not on the national immunisation program but is available for purchase privately. Therefore uptake is elective and the impact of its introduction is yet to be determined in this country.

A number of changes in IMD epidemiology were observed in Australia in 2016. There was a notable increase in the number and proportion of IMD cases caused by serogroup W and serogroup Y, and a marked decrease in the number and proportion of cases caused by serogroup B. The incidence of serogroup W and serogroup Y was the highest ever reported by the AMSP.

In addition, as reported by the AMSP in 2015, the primary peak of IMD was observed in adults aged 45 years or older and was due to the increased number of IMD cases caused by serogroup W and serogroup Y in this age group. However these serogroups also increased in frequency in all age groups in 2016. Secondary disease peaks were observed in those aged less than 5 years, and in adolescents aged 15–19 years.

Serogroup W represented 44% of all laboratory-confirmed IMD cases in 2016, with the highest proportions in Victoria (44%), Queensland (33%), New South Wales (22%) and Western Australia (13%). Typing of these isolates found that the predominant circulating strain of serogroup W, had the *por*A antigen encoding gene type P1.5,2 and was sequence type (ST)-11. This is same genotype as the hypervirulent serogroup W strain that emerged in the United Kingdom and South America in 2009[4, 7] and has spread to now account for 25% of IMD in the UK in 2014/15, and 59% of all cases in Chile in 2012. This serogroup W strain is now considered endemic in these regions and is associated with atypical presentations, more severe clinical disease and a higher case fatality rate.[7] The initial increase in serogroup W in these regions, as is now being demonstrated in Australia, was seen in older adults, but was subsequently reported in all age groups, particularly in adolescents and

**Table 7: Laboratory-confirmed cases of serogroup W IMD, Australia, 2016, by sequence type (ST)**

| Sequence Type | W Genotype | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | P1.5,2 | P1.5,2-59 | P1.5-1,10-4 | P1.5-9,2 | P1.5-11,2 | P1.18-1,3 | Not typeable | Total |
| ST11 | 70 | 1 | 0 | 0 | 0 | 0 | 0 | **71** |
| ST22 | 0 | 0 | 0 | 0 | 0 | 3 | 0 | **3** |
| ST23 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | **2** |
| ST1287 | 7 | 0 | 0 | 0 | 0 | 0 | 0 | **7** |
| ST3298 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| ST8345 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| ST8857 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | **1** |
| ST10651 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | **1** |
| ST12351 | 8 | 0 | 0 | 0 | 0 | 0 | 0 | **8** |
| ST12590 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | **1** |
| Not typeable | 9 | 0 | 0 | 0 | 0 | 0 | 2 | **11** |
| **Total** | **97** | **1** | **2** | **1** | **1** | **3** | **2** | **107** |

infants[8]. In response, vaccination programs have been implemented in both the United Kingdom and in Chile [4, 9]

An increase in IMD cases caused by serogroup Y was also observed in the eastern states of New South Wales, Queensland and Victoria in 2016. The predominant serogroup Y genotype (85%, 34/40) was P1.5-1,10-1, which increased in number and proportion compared with 2015 (70%, 14/20) and 2014 (50%,6/12) whereas in previous years the serogroup Y genotype distribution was more heterogeneous. The emergence of serogroup Y and genotype Y:P1.5-1,10-1 has also been reported recently in Europe [10]. The phenotypic and genotypic characterization of the serogroup Y isolates is ongoing by the NNN.

Antimicrobial susceptibility testing of IMD isolates in 2016 showed an increase in penicillin resistance, the highest annual number and proportion recorded by the AMSP. The incidence of penicillin resistance in *N. meningitidis* in Australia has been less than 1% annually of IMD isolates tested in 1996-2014, rising to 3% in 2015, and 6% in 2016. The proportion of IMD isolates with penicillin MIC values in the less sensitive category has been increasing in recent years. These proportions ranged from 62–75% in 1996–2006; 67%–79% in 2007–2009; 78%-88% in 2010–2015, and in 2016, was 90%. In 2016, all isolates resistant to penicillin were serogroup W, representing 11% of all serogroup W isolates tested. Additionally, there was another serogroup W isolate with the highest ceftriaxone MIC recorded (0.125mg/L) by the AMSP. All IMD isolates were susceptible to rifampicin and ciprofloxacin.

The increase in IMD cases caused by serogroup W and serogroup Y, and the observed increase in antimicrobial resistance in serogroup W isolates are of significant concern. The NNN is continuing to lead further investigations with the Department of Health and the CDNA in to these observed changes and is closely monitoring the phenotypic and genotypic features of *N. meningitidis* causing IMD in Australia. Additional investigations including whole genome sequencing are in place to enhance IMD surveillance. The AMSP data are used for informing treatment guidelines and disease prevention strategies; and to monitor the effect of interventions.

## Acknowledgements

Meningococcal isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these isolates 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. The Australian Government Department of Health provided funding for the National Neisseria Network.

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