INVASIVE MENINGOCOCCAL DISEASE

IN

QUEENSLAND 2005

ISBN: 1921021586

© The State of Queensland, Queensland Health, 2005.

Copyright protects this publication. However, Queensland Health has no objection to this material being reproduced with acknowledgement, except for commercial purposes. Permission to reproduce for commercial purposes should be sought from the Policy and Quality Office, Queensland Health, GPO Box 48, Brisbane QLD 4001.

Suggested Citation:

Slinko V, 2006. *Invasive Meningococcal Disease in Queensland 2005*. Communicable Diseases Unit, Queensland Health, Brisbane, Australia.

Acknowledgements:

I would like to acknowledge the staff of the Communicable Diseases Unit, particularly Amy Sweeny, Craig Davis, and Frances Birrell (Epidemiology), Jill Homewood and Heidi Carroll (Public Health registrars), Bruce Imhoff and Kym Columbine (Information Systems & Data Management) and the team at Queensland Health Immunisation Programme (QHIP); Helen Smith from Meningococcal Reference Laboratory at Queensland Health Pathology & Scientific Services(QHPSS); National Centre for Epidemiology & Population Health (NCEPH) supervisors, Scott Cameron and Mohamed Patel; Brad McCall (Brisbane Southside Public Health Unit) and the staff of the Population Health Units who collected the enhanced surveillance data.

Contents

Figures

Tables

Executive Summary

Meningococcal disease is an uncommon but important public health problem in Australia. In early 2003, a federally funded conjugate meningococcal C vaccination became part of the immunisation schedule in Queensland. Free vaccination has been offered progressively since 2003 for those born on or after 1 January, 1984 through a school based program, general practitioners and local governments. The vaccine will remain free until June 2007.

In 2004, there were some preliminary indications of the positive effects of the vaccination program. This report offers a further opportunity to assess the effect that vaccination has had on invasive meningococcal disease (IMD) in Queensland.

In 2005, Queensland recorded the lowest number of cases (62) and lowest incidence of IMD (1.6/100,000 residents) since state wide surveillance began in 1993. This represents the largest decrease in numbers of any of the states and territories of Australia. Known serogroup C disease also had the lowest incidence. This decrease is likely to be the result of the vaccination program, in combination with consistent reporting of IMD cases and aggressive public health management of cases and contacts. While these results indicate the effectiveness of the vaccination program, is natural fluctuation in IMD and continued surveillance will be needed to monitor the disease.

Surveillance data for 2005 indicates that the highest incidence of IMD remains in those under five (particularly infants) with the next highest incidence in the 10-14 year age group. In those eligible for vaccine 90% of IMD was due to serogroup B, with the four cases of serogroup C occurring in unvaccinated young people.

Just over one-third of cases were associated with childcare, school or learning institutions but no cases were linked. This may reflect the prompt public health action in Queensland.

An increased incidence of IMD remains for Indigenous people compared with non-Indigenous. However, this increased risk of disease was not statistically significant in the under-five years age group. There were no deaths in Indigenous people in 2005.

IMD appears to be a sporadic disease with considerable heterogeneity in serotyping and serosubtyping. With the advent of nucleic acid amplification techniques there remained 12 cases that were unable to be serotyped or serosubtyped and no antibiotic sensitivities available. Generally, sensitivity to penicillin in Queensland is hovering around 60 % which is lower than the figures reported for Australia (32%).

Case fatality from IMD in 2005 (4.8%) was within the range of the previous five years in Queensland and lower that that reported for Australia (9.2%) for

the year. Serogroup C carried a higher risk of mortality than other known serogroups of IMD.

Management of IMD remains acceptable, however there may be room for improvement with better education of clinicians about the new *Public Health Act, 2005* which requires IMD to be reported on clinical suspicion.

1. Introduction

Meningococcal disease is an uncommon but important public health problem in Australia. The invasive form of the disease is a serious illness with a variable case fatality rate in industrialised countries as low as 7% for meningitis and as high as 19% for septicaemia(1). Those known to be at highest risk of the disease are children less than five years of age (particularly infants), followed by adolescents and young adults.

The bacterium (*Neisseria meningitidis*) is usually carried asymptomatically in the back of the throat and nose. However, only a small number of people become infected and develop invasive disease which appears most often as meningitis and/or septicaemia. Other localised infections such as arthritis, pneumonia and conjunctivitis are also possible.

The factors leading to development of invasive meningococcal disease (IMD) are poorly understood, but risk factors include smoking, exposure to tobacco smoke and living in crowded conditions (2).

There are 13 known serogroups of *Neisseria meningitidis* distinguished by differences in surface polysaccharides of the outer membrane capsule. The most common serogroups in Australia have been serogroups B and C. Further serotyping and serosubtyping of the organism distinguish differences in outer membrane proteins (3).

The prompt diagnosis and emergency treatment of IMD have been the basis of clinical management of the disease which has considerably reduced case fatality. Population health units play an important role in tracing contacts of the patient that may have transmitted the disease, and administering chemoprophylaxis to reduce further transmission to others. Queensland guidelines on the management of IMD have been developed, and are found in the Queensland Health Guidelines for the Control of Communicable Diseases in the Community (3rd edition, 2005). The national 'Guidelines for the early clinical and public health management of meningococcal disease in Australia' can be found at http://www.cda.gov.au/pubs/other/mening.htm.

Under the Queensland *Public Health Act, 2005*, IMD is immediately notifiable by telephone to Queensland Health by both laboratories and clinicians. The data are maintained on the Notifiable Conditions Systems database (NOCS) and have been collated since 1993. In 1999, enhanced surveillance for IMD was established. Enhanced surveillance is conducted by communicable diseases staff of the population health units, who also coordinate public health responses. Queensland Health has published several annual reports in the years between 1999 and 2004, (4-9).

In early 2003, conjugate meningococcal C vaccination became part of the immunisation schedule in Queensland and the National Meningococcal C Vaccination Program, a joint initiative of the Australian Government and

Queensland Health commenced. Under the program, anyone over 12 months of age and born on or after 1 January 1984 is eligible for a single dose of the vaccine.

The program was introduced to:

- reduce the illness and death in the population at highest risk of meningococcal disease
- induce long term immunity in those who are vaccinated
- reduce the population incidence of disease through reduced carrier rates of meningococcal group C.

In 2003, all secondary school students in Queensland were offered vaccination through a school program. Young people aged 15-19 years who were not attending school and all children aged 1-5 years were offered access to meningococcal C vaccine through their general practitioner or other participating vaccination service.

Throughout 2004, all primary school and Year 8 students were offered the vaccination. Children and young people aged 1-5 years and 15-19 years (who were not attending school) continued to be eligible to receive free vaccine.

In 2005 the programme was extended and any remaining people aged more than one year old and born on or after 1 January1984, are eligible to receive free vaccine through their general practitioner or usual vaccination service providers until June 2007.

In 2004, there were some preliminary indications of the vaccination program's effect. There was an apparent reduction in the incidence of IMD from the previous three years but it was not statistically significant when compared to the previous 10 years. The data from enhanced surveillance for 2005 offers a further opportunity to assess the effect that the vaccination program has had on IMD in Queensland.

With this aim of exploring the effects of vaccination to date, this report's objectives are:

- describe the epidemiology of IMD in Queensland in 2005
- describe trends of the disease with respect to age, gender, indigenous status, seasonal variation, clinical presentation, risk factors and laboratory data
- examine the effect of the meningococcal C vaccination program that commenced in 2003
- describe risk factors for dying identified in the period since enhanced surveillance began
- describe the timeliness of management.

2. Methods

2.1 Data

Data for analysis were obtained from the Queensland Notifiable Conditions Systems (NOCS) database for the year, 2005. Data for the previous years were obtained from previous reports. All data are collected by population health units using a standardised case report form. Population health units obtain surveillance data from a range of sources including relatives of the case, the case and medical practitioners.

Analysis was performed in Excel, Epi-Info Version 3.3.2, and Stata Intercooled 9 using chi square and Fisher's exact tests.

2.2 Case definition

The notification criteria for IMD were updated on 6 August 2005. The change was to make the case definition clearer and more workable, though it would be unlikely to change case detection.

Before 6 August 2005

The criteria until 6 August 2005 are taken from the 2004 report:

Confirmed case:

- isolation of *Neisseria meningitidis* from a normally sterile site.
- a clinically compatible illness with at least one of the following:
    1. Detection of *N. meningitidis* in a specimen from a normally sterile site by nucleic acid testing
    2. Detection of Gram-negative diplococci in Gram stain of specimen from a normally sterile site or from a suspicious skin lesion
    3. High titre IgM or significant rise in IgM or IgG titres to outer membrane protein antigens of *N. meningitidis*
    4. Positive polysaccharide antigen test in cerebrospinal fluid with other laboratory parameters consistent with meningitis.

Probable case:

- a clinically compatible illness and :
    1. The absence of evidence for other causes of clinical symptoms

    And either

    2. Clinically compatible disease including haemorrhagic rash

    Or

    3. Clinically compatible disease AND close contact with a confirmed case within the previous 60 days.

After 6 August 2005

After 6 August 2005, the notification criteria for confirmed and probable cases are taken from the Queensland Health Guidelines for the Control of Communicable Diseases in the Community (3rd edition), 2005:

Confirmed case: a confirmed case requires either

1. laboratory definitive evidence

OR

2. laboratory suggestive evidence AND clinical evidence.

Probable case: a probable case requires clinical evidence only, but will not be reported to NOCS unless there is no evidence of other diseases.

Clinical evidence, laboratory definitive evidence and laboratory suggestive evidence are outlined below:

Clinical evidence:

Disease which in the opinion of the treating clinician is compatible with invasive meningococcal disease; eg meningitis, septicaemia or other invasive disease (eg orbital cellulitis, septic arthritis)*#

*Although not meeting the definitions of a confirmed case, meningococcal infection of the conjunctiva is considered an indication for public health action because of the risk of invasive disease in the patient and secondary invasive disease.

#Other localised infections eg pneumonia can occur, but are rare and are not included in the definition of invasive meningococcal disease. *N. meningitidis* is also commonly isolated in sputum, throat swabs, genital swabs and from other non-sterile sites. Without other evidence of invasive disease, these isolates are not notifiable and no public health action is indicated.

A probable case requires:

1. The absence of evidence of other causes of clinical symptoms (but do not wait for other causes of clinical symptoms to be eliminated before notifying urgently to the population health unit if either 2 or 3 is satisfied)
   and either
2. Clinically compatible disease including haemorrhagic rash
   or
3. Clinically compatible disease AND close contact with a confirmed case within
   the previous 60 days.

Laboratory definitive evidence:

1. Isolation of *N. meningitidis* from a normally sterile site.
   or
2. Detection of *N. meningitidis* in a specimen from a normally sterile site by nucleic acid testing

Laboratory suggestive evidence:

1. Detection of Gram-negative diplococci in gram stain of specimen from a normally sterile site or from a suspicious skin lesion
   or
2. High titre IgM or significant rise in IgM or IgG titres to outer membrane protein antigens of *N. meningitidis*
   or
3. Positive polysaccharide antigen test in cerebrospinal fluid with other laboratory parameters consistent with meningitis. However false negatives are common especially group B.

2.3 Case ascertainment

Nucleic acid testing /polymerase chain reaction (PCR) has been used in Queensland since 1999, and the IgM test was introduced in 2000.

Cases for 2005 are classified according to date of onset being in 2005.

2.4 Rate calculations

Incidence for various years has been calculated using Australian Standard Geographical Classification (ASGC) figures as denominators:

- 2005: 2005 Estimated Resident Populations (ERP's) by Statistical Local Area (SLA) tables, Australian Bureau of Statistics (ABS), catalogue no. 3235.3.55.001, area grouping added by Queensland Health's Health Information Branch (HIB) and based on 2001 census counts
- 2002- 2004 used the same source as above for the relevant year;
- 2001 used 2001: ERP's by SLA tables, ABS, catalogue no. 3235.3 (Revised June 2001)
- 1999-2000: for relevant year ERP's by SLA tables, catalogue no. 3235.3
- 1994-1998: for relevant year ERP's by SLA tables, catalogue no. 3217.3.

2.5 Indigenous data

Statistics for Indigenous incidence are calculated using ABS catalogue no. 3238.0.55.002 Experimental Projections of Aboriginal and Torres Strait Islander Australians, ATSIC Regions, 2001-2009.

2.6 Antibiotic sensitivities

Antibiotic susceptibility is routinely done by Queensland Health Scientific Services (QHSS) on every meningococcal isolate. Data for this report were received from QHSS.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This is done by a national standardised agar plate dilution method.

With the advent of molecular diagnosis the percentage of cases with antibiotic susceptibility results has decreased.

(i) Penicillin

The following parameters are used to define the various levels of penicillin susceptibility/resistance (10):

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 (11).

(iii) Third generation cephalosporins

Breakpoints for ciprofloxacin:

Sensitive:	MIC <= 0.03 mg/l
Less Sensitive:	MIC 0.06 - 0.5 mg/l
Resistant:	MIC 1.0 – 2.0 mg/l

Breakpoints for ceftriaxone:

Sensitive:	MIC <= 0.03 mg/l
Less Sensitive:	MIC 0.06 - 0.125 mg/l
Resistant:	MIC >= 0.25 mg/l

(iii) Rifampicin

Breakpoints for rifampicin:

Sensitive:	MIC <= 0.5 mg/l
Resistant:	MIC >= 1.0 mg/l

3. Results

3.1 Incidence

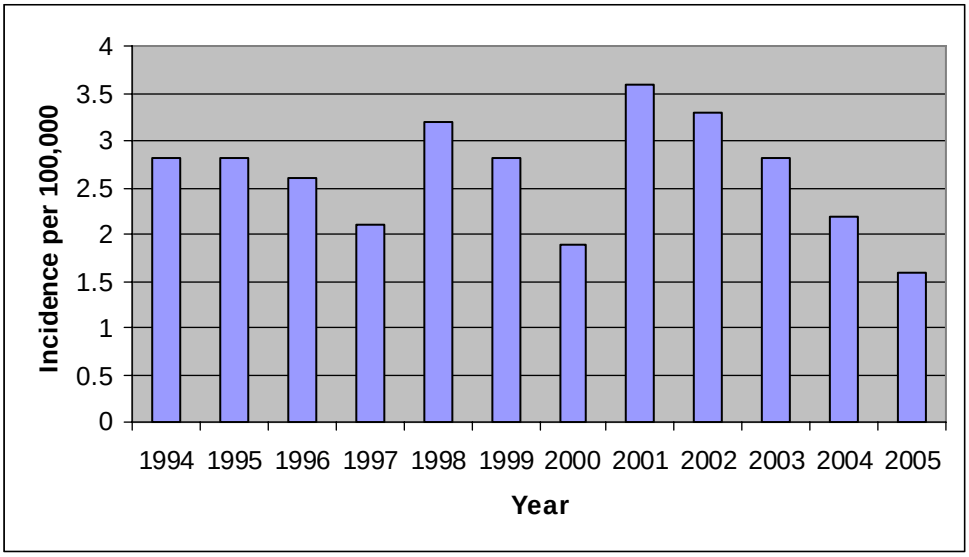
There were 62 cases of invasive meningococcal disease (IMD) notified in Queensland (Qld) in 2005. This is the lowest number of cases since Queensland-wide surveillance began in 1993.

There were also two cases of meningococcal conjunctivitis which are notifiable but did not fulfil the conditions of invasive disease and so were excluded from further analysis of IMD for 2005.

This represented an incidence of IMD of 1.6/100,000 Queensland residents (Figure 1). This reduction in incidence from that in the previous 11 years combined is statistically significant: the risk (relative risk: RR) of having IMD in 2005 was 0.6 times (95%CI 0.4-0.7) as likely compared with the years 1994-2004.

Of the 62 cases, 58 were laboratory confirmed (94%) and four were probable (6%). There were three deaths in 2005, which represents a case fatality rate of 4.8%.

Figure 1: Incidence of IMD per 100,000 population in Qld, 1994-2005



3.2 Age and gender distribution

In Queensland in 2005, 21 cases (34%) occurred in children under 5 years of age (including 9 cases (15%) in children < 1 year) (Figure 2). This was not statistically significantly different to the proportion of IMD in children under five for the period 1995-2004 (Table 1). Consistent with the previous years, infants (<1 year of age) had the highest incidence rate of IMD (Table 2).

In 2005, 10-14 yr olds exhibited the next highest incidence (2.9/100,000) with eight cases (12.9%) (Table 2). There were 13 cases (21%) in 40+ year olds with an incidence of 0.8/100,000.

Figure 2: IMD by age group in Qld, 2005

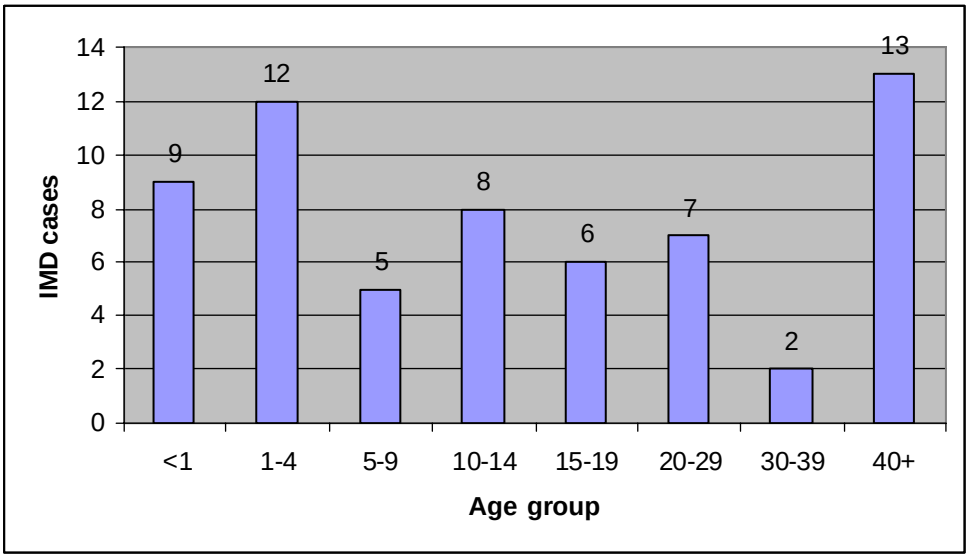


Table 1: Percent of IMD cases by age group and year, Qld, 1994-2005

Age Group (years)	94 % (95)	95 % (96)	96 % (89)	97 % (72)	98 % (108)	99 % (93)	00 % (66)	01 % (129)	02 % (124)	03 % (105)	04 % (84)	2005 % (n=62)
<1	14.7	13.5	12.4	15.3	13.9	11.8	9.1	10.9	12.1	13.3	10.7	14.5
1-4	21.1	24	22.5	36.1	23.1	22.6	21.2	18	12.9	10.5	16.7	19.4
5-9	3.2	4.2	12.4	1.4	7.4	11.8	10.6	12.5	8.9	6.7	4.8	8.1
10-14	7.4	3.1	5.6	9.7	6.5	3.2	7.6	4.7	4	9.5	7.1	12.9
15-19	23.2	21.9	20.2	18.1	18.5	17.2	18.1	18	22.6	17.1	29.8	9.7
20-29	12.6	14.6	13.5	9.7	12	15.1	18.1	19.5	12.9	21	14.3	11.3
30-39	3.2	6.3	2.2	2.8	3.7	2.2	3	3.1	9.7	9.5	6	3.2
40+	14.7	12.5	11.2	6.9	14.8	16.1	12.1	12.5	16.9	12.4	10.7	21
All ages	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**	**100**

Table 2: Age-specific incidence of IMD per 100,000 by year in Qld, 1994-2005

Age Group	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005
<1	30.8	28.6	24.2	24.2	33	24.2	14.8	28.2	33.5	29.5	18.9	17.4
1-4	10.3	11.8	10.3	13.4	12.9	10.8	7.2	12	8	5.5	6.9	5.9
5-9	1.2	1.7	4.6	0.4	3.3	4.6	1.6	6.1	4.2	2.6	1.5	1.9
10-14	2.8	1.2	2	2.8	2.8	1.2	2	2.3	1.9	3.6	2.1	2.8
15-19	9.3	8.8	7.6	5.5	8.4	6.7	5.3	8.7	10.5	6.7	9.1	2.2
20-29	2.4	2.8	2.4	1.4	2.6	2.8	1.9	4.9	3.1	4.2	2.2	1.3
30-39	0.6	1.2	0.4	0.4	0.8	0.4	0.4	0.7	2.2	1.8	0.9	0.3
40+	1	0.9	0.7	0.4	1.2	1.1	0.6	1	1.3	0.8	0.5	0.7
Total	**2.8**	**2.8**	**2.6**	**2.1**	**3.2**	**2.8**	**1.9**	**3.6**	**3.3**	**2.8**	**2.2**	**1.6**

The incidence rate of IMD for infants in the last five years has declined from 33.5 to 17.4/100,000 in 2005, a reduction of 48% (Table 2). Fairly steady reductions in incidence rates have also been observed in children 1-4 and 5-9 years old, and adults 20-29 years old over the last five years. In 2005, teenagers 15-19 years old had the lowest incidence rate since Queensland recording began (2.2 per 100,000). However, there has been considerable fluctuation in the incidence rate for IMD for this age group.

In 2005 there were 28 females (45%) and 34 males (55%) with IMD.

3.3 Indigenous status

Indigenous status was determined in all cases: six of 62 cases (9.7%) were Indigenous. The Indigenous and non-Indigenous rate of IMD for 2005 was 4.1 and 1.5 per 100,000 respectively, producing a relative risk (95% CI) of 2.8 (1.2-6.5) for Indigenous persons compared to non-Indigenous persons. In 2004 there was also a significantly increased risk of IMD in Indigenous persons: RR (95% CI) = 3.9 (2.1-7.1).

All Indigenous cases of IMD in 2005 were aged <= 12 years. Of the 21 cases of IMD in children <5 yrs, three (14%) were Indigenous. The rate for Indigenous <5 years and for non-Indigenous <5 years is 16.2 and 7.6/100,000 respectively. This difference was not statistically significantly different. The small numbers of Indigenous children in 2005 may play a role in not finding a significant association; in 2004, Indigenous children were at a significantly higher risk for IMD than non-Indigenous children: RR (95% CI) = 3.1 (2.7-14.2.

There were no deaths in the Indigenous population from IMD in 2005.

3.4 Seasonal variation

In the years 2001-2003, there was an apparent winter/early spring peak in cases of IMD. In 2005, as in 2004, the seasonal variability is less apparent than previously (Figure 3 & Figure 4).

Figure 3: IMD cases by month/year of onset 2000-2005

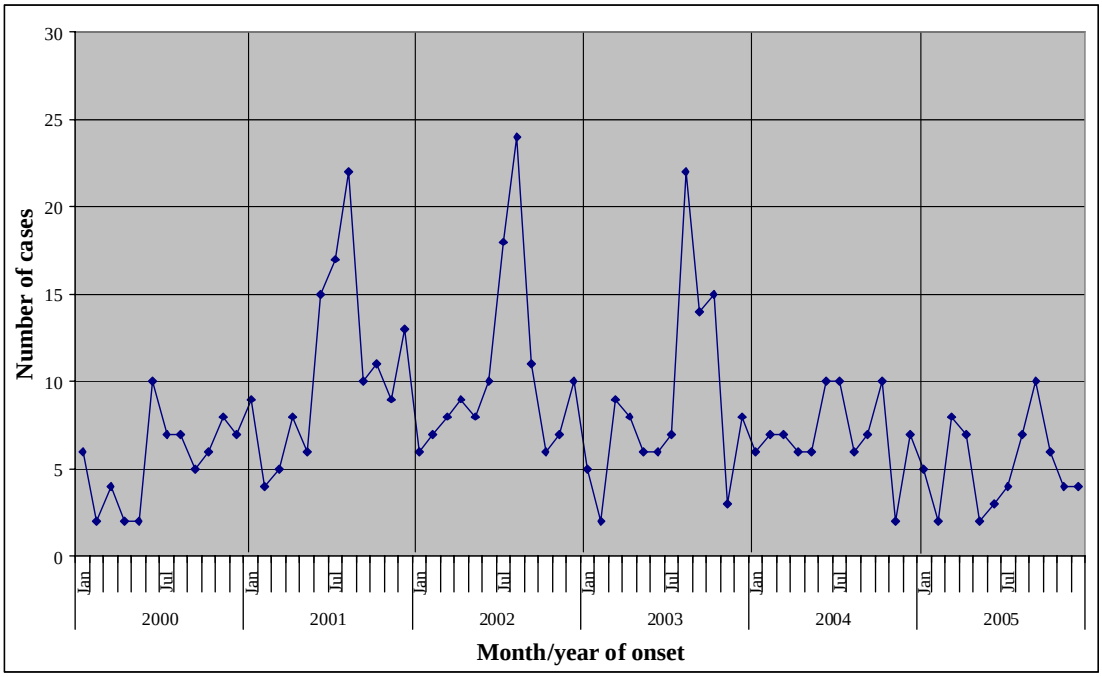
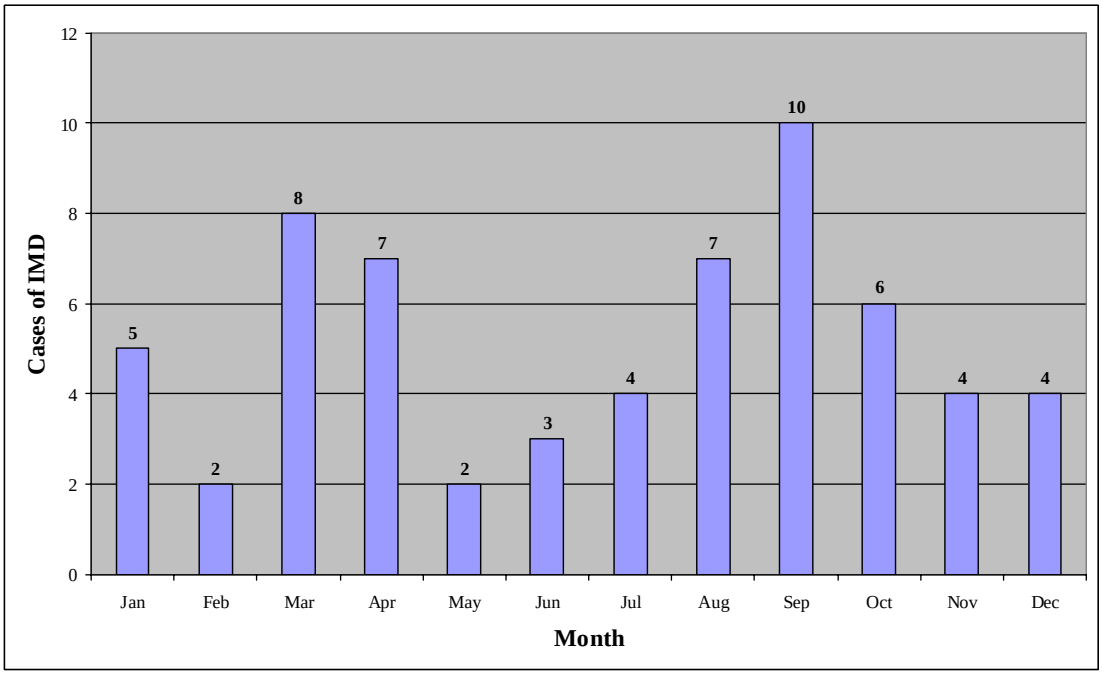


Figure 4: IMD by month of onset in Qld in 2005

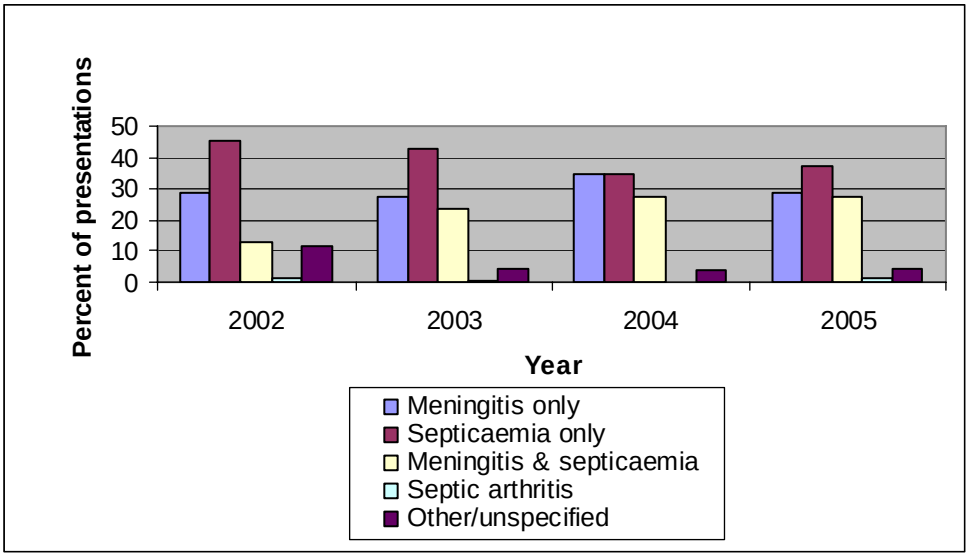


3.5 Clinical presentation

In 2005, IMD clinical presentations were 18 (29%) meningitis, 23 (37%) septicaemia and 17 (27%) meningitis and septicaemia. There was one presentation (2%) of septic arthritis alone and 3 cases (5%) were unspecified.

In 2004, these presentations were more evenly distributed, though the distribution of cases in 2002-3 resembles that in 2005 (Figure 5).

Figure 5: **Clinical presentations of IMD**



For 2005, 40 (64.5%) cases had a petechial rash. This is similar to 2000-2004 (range 59%-69%). Of those with a rash, nine (22.5%) had meningitis, 18 (45.0%) had septicaemia and 12 (30.0%) had meningitis and septicaemia.

3.6 Risk Factors

(i) Geographical distribution

Table 3: IMD incidence 2000-2005, and number of cases and percent of cases by statistical division in Qld, 2005

Statistical Division	Incidence per 100,000 population						% of cases 2005	No. of cases 2005
	2000	2001	2002	2003	2004	2005		
Brisbane	1.8	3.4	3.4	3.1	1.9	1.7	50.0%	31
Central West	0	0	8	0	8.2	0	0.0%	0
Darling Downs	2	1	2.4	0.9	1.8	0.4	1.6%	1
Far North	2.2	6.2	1.8	1.7	3.4	0.8	3.2%	2
Fitzroy	2.2	3.3	3.9	3.8	1.6	2.1	6.5%	4
Mackay	1.6	2.4	4.8	4.7	4.6	0.7	1.6%	1
Moreton	2.4	4	3.6	2.7	2.8	1.1	14.5%	9
North West	0	2.8	2.8	8.8	5.9	0	0.0%	0
Northern	1	2.5	3.4	2.4	0.9	4.1	14.5%	9
South West	3.9	0	0	0	0	0	0.0%	0
Wide Bay-Burnett	1.3	5.1	4.6	2.3	1.2	1.2	4.8%	3
Other	n/a	n/a	n/a	n/a	n/a	n/a	3.2%	2
Total	**1.9**	**3.5**	**3.3**	**2.8**	**2.2**	**1.6**	**100.0%**	**62**

Comparing 2005 with the previous five years, the 2005 incidence rate was the lowest for all statistical divisions in Queensland, with Northern as the exception (Table 3).

For the cases classified as other: one was an interstate resident and one was a New Zealand resident.

The incidence of IMD by Area Health Service is shown in Table 4.

Table 4: Incidence/100,000 population of IMD by Health Zone in Qld, 2005

Area Health Service	Number of cases 2005	Incidence /100,000
Northern	12	1.9
Central	24	1.6
Southern	26	1.4
Total	62	1.6

(ii) Links with other cases

There were no known links to other cases.

(iii) Child care and other institutional contact

Overall, there were 23 (36.5%) cases associated with institutions.

There were seven cases associated with preschool/childcare/vacation care settings. One was five years of age and the rest were less than four years. This represents 32% of all the cases in children aged <= 5 yrs. This compares with 13% in 2004 and 36% in 2003. There were no known cases of serogroup C infection.

Six of the cases of IMD for 2005 attended primary school and eight cases attended high school (although one was on holidays). All of these cases were infected with group B meningococci (80%) or unknown (20%) serogroups. There was one school teacher whose serogroup was identified as Y. Two were at tertiary institutions: one serogroup B and one serogroup C.

There were no cases identified as being associated with residential care institutions.

3.7 Laboratory diagnosis

Of the 62 cases recorded in 2005, 58 were laboratory confirmed (94%). 44 (64%) of these 58 were diagnosed by isolation of *Neisseria meningitidis* from a clinical specimen (Table 5).

Table 5: Method of diagnosis of IMD in Qld, 2004-5

Laboratory tests	Number (Percent) of laboratory cases	
	2004	2005
Isolation/PCR/Micro*	22 (26.8%)	15 (25.9%)
Isolation/PCR	10 (12.2%)	4 (6.9%)
Isolation/Micro*	28 (34.1%)	18 (31%)
Isolation only	4 (4.9%)	7 (12.1%)
PCR/Micro*	6 (7.3%)	2 (3.4%)
PCR only	9 (11.0%)	12 (20.7%)
Micro only	3 (9.8%)	0 (0%)
Total	**82 (100%)**	**58 (100%)**

*Micro is detection of Gram negative diplococci in Gram stain of specimen from a normally sterile site or from suspicious skin lesion

Of these, 37 were diagnosed by cultures from blood, 12 from cerebrospinal fluid (CSF) and one from hip joint fluid and one from another normally sterile site (Table 6).

Table 6: Site of isolation for IMD confirmed by culture of *N. meningitidis* in Qld, 2005

Site of isolation	Number (Percent) of cases 2005
Blood & CSF	5 (11.4%)
Blood, CSF & other normally sterile site	1 (2.3%)
Blood only	31 (70.4%)
CSF only	6 (13.6%)
Joint fluid	1 (2.3%)
Total	**44 (100%)**

33 (57%) cases were diagnosed by nucleic acid amplification/polymerase chain reaction (PCR) and 12 of these by PCR alone: 19 were from blood, 17 from CSF and five from both blood and CSF.

(i) Serogroups in 2005

Serogroups were determined for 56 (90%) of the cases in 2005, similar to 2003 (90%) and 2004 (89%).

Of those serogrouped, 43 (76.8%) were serogroup B, 12 (21.4%) were serogroup C and 1 (1.8%) was serogroup Y (Table 7). In comparison with previous years (1995-2004) the incidence of meningococcal B disease has not statistically significantly decreased (RR 0.8, 95% CI 0.6-1.1). However, serogroup C disease in 2005 was statistically significantly reduced: the risk of having known invasive serogroup C disease in 2005 was 0.5 times as likely as it was in 1995-2004 (95% CI 0.3-0.8).

Table 7: Trends in serogroups for meningococcal isolates in Qld, 1995-2005

Year	B	C	A	W135	X	Y	Z	Total Serogrouped
1995	38(58.4%)	23(35.4%)	1	1		2		65
1996	45(59.2%)	23(30.3%)		4		3	1	76
1997	47(73.4%)	13(20.3%)		4				64
1998	57(71.3%)	11(13.8%)		6		6		80
1999	46(68.7%)	16(23.9%)		3		2		67
2000	37(77.1%)	10(20.8%)		1				48
2001	70(64.8%)	32(29.6%)		1		5		108
2002	60(51.7%)	46(39.7%)		4	1	5		116
2003	54(56.8%)	39(41.1%)	1			1		95
2004	50(71.4%)	20(26.7%)	1	3		1		75
2005	43(76.8%)	12(21.4%)				1		56

(ii) Serotypes and serosubtypes

Of the 56 cases that had a serogroup determined in 2005 (Table 8), 43 (77%) were serotyped or serosubtyped. The most common serosubtypes were B: NT, 1.4 (five cases) and C: 2a, 1.5 (five cases).

Table 8: Serogroups, serotypes and serosubtypes of IMD in Qld, 2005

Serogroup	Serotype	Serosubtype	Number	Percent of all typed	Percent of all in serogroup
B	1	P1.14	3	5.4%	7.0%
		NT	1	1.8%	2.3%
	4	P1.4	2	3.6%	4.7%
		P1.7	1	1.8%	2.3%
	15	P1.6	1	1.8%	2.3%
		P1.7	4	7.1%	9.3%
		NT	1	1.8%	2.3%
	NT	P1.12,13	1	1.8%	2.3%
		P1.13	1	1.8%	2.3%
		P1.14	3	5.4%	7.0%
		P1.4	5	8.9%	11.6%
		P1.6	1	1.8%	2.3%
		NT	7	12.5%	16.3%
	no data	no data	12*	21.4%	27.9%
Subtotal			43		100.0%
C	2a	P1.4	1	1.8%	8.3%
		P1.5	5	9.0%	41.7%
		P1.5,2	2	3.6%	16.7%
		NT	2	3.6%	16.7%
	NT	P1.5,2	1	1.8%	8.3%
	no data	no data	1*	1.8%	8.3%
Subtotal			12		100.0%
Y	NT	NT	1	1.8%	100.0%
Total			56	100.00%	

* indicates where no serotyping/subtyping possible = 13 cases

There were two known cases of B:4:1.4, the typing used in Queensland for the New Zealand serosubtype which has been the cause of outbreaks in New Zealand and for which a vaccine has been used in that country. One of the most common subtypes in 2005, B:NT:1.4 may also represent some cases of this serosubtype which could not be definitively serotyped. One of these cases had been overseas in the last 60 days, but the countries visited were not known. There was one case of serogroup B acquired in New Zealand but there was no data for serotype or serosubtype.

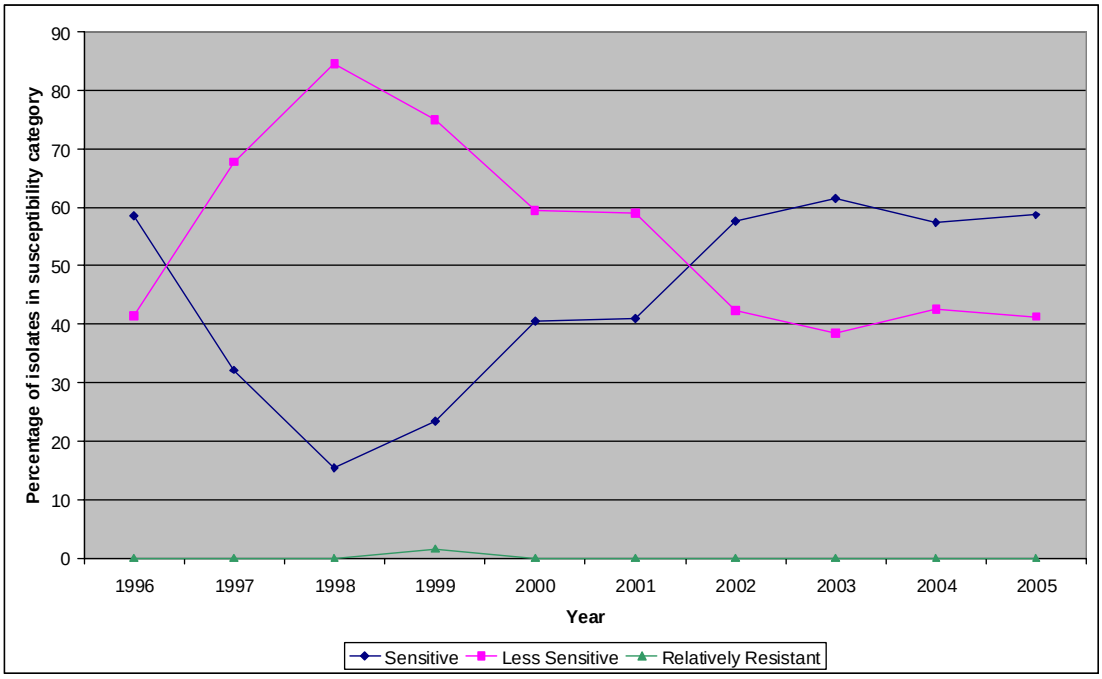
(iii) Antibiotic sensitivities

Penicillin Minimal Inhibitory Concentrations (MIC's) are presented in Table 9. Figure 6 shows the percentage of isolates in the penicillin sensitivity categories.

Table 9: Penicillin MIC's for IMD in Queensland, 1996-2005

Minimal Inhibitory Concentration (MIC) mg/l	Number (%) **1996**	Number (%) **1997**	Number (%) **1998**	Number (%) **1999**	Number (%) **2000**	Number (%) **2001**	Number (%) **2002**	Number (%) **2003**	Number (%) **2004**	Number (%) **2005**
0.008	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	2 (4.5)
0.015	6 (8.6)	3 (4.8)	2 (2.6)	2 (3.1)	2 (4.8)	2 (2.4)	0 (0)	6 (9.2)	3 (5.5)	0 (0)
0.03	35 (50)	17 (27.4)	10 (12.8)	13 (20.3))	15 (35.7)	32 (38.6)	49 (57.6)	34 (52.3)	29 (52.7)	24 (54.5)
Sensitive (<=0.03 mg/l)	41 (58.6)	20 (32.2)	12 (15.4)	15 (23.4)	17 (40.5)	34 (41)	49 (57.6)	40 (61.5)	31 (58.2)	26 (59)
0.06	25 (35.7)	30 (48.4)	39 (50)	34 (53.1)	18 (42.9)	43 (51.8)	28 (32.9)	18 (27.7)	15 (27.3)	11 (25)
0.12	3 (4.3)	5 (8.1)	19 (24.3)	11 (17.2)	6 (14.3)	4 (4.8)	4 (4.7)	5 (7.7)	2 (3.6)	2 (4.5)
0.25	1 (1.4)	3 (4.8)	5 (6.4)	2 (3.1)	0 (0)	1 (1.2)	4 (4.7)	2 (3.1)	5 (9.1)	4 (9.1)
0.5	0 (0)	4 (6.5)	3 (3.8)	1 (1.6)	1 (2.4)	1 (1.2)	0 (0)	0 (0)	1 (1.8)	1 (2.3)
Less Sensitive (0.06-0.5 mg/l)	29 (41.4)	42 (67.8)	66 (84.6)	48 (75)	25 (59.5)	49 (59)	36 (42.4)	25 (38.5)	23 (41.8)	19 (41)
Relatively Resistant (>= 1 mg/l)	0 (0)	0 (0)	0 (0)	1 (1.6)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Total	72 (100)	62 (100)	78 (100)	64 (100)	42 (100)	83 (100)	85 (100)	65 (100)	55 (100)	44 (100)

Figure 6: Percentage of IMD isolates and penicillin susceptibilities 1996-2005



All isolates 1996-2005 were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins), and to the prophylactic agent ciprofloxacin.

Susceptibility to rifampicin is presented in Table 10.

Table 10: Rifampicin MIC's for IMD isolates in Qld, 1996-2005

Minimal Inhibitory Concentration (MIC) mg/l	No. (%) **1996**	No. (%) **1997**	No. (%) **1998**	No. (%) **1999**	No. (%) **2000**	No. (%) **2001**	No. (%) **2002**	No. (%) **2003**	No. (%) **2004**	No. (%) **2005**
0.125	58 (82.9)	50 (80.6)	71 (91)	60 (93.8)	37 (88.1)	57 (68.7)	67 (78.8)	41 (63)	38 (69.1)	23 (52.3)
0.25	7 ((10)	5 (8.1)	5 (6.4)	2 (3.1)	4 (95.2)	14 (16.9)	13 (15.3)	15 (23.1)	10 (18.2)	13 (29.5)
0.5	1 (1.4)	5 (8.1)	0 (0)	1 (1.6)	1 (2.4)	9 (10.8)	5 (5.9)	6 (9.2)	6 (10.9)	7 (15.9)
Sensitive (<= 0.5 mg/l)	67 (95.7)	60 (96.8)	76 (97.4)	63 (98.4)	42 (100)	80 (96.4)	85 (100)	63 (96.9)	54 (98.2)	43 (97.7)
1	2 (2.9)	2 (3.2)	2 (2.6)	1 (1.6)	0 (0)	3 (3.6)	0 (0)	2 ((3.1)	1 (1.8)	1 (2.7)
4	1 (1.4)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Resistant (>= 1.0 mg/l)	3 (4.3)	2 (3.2)	2 (2.6)	1 (1.6)	0 (0)	3 (3.6)	0 (0)	2 (3.1)	1 (1.8)	1 (2.7)
Specimen of Resistant Isolates	CSF x 2 blood x 1	joint x 1 blood x 1	CSF x 1 blood x 1	blood x 1		CSF/ blood x 1 blood x 2		CSF/ blood x 2	blood x 1	joint x 1
Total	70 (100)	62 (100)	78 (100)	64 (100)	42 (100)	83 (100)	85 (100)	65 (100)	55 (100)	44 (100)

3.8 Invasive Meningococcal C Disease and vaccination

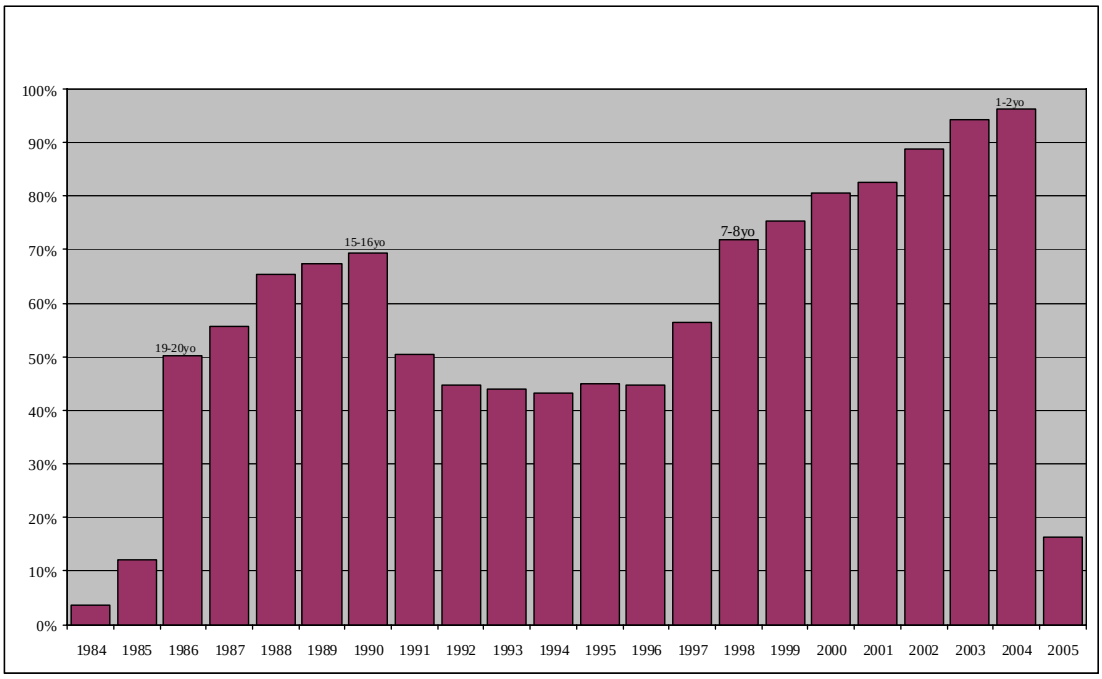
(i) Vaccination coverage

Data entry into Vaccine Information Vaccine Administration System (VIVAS) is still ongoing and so coverage information by year of birth is incomplete (particularly for the primary school programme).

Estimated vaccination coverage rates are presented in Figure 7. Coverage rates were calculated using 2004 single-year ERP figures as the denominator. Previous coverage rates from ERP figures suggest ERP figures vary in their accuracy and may under-estimate the actual numbers. There may also be underestimation due to delays in recording vaccination records and if the vaccine was privately funded and/or not sent to VIVAS.

The highest coverage rate was in 1-2 year olds with 96%. Those aged nine and 14 years had the lowest coverage rates at 43-50%, but this is where data entry in ongoing. Those aged 15-19 years old had higher coverage rates (50-69%). The low coverage rate for those born in 2005 is a reflection of incomplete data at the time of extraction, and ineligibility of children less than 12 months for free vaccine.

Figure 7: Meningococcal C vaccination coverage rates by year of birth in Qld (data extracted 4 April 2006)



Note: data entry of school based programme records (particularly primary school ie. those born in the 1990's) is still incomplete.

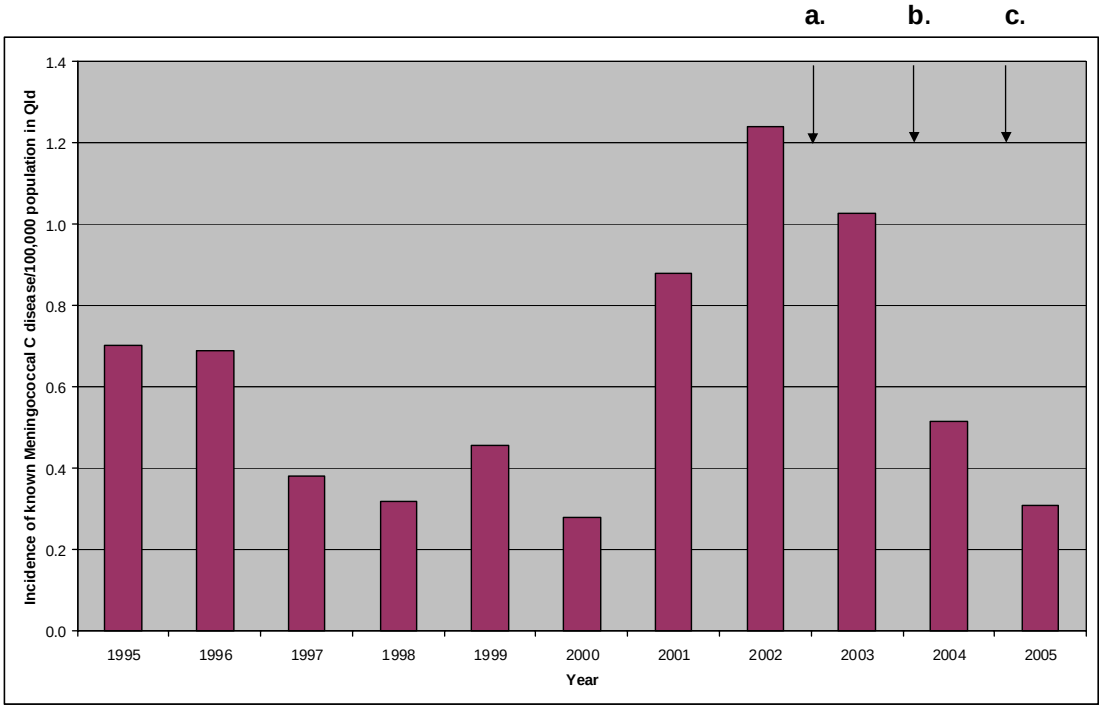
Table 11: Number of Meningococcal C vaccinations given by year in Qld.

Year of vaccination	Number
2002	18264
2003	334680
2004	250915
2005	75977
2006	13591
Total	693427

(ii) Incidence

Figure 8 illustrates the known incidence of meningococcal C disease in the Queensland population. As previously mentioned, 2005 shows a statistically significant decrease in IMD with the same being true of known serogroup C disease: it is 0.5 times as likely to have known meningococcal C disease in 2005 as it was in 1995-2004 (95% CI 0.2-0.8).

Figure 8: Incidence of known Meningococcal C disease per 100,000 in Qld, by year 1995-2005



a. grades 8-12 school vaccination programme introduced, vaccination of 1-5 yr olds & others not covered by continuing school programme through usual providers

b. grades 1-8 school vaccination programme introduced

c. vaccination of any others born on/after 1/1/1984 by usual providers

The incidence of meningococcal C disease was compared between pre vaccination (2000-2002) and post-vaccination (2004 & 2005) periods. The year 2003 was not included in the analysis, as the programme was initiated in this year. There was a statistically significant decrease in the incidence of

meningococcal C disease post-vaccination campaign. The vaccination programme was associated with a 30% decrease in known serogroup C IMD (RR 0.7, 95%CI 0.5-1.0). As Table 12 shows, there is a significant trend for reduction of meningococcal C cases in all ages from before the vaccination programme started (2000-2002) to 2004 and then 2005 (chi-square for trend, p<0.001).

Table 12: Cases and incidence of IMD serogroup C per 100,000 before (2000-2002) and after (2004, 2005) the vaccination programme

Age group	2000-2002		2004		2005	
	Number	per 100,000	Number	per 100,000	Number	per 100,000
1-9 yrs	18	1.3	0	0	2	0.43
1-14 yrs	26	1.2	2	1.06	2	0.28
1-19 yrs	50	1.69	14	1.37	3	0.3
1-21 yrs	vaccinat'n	programme	extended		4	0.37
All ages	88	0.81	20	0.51	12	0.42

In 2005, there were 12 cases (19%) of IMD serogrouped as type C. Of these, there were four cases that were eligible for the vaccine who were unimmunised:

- two were aged one year of age, one of whom died;
- one was 18 years of age at university;
- one was 21 years of age whose occupation was home duties.

The other eight cases of meningococcal C disease were in those not eligible for free vaccination, one of whom died. Their ages ranged from 23 to 62 years.

Of those with IMD and eligible for free vaccination (33 cases), 18 cases were known to be fully immunised. Of the others, 10 were not immunised, two were considered not applicable, two were considered unknown and for one case there was no data. There were no known cases of serogroup C in those known to be fully immunised (no known vaccination failures): serogroup B was identified in 15 cases. For three cases there was no serogroup available.

Also in 2004, there were no known vaccination failures: of those vaccinated there were 16 cases of serogroup B, one case of W135 and three unknown serogroups. In 2003, there was one child (2 years of age, non-Indigenous) who had been vaccinated for meningococcal C disease one month before the onset of known serogroup C disease and who died a day later of the condition.

In 2005, in the <1 year age group (nine cases), there were no known cases of meningococcal C disease: there were eight cases of meningococcal B disease and in one case the serogroup was unknown. Meningococcal C vaccine can be given to those <1 year of age (three doses required, 1-2 months apart) but it not available as a free vaccine in Queensland.

None of the cases of IMD in 2005 were vaccinated unless they were eligible for free vaccine: there were 18 cases who were fully vaccinated.

Table 13: IMD serogroup C by age group serogrouped in Qld, 2005 in relation to 1995-2004

Age group (yrs)	Percent of serogroup C of age group serogrouped											Total cases group-ed 2005	No. of cases of C 2005
	95	96	97	98	99	00	01	02	03	04	2005		
< 1							10	0	16.7	0	0	8	0
1-4							0	23	33.3	0	18.2	11	2
5-9							41.6	64	33.3	0	0	5	0
10-14		no data available					75	60	25	40	0	6	0
15-19							36.4	51.9	38.9	52.2	16.7	6	1
20-29							39.1	37.5	57	33	33.3	6	2
30-39							25	50	33.3	40	0	1	0
40+							33.3	38.9	66.7	11	53.8	13	7
Total	**35.4**	**30.3**	**20.3**	**13.8**	**23.9**	**20.8**	**29.6**	**40**	**41.5**	**26.7**	**21.4**	**56**	**12**

Figure 9: IMD cases by age and serogroup in Qld, 2005

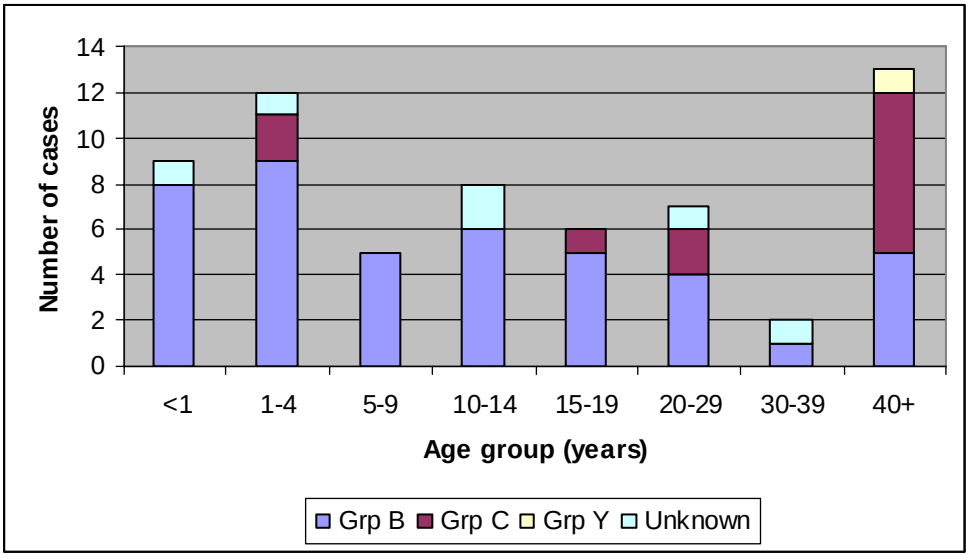


Table 14: IMD cases by age and serogroup in Qld, 2005

	<1 yr	1-4 yrs	5-9 yrs	10-14 yrs	15-19 yrs	20-29 yrs	30-39 yrs	40 + yrs
Grp B	8	9	5	6	5	4	1	5
Grp C		2			1	2		7
Grp Y								1
Unknown	1	1		2		1	1	
Total	9	12	5	8	6	7	2	13

Table 13, Figure 9 & Table 14 illustrate the trends in known meningococcal C disease by age group.

3.9 Outcomes

(i) Deaths

Table 15: Mortality due to IMD, by year 2000-2005 in Qld

Year	Deaths	Total Cases	Case Fatality Rate (%)
2000	4	66	6.1
2001	11	129	8.5
2002	5	124	4.0
2003	9	105	8.6
2004	3	84	3.6
Subtotal 2000-2004	32	508	6.3
2005	3	62	4.8
Total 2000-2005	**35**	**570**	**6.1**

In 2005, there were three deaths due to IMD (of 62 cases) with a case fatality 0f 4.8%. This was within the range of previous years 2000-2004 (3.6%-8.6%) and less than the average case fatality rate for the same period of 6.3% (Table 15). There was one death each from meningococcal B: NT: P1.4, C: 2a: P1.5 and C: 2a: NT infections. All were non-Indigenous with ages 0.5, 1 and 58 years (Table 16).

Table 16: Deaths due to IMD by age group 2000-2005 in Qld

Age Group	Deaths 2000-2004	Deaths 2005	Total cases 2000-2005	Case Fatality 2000-2005
<1	3	1	69	5.8%
1-4	6	1	92	7.6%
5-9	3		45	6.7%
10-14	2		40	5.0%
15-19	5		114	4.4%
20-29	3		92	3.3%
30-39	4		35	11.4%
40+	6	1	81	8.6%
Total	32	3	570	6.1%

(ii) Risk factors for dying

As in 2004, the small numbers of fatal cases restricts the determination of associations with dying for 2005.

In 2005, all cases had septicaemia and two had meningitis; all had a petechial rash; none were Indigenous and none of the cases was seen by a GP. One case had onset of illness, presentation to hospital and death on the same day, a second case had onset of symptoms the day before hospital presentation

and death (on the same day) and the third had presentation to hospital 2 days after onset of illness and subsequently died a week later.

Risk factors for dying in 2005 (Table 17) are similar to previous years where identified serogroup C was associated with a higher risk of death. Gender and Indigenous status were not associated with a higher risk of death. On figures available from 2001-2005, the presence of a rash was a significant predictor of mortality: RR (95%CI) = 2.8 (1.0-9.4).

Table 17: Risk factors for death among 570 cases of IMD in Qld, 2000-2005

Characteristic		Deaths (n=35)		All cases (n= 570)		Statistical significance
		n	% deaths	n	% total	
Sex	M	19	54	310	54	
	F	16	46	260	46	not significant
Indigenous*		3	9	53	9	
Non-indigenous*		31	89	473	83	not significant
Serogroup	C	21	60	159	28	RR (95%CI) = 5.0 (2.2-12.2)
	B	9	26	314	55	RR (95%CI) = 0.2 (0.1-0.5)
	Other	1	3	25	4	not significant
	Unknown	4	11	72	13	N/A

*Indigenous/non-Indigenous status determined in 526 cases and 34 deaths

3.10 Management of Invasive Meningococcal Disease

The recording of surveillance data such as dates and times of onset of illness, general practice and/or hospital presentation, notification to a public health unit and response by the public health unit were generally poorly completed in 2005. In several cases, there were major discrepancies in the flow of times recorded (or lack of reporting), so only data with a logical flow has been reported.

(i) General practice management

Of 60 cases where there was some attempt to record the time of onset of illness and review in hospital, a GP was consulted in 22 cases (36%). Of these cases, the GP referred the case to hospital in 18 cases (82%). However, one case saw two GP's but was not referred to hospital by either. Meningococcal disease was considered in 8 cases (36%), though in another 8 cases (36%) IMD was not considered and in 6 cases (27%) it was unknown/no data. The GP gave antibiotics in 3 cases (14%), though there was no data in 1 case. Benzyl penicillin was used in 2 cases and one was given ceftriaxone.

Table 18: Time from GP consultation to hospital review in 2001-2005

Time	2001 n=21	2002 n=29	2003 n=17	2004 n=17	2005 n=18	
	%	%	%	%	%	number
< 30 mins	0	6.9	11.8	17.6	5.6	1
30-< 60 mins	33.3	27.6	5.9	0	11.1	2
1-< 6 hrs	38.1	31	52.9	52.9	55.5	10
6-< 24 hrs	19	17.2	17.6	23.5	22.2	4
1-2 days	4	13.8	5.9	0	5.6	1
> 2 days	4	3.4	5.9	5.9	0	0
Total	100	100	100	100	100	18

The times from GP consultation to hospital review were analysed when possible (Table 18). The proportion of cases seen in each time frame in 2005 fall within the ranges of previous years, except there was a greater percentage reviewed at 1-<6 hours after seeing the GP and none seen after two days.

Of those were there is no prior review by a GP recorded (38 cases), the majority (25 cases/66%) were seen at hospital after 6 pm and before 8 am (18 cases/47%) or there were no times of attendance at hospital recorded (7 cases/18%).

(ii) Onset of illness to presentation at hospital

Table 19: Time from onset of illness to hospital review for IMD cases 2000-2005

Time	2000 n=53	2001 n=111	2002 n=80	2003 n=49	2004 n=40	2005 n= 32	
	%	%	%	%	%	%	number
< 1 hour	0	0	2.5	4.1	2.5	0	0
1- 6 hours	50.9	36	62.5	10.2	7.5	9.4	3
6-24 hours	15.1	24	13.8	53.1	47.5	40.6	13
24-48 hours	18.9	32	15	22.4	35	31.3	10
> 2 days	15	8	6.3	10.2	7.5	18.8	6
Total	100	100	100	100	100	100	32

There were 32 of the 62 (52%) cases that had reasonable time data for both onset of illness and review at hospital (Table 19). This ranged from 2 ½ hrs to almost 140 hours, with the median at 23 ½ hours (Table 20). All times to review at hospital were in the same range as previous years except that those for more than two days were slightly greater than previously but not statistically significant.

Table 20: **Median time from onset of symptoms to hospital review by year 2002-2005**

Year	Median time to hospital from onset of symptoms (hours)
2002	19
2003	16
2004	20.5
2005	23.5

For the three fatal cases of IMD in 2005, the time from onset of symptoms to hospital review was variable. One case is recorded as nine minutes (which is unlikely), a second case is less than 24 hours though the case had been unwell the week before and had been treated with antibiotics for tonsillitis and the third was almost two days. Since these data are considered unreliable, no statistical inference has been drawn between fatal and non fatal cases.

(iii) Time from hospitalisation to notification to a public health unit (PHU)

48 of the 62 cases (77%) had both these times recorded. As with previous years, this time period appears to be the longest in the disease management chain. In 2005, notification within 24 hours occurred in 56% of cases (Table 21) compared with 59% in 2004, 67% in 2003 and 57% in 2002. The shortest time to notification was before the case reached hospital (it is assumed that the GP who was consulted notified the PHU) and the longest was just under one week. In 2005, the percentage of notifications within 2-7 days is the highest since 2001. The *Public Health Act* requires immediate notification.

Table 21: Time from hospitalisation to notification 2001-2005

Time	2001 n=121 %	2002 n=97 %	2003 n=75 %	2004 n= 63 %	2005 n= 48 %	2005 n= 48 number
<1 hour	0	2.1	2.3	4.8	4.2	2
1-< 6 hours	36	32	29.3	27	20.8	10
6-< 24 hours	30	33	34.7	27	31.3	15
1-2 days	24	21.6	21.3	30.2	25	12
2-7 days	10	10.3	12	11.1	18.8	9
> 7 days	0	0	0	0	0	0
Total	100	100	100	100	100	48

(iv) Time for population health units to respond

In the vast majority of notifications (50 cases/81%) the PHU responded within 30 minutes and in one other case within 24 hours. In nine cases (13%) the exact time in which the PHU responded is not clear but it seems that these were all on the same day.

In only two of the 62 cases was the response greater than 24 hours. In one case, the population health unit was notified “late PM” on a Friday and it was

acted upon on the following Monday morning at 8 am. In another case, the details are recorded as being notified at 14:15 on one weekday and acted upon two days later at 14:15, although the dates may have been incorrectly recorded.

4. Discussion

In 2005, Queensland recorded the lowest number of cases and lowest incidence of IMD since state wide surveillance began in 1993. This is very promising since better laboratory techniques would have improved case ascertainment (12 cases were diagnosed by PCR only). This represents the largest decrease in numbers of any of the states and territories of Australia (12).

In 2005, there was also the lowest incidence of known serogroup C disease. The trend towards reduction in known serogroup C disease has been observed since the meningococcal C vaccination program was commenced in 2003 and extended in 2004 and 2005. However, there has been natural fluctuation in IMD over the last 11 years and it would be important to continue to monitor the incidence of this disease in the future.

As in previous years, the highest incidence of IMD is in those less than one year of age, but reassuringly, there were no cases of known serogroup C in this age group. The free vaccination program does not cover those less than one year of age.

Although incidence in children and adolescents is within the range of previous years, there were only four cases of known serogroup C disease in this vaccine-eligible group. All were unvaccinated. Two would have been difficult to reach with vaccination programs: one at a tertiary institution and the other occupied with home duties. The other two were both aged one year and presumably late for their 12 month vaccinations (one of whom died). This highlights the need for a reliable and timely reminder system for childhood vaccinations and continued community awareness of the importance and benefits of vaccination. The lower proportion of serogroup C in IMD in Queensland in 2005 is consistent with the latest information available for Australia, where 90% of IMD in under-fives was attributable to serogroup B (12).

Most areas of Queensland recorded the lowest or equal lowest incidence for the disease compared with the previous five year period. There was an exception: the Northern statistical division which experienced the highest incidence over the same period.

The discrepancies between Indigenous and non-Indigenous health in Australia are well known. In 2005, there was a still a statistically significant difference between these two groups in Queensland in all ages incidence of IMD, but not in the under-five age group. There were also no deaths from IMD in the Indigenous population. Indigenous status was recorded in all cases. This is in contrast with the difficulty experienced recording this identifying information in surveillance for many other diseases.

IMD continues to appear to be a sporadic disease. There were no established links with other cases in Queensland. While just over one third of

cases in 2005 were associated with institutions, this is probably more a reflection that those age groups with the higher incidences of IMD are likely to be at school, a tertiary institution or at daycare. The evidence for an association between attendance at daycare centres and IMD is not clear (13).The fact that no secondary cases of IMD occurred in 2005 is surely a tribute to effective and timely public health management of each case of IMD and their associated contacts.

The considerable heterogeneity in serotyping and serosubtyping of isolates supports sporadic disease as well. The two most common serotypes only had five cases each. There were two cases of B:4:P1.4 in Queensland in 2005. This has been the cause of high rates of disease in New Zealand for many years, but in Australia has never reached any great proportion of isolates associated with IMD.

It is of interest that there were 12 cases of those serogrouped that could not be serotyped or serosubtyped because they were identified by nucleic acid amplification/PCR only. With the advent of this molecular diagnosis technique, the percentage of cases with antibiotic susceptibility results has also decreased. In isolates, sensitivity to penicillin has remained much the same over the last five years in Queensland, with only one relatively resistant specimen isolated in 1999. In 2005, the percentage of organisms less sensitive to penicillin was 41% in Queensland. This compares with the Australia-wide figure of 68% (12). Sensitivity to rifampicin, commonly used as prophylaxis for contacts is also much the same as previous years with only one relatively resistant isolate from a joint fluid specimen in 2005. All isolates remain fully susceptible to third generation cephalosporins.

In 2005, as in 2004, the usual seasonal variation with a peak in winter/early spring, which had been evident with IMD in the past, is no longer apparent in Queensland. This seasonality appears to continue throughout the rest of Australia (12).

Clinical presentation in 2005 was similar to previous years with the vast majority (94%) of the presentations as meningitis and/or septicaemia. 65% of cases had a petechial rash. This continues to emphasize that the absence of a rash cannot be considered to exclude the diagnosis of IMD.

Case fatality from IMD in 2005 was 4.8% , a decrease from the previous five years, 2000-2004 (6.3%), and less than that published for the whole of Australia for 2004 (9.2%)(12). Group C IMD carried with it a significantly higher risk of mortality than other known serogroups of IMD. Mortality is currently the only outcome recorded by the enhanced surveillance system. In terms of health impacts, it may be useful to try to quantify some of the morbidity associated with this disease such as days of hospitalisation (particularly in intensive care units), loss of limbs or hearing.

Seeking early medical attention and early treatment remain the best ways to prevent adverse outcomes from this disease. Enhanced surveillance attempts to quantify delays in the management chain. Unfortunately data

collected is often incomplete and sometimes lacks logical flow. This may mean the enhanced surveillance forms need better design or that more care needs to be taken by those health professionals filling them in. This year's data on timing was taken directly from the enhanced surveillance forms and not transcribed through NOCS. There has been some discussion as to whether data entry people in NOCS should be validating the timing of management. It is more than likely that debate will continue as to whether data entry people should be questioning health professionals.

From the data that does have adequate validity on timing, just over one third of cases consulted a GP prior to hospitalisation and 82% of these cases were referred to hospital by the GP. It is of concern, that one case consulted two GP's and was not referred to hospital by either, but it may also reflect that IMD does not necessarily always present as severe disease. Even though meningococcal disease was considered in eight cases, antibiotics were only administered in three cases. There is no explanation as to why the others were not treated by the GP. It would be interesting to know if there was diagnostic uncertainty, lack of severity and perhaps urgency, or the proximity of a hospital: these explanations have been cited previously (7).

Any data on onset of illness may be difficult to record accurately as symptoms can sometimes be vague. Times to review by GP or to presentation to hospital in 2005 are within the range of previous years.

As in previous years, the time from hospitalisation to notification to a public health unit remains the longest. This may reflect the time taken to reach a diagnosis in some cases. However, it is important to remind clinicians that the *Public Health Act* in Queensland requires immediate telephone notification on suspicion of IMD.

In the vast majority of notifications, the population health unit responded within 30 minutes.

From this discussion, recommendations include:

- continuing the current meningococcal C vaccination program as there has been a significant reduction in illness and death in vulnerable groups. However, this does not infer cost-effectiveness.
- the updating of the vaccination reminder system to make it reliable and timely
- continued community awareness of the importance and benefits of vaccination
- in terms of the meningococcal C vaccination programme objectives, further time needs to elapse before long term immunity can be assessed. Continuing vigilance with surveillance and reporting would be ideal. However, thought needs to be given as to whether a nasal carriage study should be attempted.
- enhanced surveillance forms need to be improved and be more user friendly. The use of a codebook or glossary to improve the quality of the data may be necessary.

- there needs to be improved education of clinicians about the new *Public Health Act* in Queensland, in particular about the importance of notifying the appropriate public health unit urgently about suspected IMD by telephone. A suggestion would be that literature about the new obligations for medical professionals be provided via the medical registration board.

5. References

1. Apicella M. 2005. Neisseria meningitidis in Mandell, Douglas & Bennett's Principles and Practice of Infectious Diseases. Elsevier, Philadelphia.

2. McCall B, Neill A, Young M. 2004. Risk factors for invasive meningococcal disease in southern Queensland, 2000-2001. *Internal Medicine Journal.* 34: 464-468.

3. Communicable Diseases Network of Australia. *Guidelines for the early clinical and public health management of meningococcal disease.*

Available: http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/cda-pubs-other-mening.htm/$FILE/mening_guide.pdf

4. Ward J. 2000. *Enhanced Surveillance for Meningococcal Disease in Queensland in 1999*. Queensland Health.

5. Pugh R. 2002. *Meningococcal Disease in Queensland, Year 2000*. Queensland Health.

6. Pugh R. 2002. *Invasive Meningococcal Disease in Queensland, 2001*. Queensland Health.

7. Pugh R. 2003. *Invasive Meningococcal Disease in Queensland, 2002.* Queensland Health.

8. Quagliotto C. 2004. *Invasive Meningococcal Disease in Queensland 2003.* Queensland Health.

9. Homewood J. 2006. *Invasive Meningococcal Disease in Queensland 2004.* Queensland Health.

10. Australian gonococcal surveillance programme. 1984. Penicillin sensitivity of gonococci in Australia: development of an Australian gonococcal surveillance programme*. Br J Vener Dis.* 60: 226-230.

11. The Australian Meningococcal Surveillance Programme. 2004. Annual report of the Australian Meningococcal Surveillance Programme 2003*. Commun Dis Intell*. 28:194-206.

12. Australian Meningococcal Surveillance Programme. 2005. Annual report of the Australian Meningococcal Surveillance Programme, 2005. *Communicable Diseases Intelligence*. 30:2: 211-221.

13. McCall B, Neill A, Young M. 2004. Risk factors for invasive meningococcal disease in southern Queensland, 2000-2001. *Internal Medicine Journal.* 34: 464-468.