Annual report of the Australian Meningococcal Surveillance Programme, 1997

The Australian Meningococcal Surveillance Programme[1]

Abstract

The National Neisseria Network (NNN) has undertaken meningococcal isolate surveillance by means of a collaborative laboratory based initiative since 1994. The phenotype (serogroup, serotype and serosubtype) and antibiotic susceptibility of 343 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined in 1997. Ninety six percent of the invasive isolates were serogroup B or C. Serogroup B strains predominated in all States and Territories and were isolated from sporadic cases of invasive disease. Phenotypes B:4:P1.4 and B:15:P1.7 were prominent. Serogroup C isolates were most often encountered in New South Wales, especially in adolescents and young adults, and in that State were nearly as numerous as serogroup B strains. C:2a:P1.5 was the most frequently encountered phenotype and C:2b:P1.2 strains were also distributed widely. A number of clusters of cases of serogroup C disease were noted, mainly with phenotype C:2a:P1.5. About three quarters of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). Three isolates showed reduced susceptibility to rifampicin and one was chloramphenicol resistant. *Commun Dis Itell* 1998;22:205-211.

Introduction

Invasive meningococcal diseases, manifested mainly as bacteraemia and/or meningitis, remain a conspicuous cause of morbidity and mortality in Australia[1] and in 1997 attracted considerable public attention. The host response and outcome of disease in an individual patient, and the patterns of the infection within a community may be materially altered by the characteristics of the infecting organism. The public health response to an outbreak or cluster of cases is also influenced by certain features of the invasive meningococci, for example vaccines are available for some

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1. Corresponding author, J. W. Tapsall, Department of Microbiology, The Prince of Wales Hospital Randwick NSW 2031

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serogroups but not for others, and some phenotypes have been linked to disease outbreaks.

A national programme for the examination of strains of *Neisseria meningitidis* from cases of invasive meningococcal disease was commenced in 1994 with the co-operation and participation of reference laboratories in each State and Territory. This laboratory-based activity is designed to supplement data from existing clinical notification schemes by adding information on the serogroup, the serotype and subserotype (the phenotype) of invasive isolates as well as antibiotic sensitivity data to clinical data. In certain instances, other laboratory investigations are undertaken, such as pulsed field gel electrophoresis (PFGE), which, with other molecular techniques, determine the genotype of meningococci and provides further epidemiological information.

Reports summarising data gathered since the inception of the programme have been published in *CDI*.[2-4] The following report deals with the calendar year 1997.

Methods

The National Neisseria Network (NNN) is a collaborative programme for the laboratory surveillance of the pathogenic Neisseria, *N. meningitidis* and *N. gonorrhoeae.*[2-5] Meningococcal isolate surveillance is performed by a collaborative network of reference laboratories in each State and Territory (see acknowledgements for participants). Each case was based upon isolation of a meningococcus from a normally sterile site. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was obtained by each laboratory from clinicians and public health units. The isolate surveillance programme categorises cases on the basis of site of isolation of the organism. It is recognised that the total number of isolates was an underestimate of the number of cases, particularly of meningitis where lumbar puncture was not performed or was delayed and culture was sterile. However the above approach has been used since the beginning of this programme and is continued for comparative purposes.

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane proteins antigens using a standard set of monoclonal antibodies obtained from Dr. J. Poolman, National Institute for Public Health (RIVM), The Netherlands.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration to antibiotics used for therapeutic and prophylactic purposes. This programme uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique:

sensitive,	MIC $\leq$ 0.03 mg/l;
less sensitive,	MIC = 0.06 - 0.5 mg/l;
relatively resistant	MIC $\geq$ 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.

Results

Number of isolates

A total of 343 isolates of invasive meningococci were examined in 1997. There were 153 isolates from patients whose infections acquired in New South Wales (44.6% of all isolates), 62 (18.0%) from Queensland, 56 (16.3%) from Victoria, 24 (7.0%) from Western Australia, 20 (5.8%) from South Australia, 9 (2.5%) from Tasmania, 12 (3.5%) from the Northern Territory and 7 (2.0%) from the ACT (Table 1).

Seasonality

Fifty four (15.7%) of cases occurred between January 1 and March 31, 83 (24.2%) between April 1 and June 30, 105 (30.6%) between July 1 and September 30 and 101 (29.5%) between October 1 and December 31. A winter peak of meningococcal disease is normal.

Distribution of disease by sex and age

Overall there was slight excess of invasive meningococcal disease in males, 178 cases compared with 165 cases in females (M:F ratio 1.08:1). The ratio was highest for males in those patients ages 4 years or less (83 cases in males, 59 in females, M:F ratio 1.4:1) and those aged 15 to 24 years (48 cases in males, 37 in females, M:F ratio 1.3:1). In those aged 45 years or more, there were 30 cases in females and 14 in males (M:F ratio 0.46:1).

The age distribution of patients infected with invasive isolates in each State and Territory is shown in Table 2. Nationally, the peak incidence of meningococcal disease occurred in children aged less than 5 years, with 41.5% of all cases in this age group. Another peak was noted in the 15 - 19 years age group, where 53 cases (15.5% of the total) were recorded. A further 32 cases (9.0%) occurred in those aged 20 -24 years. New South Wales differed from the national pattern in that the number of cases of invasive disease in those aged 15 - 24 years was the same as that

Contents, ***continued***

Table 1. *Neisseria meningitidis* isolates, Australia, 1997, by State or Territory and serogroup

State/ Territory	Serogroup										
	B		C		Y		Other[1]		NG[2]		Total
	N	%	N	%	N	%	N	%	N	%	N
ACT	5	72	1	14	1	14	0	0	0	0	7
NSW	78	51	72	47	3	2	0	0	0	0	153
NT	8	67	4	33	0	0	0	0	0	0	12
Qld	43	69	14	23	0	0	3	5	2	3	62
Tas	5	50	3	30	0	0	1	10	1	10	10
SA	14	70	4	20	2	10	0	0	0	0	20
Vic	44	79	9	16	0	0	0	0	3	5	56
WA	22	96	1	4	0	0	0	0	0	0	23
Total	219	64	108	32	6	1.6	4	0.8	6	1.6	343

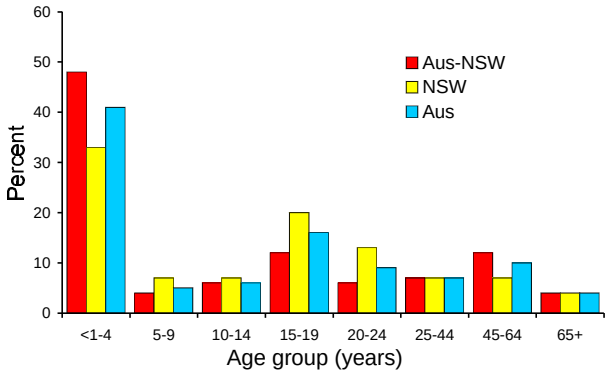
1. Other includes serogroup Z and serogroup W135. There were no serogroup A isolates
2. NG = non-groupable

Table 2. *Neisseria meningitidis* isolates, Australia, 1997, by State or Territory and age.

State/Territory	Age group (years)										
	< 1	1-4	5-9	10-14	15-19	20-24	25-44	45-64	65+	NS	All
ACT	2	2	0	0	0	0	0	3	0	0	7
NSW	21	30	10	11	30	20	10	11	6	4	153
NT	3	1	2	1	0	0	0	2	1	2	12
Qld	12	21	2	5	9	4	2	6	1	0	62
SA	7	3	0	1	0	1	3	3	2	0	20
Tas	2	0	1	0	2	1	1	1	1	0	9
Vic	9	18	2	2	6	4	7	6	2	0	56
WA	4	7	1	2	6	2	1	1	0	0	24
Total N	60	82	18	22	53	32	24	33	13	6	343
%	17.5	24	5.2	6.4	15.5	9.3	7	9.6	3.8	1.7	100

for those aged less than 5 years, each group accounting for one third of the infections in that State. In contrast, in Queensland and Victoria about half the cases were less than 5 years old, with a lower secondary peak in the young adult group (Figure 1).

Figure 1. Percentage of isolates of invasive meningococci in each age group, 1997, for all Australia, New South Wales and Australia excluding New South Wales



Serogroup, Serotype and Serosubtype (phenotype) Distribution

The distribution of the isolates by serogroup is shown in Table 1. Overall, the 219 serogroup B isolates represented 64% of all strains and the 108 serogroup C strains were 31.5% of the total. Serogroup Y, Z and W135 strains (6, 2 and 2 respectively) were also identified. Four isolates were not serogroupable and 2 were nonviable. No serogroup A isolates were encountered in 1997.

The regional data show some important serogroup differences between centres, and New South Wales in particular had a distinct serogroup distribution. Serogroup B predominated in aggregated national data and especially in Western Australia (all but one of the 24 strains), Victoria (79% of isolates), and South Australia and Queensland (70%). In contrast, in New South Wales the 78 group B strains accounted for only 51% of isolates. Group B

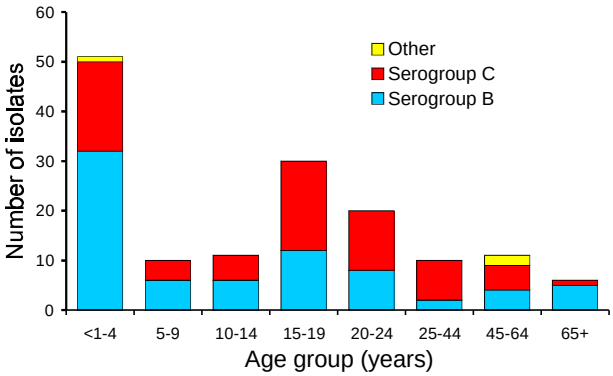
disease comprised unlinked and apparently sporadic cases.

With serogroup C isolates there was again a contrast between New South Wales and the other regions. Seventy two of the 108 group C strains isolated in Australia were from New South Wales. Group C meningococci represented 47% of the New South Wales isolates whereas proportions of group C strains were much lower in other States and Territories, ranging from 34% in the Northern Territory (4 cases) and Tasmania (3 cases) to 16% (9 cases) in Victoria. Only one group C strain was isolated from Western Australia.

Additionally, the distinct serogroup distribution in New South Wales had an age specific pattern. Figure 2 shows the number of cases of invasive disease in New South Wales by age and serogroup. Serogroup B isolates predominated in the 0 - 4 year age group isolates (32 of 51) and generally conformed to the national pattern, whereas serogroup C strains were most prominent (30 of 50) in the adolescent and young adult age group (15 - 24 years). This latter picture was not present in other States and Territories.

There was considerable phenotypic heterogeneity amongst invasive isolates as determined by serotyping and serosubtyping and the strains from New South Wales also showed a different phenotypic pattern to that observed in other States. The predominant

Figure 2. Number of isolates of invasive meningococci, New South Wales, 1997, by age and serogroup



serotype/serosubtypes in each State and Territory are shown in Table 3. Serogroup B meningococci are more difficult to characterise by serological methods, and a number were not successfully phenotyped. B:4:P1.4 strains were present in New South Wales, Queensland, Victoria, Western Australia and the ACT and B:15:P1.7

Table 3. Most frequently isolated serotypes and serosubtypes of *Neisseria meningitidis*, Australia, 1997, by State and Territory.

	Serogroup			
	B		C	
State/Territory	Serotypye:serosubtype	N	Serotype:serosubtype	N[1]
ACT	4:P1.4	1	2b:P1.2	1
NSW	4:P1.4	17 (11)	2a:P1.5	39 (15)
	NT:NST	13 (9)	2b:P1.5,2	8 (10)
	2b:P1.10	11 (8)	2a:P1.5,2	3 (2)
	15:P1.7	7	2b:P1.2	2 (0)
NT	4:P1.5	2	2a:P1.5,2	2
			2a:P1.5	2 (2)
Qld	4:P1.4	3 (6)	2b:P1.5,2	0 (10)
	NT:P1.4	9 (4)	2a:P1.5	1 (4)
	15:P1.7	3	2a:P1.5,2	3 (4)
	2b:P1.10	2	2b:P1.2	3
SA	15:P1.7	16 (3)	one isolate only	
	2b:NST	(2)		
	NT:P1.10	(2)		
Tas	Various		2b:P1.2	2
Vic	NT:P1.4	8 (13)	2a:P1.5	1
	15:P1.7	3	2b:P1.10	1
	4:P1.4	3 (2)	2b:P1.2	1
	2b:P1.10	2		
WA	15:P1.7	5	2a:P1.5	1
	NT:P1.4	5 (5)		
	4:P1.4	1		

1. The numbers of isolates of each phenotype in 1996 are shown in parenthesis

strains in New South Wales, Queensland, Victoria and Western Australia.

There was less heterogeneity amongst serogroup C meningococci. There were 44 serogroup C strains of phenotype 2a:P1.5, comprising 40% of all group C strains. Thirty nine of these were found in New South Wales and, of these, 19 were in the young adult group. Strains of this type were also isolated in Queensland, Victoria and the Northern Territory. The single serogroup C strain in Western Australia was also a 2a:P1.5 phenotype. In New South Wales this phenotype was associated with several clusters of invasive disease. Another cluster of cases was associated with strains of phenotype C:2a:P1.5,2, this phenotype also appearing in Queensland and the Northern Territory.

Site of isolation

There were 129 isolates from CSF either alone or with a blood culture isolate and 200 from blood culture alone. Fourteen isolates were from other sterile sites including synovial fluid and skin lesions.

Outcome data for 1997

Outcome data (survived or died) were available for 240 patients. Sixteen deaths were recorded (6.6%) (Table 4).

There were two deaths in 86 patients (2.3%) with meningitis. Both patients were infected with serogroup B strains. Thirteen deaths were recorded in 146 bacteraemic patients (8.9 %). There were 89 cases of serogroup B meningococcal bacteraemia with six deaths and another 44 cases were caused by serogroup C strains among whom five fatalities were recorded. Two of four patients with septicaemia with serogroup Y meningococi died.

Other antibiotics.

The 339 isolates which were tested for susceptibility to ceftriaxone and, by extrapolation, to other third generation cephalosporins, were susceptible to these therapeutic agents. Two hundred and sixteen isolates were examined for chloramphenicol resistance and a single strain from New South Wales was resistant at a MIC of 8 mg/L.

Three hundred and thirty nine isolates were also tested for susceptibility to the prophylactic agents rifampicin and ciprofloxacin. Two isolates from Queensland had raised MICs to rifampicin (MICs of 1 mg/l). All isolates tested were sensitive to ciprofloxacin. Sulphonamide testing was not performed.

Discussion

The number of isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme increased to 343 in 1997 from the 297 in 1996. These are 69% of the 496 notifications of meningococcal disease for 1997 and 70% of the 426 notifications for 1996. The number of isolates examined in 1994 was 216, which was 56% of the 383 notifications for that year. Most of the increase since 1994 has been the result of improved surveillance. The numbers of invasive isolates in most States and Territories in 1997 changed only slightly from those in 1996. However in New South Wales the number of isolates available for further examination increased from 95 in 1996 to 153 in 1997. Two factors in New South Wales contributed to this increase; the number of clinical cases notified increased from 166 in 1996 to 219 in 1997 and isolates from a higher proportion of culture positive cases were available. The number of isolates available for examination will always be less than the number of clinically notified cases because surveillance case definitions include culture negative cases. The increased number of cases in New South Wales and changing meningococcal phenotypes accounted for most of the differences from previous reports.

The dominant pattern observed in most centres was one of sporadic serogroup B disease. This was the pattern also in New South Wales, but additionally in that State there was

Table 4. ***Neisseria meningitidis*** **isolates, Australia 1997, outcome of meningitic and septicaemic cases by serogroup**

		Serogroup				
Disease Type	**Outcome**	**B**	**C**	**Y**	**NG[1]**	**Total**
Meningitis	Survived	64	19	1	0	84
	Died	2	0	0	0	2
	Total	66	19	1	0	86
Septicaemia	Survived	83	44	2	4	133
	Died	6	5	2	0	13
	Total	89	49	4	4	146
All cases[2]	Survived	150	66	3	5	224
	Died	8	6	2	0	16
	Total	158	72	5	5	240

1. Non groupable
2. Includes two serogroup B, two serogroup C and one non-groupable strain from joint aspirates and one serogroup B and one serogroup C from skin lesions, all of whom survived, and a serogroup C sterile site aspirate in a fatal case.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Using defined criteria, 92 of 339 strains tested (27%) were fully sensitive to penicillin and 247 (73%) less sensitive (MIC 0.06 to 0.5 mg/l). MICs recorded ranged between 0.015 and 0.5 mg/l.

increased serogroup C disease, most noticeably in young adults and adolescents. The serogroup C disease was also mainly sporadic, but clusters of cases, typical of group C meningococcal disease, were also noted. This serogroup C activity in young adults was responsible for the different age distribution of disease in New South Wales. Serogroup B and serogroup C isolates together accounted for 95.5% of all invasive meningococci. No serogroup A meningococci were isolated in 1997. This picture is typical of the pattern of meningococcal disease in developed countries.

Phenotyping data, obtained on the basis of serotyping and serosubtyping was again available in 1997. Of interest amongst the group B strains were phenotypes B:4:P1.4 and B:15:P1.7, associated with hyperendemic disease in New Zealand and Europe respectively. B:4:P1.4 strains were most frequently encountered in New South Wales, and were also present in low numbers in other States and the ACT. B:15:P1.7 isolates represented a high proportion of strains from Western Australia and were also widely distributed.

Of special interest in the 1996 report was the recognition of the phenotypes C:2a:P1.5 and C:2a:P1.5.2 in all States and Territories except South Australia and Western Australia. These phenotypes have been implicated in hyperendemic meningococcal disease in Canada for a number of years[6] and have also been reported in Europe. Studies here indicate that the C:2a:P1.5 isolates in New South Wales are of the ET 37 (15) complex seen overseas (Jane Jelfs, South Western Area Pathology Service, personal communication). They were responsible for a cluster of cases in Western Sydney in 1996 and continued to be isolated throughout 1997. The C:2a:P1.5 phenotype was responsible for 39 cases of invasive disease in New South Wales in 1997, a number of these occurring as clusters. This phenotype was also present in Queensland, Victoria and the Northern Territory. The Western Australian isolate of this phenotype was probably acquired in New South Wales. The C:2a:P1.5,2 phenotype was seen in a small cluster of cases in 1997.

Overall, the outcome data in 1997 (6.8% mortality) were similar to those recorded in 1996 (6% mortality) and are in the expected range where early diagnosis, and appropriate antibiotic therapy and supportive measures are available.[7]

A decrease in susceptibility of meningococci to penicillin has been noted in many parts of the world. Isolates have occasionally been shown to be resistant to other antibiotics which are used currently in the therapeutic or prophylactic management of meningococcal disease. This programme therefore includes routine examination of the antibiotic susceptibility of invasive isolates as part of its surveillance. By using consistent methods over the past four years the data now provide evidence of an emerging trend in Australia.

Since 1994 there has been an increase in the proportion of invasive meningococci showing some decrease in penicillin susceptibility. In 1994, 52% of strains were in the 'less sensitive' range (MIC 0.06 - 0.5 mg/L). In 1995, 155 (63%) of 247 strains tested were 'less sensitive'. The proportion of less sensitive isolates increased further to 74% of 297 isolates in 1996. This proportion remained unchanged in 1997 (73%). The isolation of a meningococcus with a MIC in the less sensitive range does not mean that therapeutic failure will occur, but the increase in the number and proportion of stains in this category is an epidemiological marker of the slow progression to resistance.

The definition of what constitutes 'resistance' to the prophylactic agent rifampicin varies. This programme has chosen to monitor the number of isolates with MICs of 1 mg/l or more. In 1997, two isolates from Queensland had rifampicin MICs of 1 mg/L or more, compared with three strains in 1996. One isolate in New South Wales was chloramphenicol resistant.

The Australian Meningococcal Surveillance Programme (AMSP) has examined a total of more than 1100 strains from all States and Territories since 1994 and has assisted in clarifying and expanding information on invasive meningococcal isolates in Australia. The programme is investigating the utility of other means of enhancing laboratory diagnosis of meningococcal disease, notably PCR based diagnosis using cerebrospinal fluid, and serology for retrospective diagnosis. Other methods of strain differentiation, most notably pulsed field gel electrophoresis are available. For further details, contact the relevant NNN member (see acknowledgements).

Acknowledgements

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

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AMSP participants

Participants in the Australian Meningococcal Surveillance Programme (to whom strains should be referred and enquiries directed):

QUEENSLAND

Mr John Bates
Public Health Microbiology
Queensland Health Scientific Services
39 Kessels Road
Coopers Plains Qld 4108
phone (07) 3274 9101
fax (07) 3274 9008
email batesj@health.qld.gov.au

WESTERN AUSTRALIA

Mr. C. Richardson / Mr. P. Campbell / Ms K. Stowe
Department of Microbiology
Princess Margaret Hospital for Children
1 Thomas Street
SUBIACO WA 6008
phone (08) 9340 8273
fax (08) 9380 4474
email chris.richardson@health.wa.gov.au

TASMANIA

Dr. K. Ott
Department of Microbiology and Infectious Diseases
Royal Hobart Hospital
GPO Box 1061L
HOBART TAS 7001
phone (026) 2388 410

SOUTH AUSTRALIA

Mr A.Lawrence
Microbiology Department
Women's and Children's Hospital
72 King William Road
NORTH ADELAIDE SA 5006
phone (08) 8204 7326
fax (08) 8204 6051
email lawrencea@wch.sa.gov.au

ACT

Dr. P. Collignon
Department of Microbiology
Royal Canberra Hospital
PO Box 11
WODEN ACT 2606
phone (06) 244 2425
email peter.collignon@dpa.act.gov.au

VICTORIA

Dr. J. Griffith / Dr. G. Hogg
Microbiological Diagnostic Unit
University of Melbourne
PARKVILLE VIC 3052
phone (03) 9344 5701
fax (03) 9344 7833
email j.griffith@microbiology.unimelb.edu.au

NORTHERN TERRITORY

Dr G. Lum
Microbiology Department
Royal Darwin Hospital
Casuarina NT
phone (089) 8922 8888
fax (089) 8922 8843
email glum@ozemail.com.au

NEW SOUTH WALES

A/Prof. J. Tapsall
Microbiology Department
The Prince of Wales
RANDWICK NSW 2031
phone (02) 9382 9079
fax (02) 9398 4275
email j.tapsall@unsw.edu.au

OR

A/Prof. Rosemary Munro
Department of Microbiology and Infectious Diseases
South Western Area Pathology Service
Locked Mail Bag 90
LIVERPOOL NSW 2179
phone (02) 9828 5128
fax (02) 9828 5129
email r.munro@unsw.edu.au

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Annual report of the Australian Gonococcal Surveillance Programme, 1997

The Australian Gonococcal Surveillance Programme[1]

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) examined 2,817 isolates of *Neisseria gonorrhoeae* in the period 1 January to 31 December 1997, a number similar to that reported in 1996. The biggest change in incidence of gonococcal disease occurred in New South Wales and Queensland where a 20% rise in the number of isolates was noted. In the latter case this was due to improved surveillance, but in the former represented a real increase. The sites of infection and antibiotic susceptibility patterns varied considerably between regions reflecting considerable differences between rural and urban gonorrhoea in Australia. Strains examined in South Australia, New South Wales and Victoria were predominantly from male patients and rectal and pharyngeal isolates were common. In other centres the male to female ratio was lower and most isolates were from the genital tract. Resistance to the penicillin and quinolone groups of antibiotics were also highest in urban centres, but penicillins remained suitable for use in many parts of rural Australia. Quinolone resistance in gonococci continued to increase. This was particularly so in Sydney where quinolone resistant *N. gonorrhoeae* (QRNG) accounted for about 15% of all isolates and spread of QRNG was predominantly by local contact. QRNG in other centres continued to be isolated at a lower frequency, mostly from overseas travellers. All isolates remained sensitive to spectinomycin and ceftriaxone. ***Commun Dis Itell*** **1998;22:212-216.**

Introduction

There is renewed interest in rates of gonococcal disease and control of gonorrhoea following converging epidemiological and biological studies showing the significant role of this disease as an amplification factor in the spread of HIV. [1-4] The gonococcus has a well demonstrated capacity to develop antibiotic resistance by numerous chromosomal and extrachromsomal mechanisms. Continuing and long term surveillance is required to monitor and respond to changes in resistance which can occur in a short space of time.[5]

The Australian Gonococcal Surveillance Programme (AGSP) is a collaborative programme conducted by reference laboratories in each State and Territory. The primary aim of the programme is to monitor antibiotic susceptibility of Australian isolates of *Neisseria gonorrhoeae*, to assist in the formulation of treatment regimens appropriate to proper management of gonorrhoea. Management of gonorrhoea is based on single dose antibiotic therapy at first diagnosis, a strategy that assists patient compliance. There is a close correlation between the likely outcome of treatment and the *in vitro* susceptibility of the causative organism. However treatment is usually provided before results of susceptibility tests on individual isolates can be performed. Treatment regimens are therefore formulated using knowledge of the *in vitro* sensitivity of prevalent gonococci.[5] That is, the overall pattern of susceptibility of prevalent gonococci is the critical determinant of appropriate antibiotic therapy rather than individual strain susceptibility identified on a case by case basis.[6]

Quarterly reports have been provided to *CDI* since antibiotic sensitivity data were first produced by the AGSP in 1981.[7-10] Initially only data on penicillin resistance were reported and the AGSP documented the appearance and spread of penicillinase producing gonococci (PPNG) in Australia.[11] Monitoring of resistance to other antibiotics was added as newer therapeutic agents became available. Currently the emergence and spread of gonococci resistant to the quinolone antibiotics, agents widely used in Australia, is of particular concern. This is the second annual summary of AGSP data in *CDI* and provides information on trends in disease as well as antibiotic sensitivity data.

Methods

The AGSP comprises participating laboratories in each State and Territory (see acknowledgements). It is a network of collaborating centres which seeks to obtain isolates for examination from as wide a section of the community as possible. For example, strains from the Northern Territory are isolated in Alice Springs, Katherine and Darwin and in the laboratories of Western Diagnostic Pathology and Queensland Medical Laboratory in the Northern Territory and further tested in AGSP centres in Perth, Adelaide and Sydney. The sources of isolates remained relatively unchanged between 1996 and 1997. Gonococci isolated in and referred to the participating laboratories were examined for antibiotic susceptibility by a standardised methodology[11] and the AGSP conducted a programme-specific quality assurance programme.[12] Antibiotic sensitivity data were submitted quarterly to the co-ordinating laboratory which collated the results and also conducted the QA programme. Additionally the AGSP received data on the sex and site of isolation of

1. Corresponding author, J. W. Tapsall, Department of Microbiology, The Prince of Wales Hospital Randwick NSW 2031

gonococcal strains. The geographic source of acquisition of resistant strains was ascertained whenever possible.

Results

Number of isolates

There were 2817 isolates examined in 1997 (Table 1) . Nine hundred and two gonococci (32% of the Australian total) were isolated in New South Wales, 595 (21%) in Queensland, 445 (16%) in Western Australia, 393 (14%) in the Northern Territory, 362 (13%) in Victoria, and 107 (4%) in South Australia, with small numbers in Tasmania and the Australian Capital Territory. Compared with data from the same sources in 1996, the greatest changes in the number and percentage of isolates were the increases in New South Wales (from 723) and Queensland (from 504) and the decrease in Western Australia (from 578). In New South Wales, where the sources of referral have been stable, the increase in the number of isolates continues a trend evident since 1994 (Figure 1). In Queensland, improved retrieval and referral of isolates to the AGSP contributed to the increase.

Figure 1. The number of gonococcal isolates from similar sources, New South Wales, 1994 - 1997

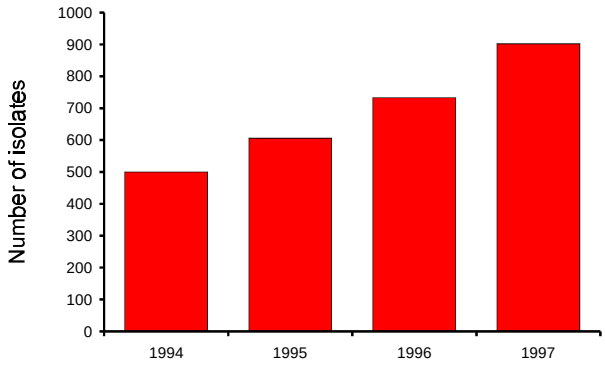


Table 1. Gonococcal isolates, Australia, 1997, by sex, site and region

Sex	Site	Region NSW	Vic	Qld	SA	WA	NT	Australia
Male	Urethra	707	255	346	65	300	95	1,778
	Rectal	73	50	16	19	5	0	164
	Pharynx	51	18	8	10	1	1	89
	Other/NS	3	3	18	0	20	148	192
	Total	834	326	388	94	326	244	2,223
Female	Cervix	62	30	196	13	108	95	505
	Other/NS	6	6	11	0	11	54	89
	Total	68	36	207	13	119	149	594
TOTAL	All sites	902	362	595	107	445	393	2,817

Source of isolates

There were 2,223 strains from men and 594 from women, giving a male : female (M:F) ratio of 3.7:1. This is higher than the 1996 ratio of 2.8:1 because of a concurrent increase in the number of strains from men (up from the 2,033) and decrease in the number from women (down from 720).

The M:F ratio was higher in South Australia (7.2), New South Wales (12.2) and Victoria (9.0) where strains were obtained more from urban populations (which have a higher rate of homosexual transmission), but lower in Western Australia (2.7), Queensland (1.9) and the Northern Territory (1.6), reflecting the large non-urban component of gonococcal disease (mainly heterosexual transmission) in those regions. Male rectal and pharyngeal isolates were most frequently found in New South Wales (together accounting for 14.9% of male isolates there), South Australia (30.9%) and Victoria (20.8%). This pattern is similar to that noted in 1996. Just under 10% of isolates were from 'other' sites. These included 9 cases of disseminated gonococcal infection, 7 in men and 2 in women. Many of the remaining isolates were from urine samples collected in northern Australia, and can best be regarded as genital tract isolates. There were also ophthalmic isolates from this region from young children and a small number of isolates from the eyes of newborn infants.

Antibiotic susceptibility patterns

In 1997, the AGSP reference laboratories examined 2,817 gonococcal isolates for sensitivity to the penicillins, ceftriaxone, ciprofloxacin and spectinomycin and for high level resistance to tetracycline (TRNG). However the patterns of gonococcal antibiotic susceptibility differed between the various States and Territories. For this reason data are presented by region as well as aggregated for Australia.

Penicillins

The categorisation of strains in Australia in 1997 by penicillin MIC is shown in Figure 2. Resistance to the penicillin group (penicillin, ampicillin, amoxycillin) may be mediated by the production of beta-lactamase

Figure 2. Penicillin resistance of gonococcal isolates, Australia, 1997, by region

FS Fully sensitive to penicillin, MIC ≤ 0.03mg/l.
LS Less sensitive to penicillin, MIC 0.06 - 0.5 mg/l
RR Relatively resistant to penicillin, MIC ≥1 mg/l
PPNG Penicillinase-producing *Neisseria gonorrhoeae*

(penicillinase-producing *N. gonorrhoeae,* PPNG) or by chromosomally controlled mechanisms (CMRNG).

Chromosomal resistance is expressed as the minimal inhibitory concentration in mg/l (MIC) which is the least amount of antibiotic which inhibits in vitro growth under defined conditions. The MIC reflects the expression of multiple and different chromosomal changes present in an organism. These multiple changes result in incremental increases in the MIC and strains are classified as fully sensitive (FS, MIC ≤ 0.03 mg/L), less sensitive (LS, MIC 0.06 - 0.5 mg/l) or relatively resistant (RR, MIC ≥ 1 mg/l). PPNG are a separate resistant category. Infections with strains in the LS or FS categories usually respond to therapy with standard treatment regimens with the penicillins. Infections with strains which are PPNG or in the RR category usually fail to respond to the penicillins.

There were 361 (12.8%) isolates with resistance to penicillin mediated by chromosomal mechanisms (CMRNG). Strains of this type were concentrated in Victoria (52 CMRNG, 14.6% of all isolates), New South Wales (246 CMRNG, 27.3% of all isolates) and South Australia (38 CMRNG, 35.5%). In contrast there were no CMRNG amongst Northern Territory isolates and only 3 (0.7%) in Western Australian strains. The 21 CMRNG in Queensland represented 3.5% of all isolates there.

The number (180), and proportion (6.4%), of PPNG rose slightly in 1997. Again the distribution of PPNG differed by region. While New South Wales had the highest number of PPNG (62), Victoria had the highest proportion (10.7%). PPNG were an important mechanism of resistance in Perth where the 33 strains accounted for 7.4% of isolates. The Australian Capital Territory was the only State or Territory where PPNG were not isolated in 1997. Most isolates were from patients infected overseas. Isolates which were fully sensitive to the penicillin group remained prominent in Victoria.

Ceftriaxone and Spectinomycin

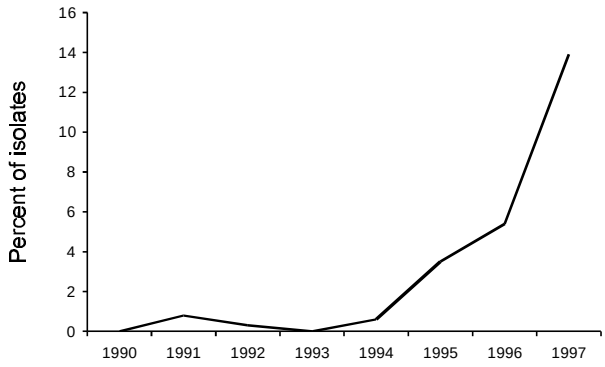
All strains from all parts of Australia were sensitive to these injectable agents.

Quinolone antibiotics

Resistance to the quinolone antibiotics is mediated only by chromosomal mechanisms and is thus incremental. The AGSP uses ciprofloxacin as the representative quinolone and defines altered resistance as a MIC ≥ 0.06 mg/l. Treatment with the currently recommended dose (500 mg) of ciprofloxacin is usually effective for strains with this less developed resistance, but lower doses of the antibiotic will often result in treatment failure. Treatment failure is also likely with high doses in infections with strains with MICs of 1 mg/l or more. Currently, gonococci with MICs up to 16 and 32 mg/l are being seen in Australia.

In 1997, a total of 204 (7.2%) of gonococcal isolates displayed altered sensitivity to the quinolones (QRNG). This is a large increase on the 108 (4%) QRNG seen in the previous year. QRNG were found in all States and Territories except Tasmania and the Australian Capital Territory. Victoria had 21 (5.8%) QRNG, Queensland 18 (3%), and Western Australia 13 (2.9%), with smaller numbers in South Australia and the Northern Territory. The biggest change in 1997 occurred in New South Wales where 144 (16%) of all isolates showed altered sensitivity to quinolones and 14% showed high level quinolone resistance. An increase in the number and proportion of high level QRNG had been noted in New South Wales in the December quarter of 1996 and this rate of isolation was sustained throughout 1997 (Figure 3). Another disturbing feature of the QRNG isolated in Sydney was their spread by local contact so that there is now sustained domestic transmission of these antibiotic resistant strains in that city. While most other centres showed a slight change in the number and percentage of QRNG isolated, the pattern of acquisition outside New South Wales is still mainly through overseas contact.

Figure 3. High level quinolone resistance (MIC≥ 1 mg/l) in gonococci, New South Wales, 1990 - 1997



High level tetracycline resistance (TRNG)

One hundred and sixty two TRNG (5.8% of isolates) were detected throughout Australia in 1997, a slight increase over the 1996 numbers. Approximately equal numbers

were found in Western Australia (40), Queensland (44) and New South Wales (47), corresponding to 9%, 7.4% and 5.2% of their isolates respectively. TRNG were also seen in Victoria, with 15 isolates (4%), and the Northern Territory, with 11 isolates (2.8%). The Australian Capital Territory was the only centre not reporting TRNG in 1997. Infections with TRNG were mainly acquired overseas in Indonesia, Thailand and Singapore. However an increasing number of isolates were acquired through local contact.

Discussion

Until 1996, the AGSP reported data on the basis of financial rather than calendar years. Also, while there have been stable sources of isolates in some regions, such as New South Wales, additional numbers of strains from Western Australia, the Northern Territory and Queensland have become available for examination in the Programme. Therefore, historical comparisons are restricted to 1996 data in this report.

A number of significant changes have been identified or confirmed in the 1997 AGSP data. The number of isolates examined increased only slightly overall (from 2,753 to 2,817), but decreased in some centres and increased in others. The most notable increase in the number of isolates was in New South Wales and Queensland where the number of strains rose by about 20% when compared with 1996 data. In New South Wales this further accelerated a trend noted since 1994 (Figure 1).

Although all participating centres have an urban and non-urban component in their mix of isolates, the relative contributions of each differs. The greater urban impact is reflected in the high male to female ratio and rate of extra-genital infection in New South Wales, Victoria and South Australia. The different pattern of gonococcal disease in northern Australia is shown in the lower male to female ratio and high rate of genital tract isolates in data from Queensland, Western Australia and the Northern Territory.

The differing urban and non-urban components of gonococcal disease are also seen in the regional differences in antibiotic susceptibility profiles of gonococci examined in different centres. In general most resistance is present in isolates from the more populous urban centres. For this reason the regional sensitivity patterns provide a more precise guide to suitable treatment than aggregated Australian data. However trends towards resistance noted in the large urban centres have in the past been indicative of subsequent directions in resistance in other regions.

The major trends in antibiotic resistance in 1997 related to the penicillins and the quinolones. Penicillin resistance in the larger centres, especially Melbourne and Sydney, has been high for many years. In these cities and in Adelaide, chromosomal resistance has been of greater importance than PPNG for a number of years and this trend was maintained or increased in 1997. In the other centres, PPNG were the main vehicle of resistance to the penicillins, but in rural areas these strains are not yet endemic and the penicillin group of drugs remains a suitable standard treatment.

The quinolone group of antibiotics is important as it is essentially the only currently available oral alternative to the penicillins in Australia. The patterns of resistance noted in 1997 are therefore cause for concern although they are as yet limited principally to Sydney. There has been a slow but progressive increase in both the number of QRNG isolated in Australia and in the MICs of the these QRNG for a number of years. It is not surprising, given the high incidence of QRNG in countries close to Australia, that QRNG have been repeatedly isolated from infected travellers entering or returning to Australia. The significant difference noted in Sydney in 1997 was not only the continuing high proportion of QRNG in that city, but their spread by local as opposed to overseas contact. It has been suggested that treatment regimens should be altered when resistance to an agent is found in 5% of isolates. The number of QRNG in Sydney in 1997 exceeds this level several fold.

The global decline in incidence of gonococcal disease in more developed countries has now been arrested and, in parts of Australia at least, the number of cases is again increasing. The choice of suitable treatment for gonorrhoea in Australia is becoming increasingly restricted, especially in the larger cities. Continued monitoring of resistance patterns is required to optimise treatment regimens.

Acknowledgements

Participating laboratories in the AGSP (to whom isolates should be referred) :

John Bates, Denise Murphy and Vicki Hicks. Queensland Health Scientific Services, Coopers Plains, Queensland

Edna Limnios, Tiffany Shultz and John Tapsall. Department of Microbiology, The Prince of Wales Hospital, Randwick, New South Wales

Julia Griffith and Geoff Hogg. The Microbiological Diagnostic Unit, University of Melbourne, Parkville, Victoria

Greg Handke and Rosemary Perrie. Infectious Diseases Laboratories, Institute of Medical and Veterinary Science, Adelaide, South Australia

Cheryll McCullough. Microbiology Department, Royal Perth Hospital, Perth, Western Australia

Mark Gardam and Keith Ott. Department of Microbiology and Infectious Diseases, Royal Hobart Hospital, Hobart, Tasmania

Gary Lum and Microbiology Staff. Microbiology Department, Royal Darwin Hospital, Casuarina, Northern Territory

Linda Halliday and Peter Collignon. Microbiology Department, Royal Canberra Hospital, Woden, Australian Capital Territory

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What is the Gonococcus Telling Us?

Basil Donovan, Director, Sydney Sexual Health Centre, Sydney Hospital, Clinical Associate Professor, Department of Public Health and Community Medicine, The University of Sydney

Biologically and politically the gonococcus could hardly be more different from the human immunodeficiency virus (HIV). Yet both organisms speak the same language. Each is rare among heterosexual, non-injecting, non-prostitute, non-indigenous Australians who have not had sex overseas ("us").[1] Instead, they are concentrated among "them": the faceless, the stigmatized, the under-served, "the other".[2]

Because it killed so obviously, HIV demanded our attention. Considerable resources have been committed to surveillance, clinical and social services, research, and health promotion programs which include removing institutional barriers to HIV control. The HIV-affected communities have been central players and skilled advocates. Australia nets an excellent return on its AIDS-dollar.[3]

By contrast, the curable gonococcus was left to its own devices along with the other sexually transmissible infections (STIs). Remarkable declines in the incidence of gonorrhoea and some other STIs in our cities during the 1980s[1] were viewed as unintentional but positive spin-offs of HIV control programs. Unfortunately, many community advocates still choose to ignore the overwhelming evidence that most other STIs directly promote the sexual transmission of HIV.[4] With some justification, the other STIs are seen as trivial distractions from the Main Game, HIV control. "HIV control in a broader sexual health context"[3] has sometimes been positioned as a threat to singularity of purpose and a potential diffusion of precious resources. The opportunity to complement behavioural HIV control strategies with biological interventions (control of other STIs) has been resisted. STI control has only been conceded as relevant for indigenous Australians.

Left out in the political cold, the gonococcus has thrived. Elegantly documented in this issue by the Australian Gonococcal Surveillance Program (AGSP),[5] the gonococcus is relentlessly returning to the hyperendemic levels that contributed to the peak incidences of HIV infections among our gay communities in the early 1980s. In Sydney the number of gonococcal isolates examined by the AGSP to the end of July 1998 exceeded the total number for 1996 (Prof J Tapsall, unpublished). At the Sydney Sexual Health Centre 67% of all cases of gonorrhoea since 1995 have been among gay men, a third of whom were HIV positive (unpublished data). The largely unspoken hope that advances in anti-retroviral therapy could yield a less infectious HIV-infected population overall (even if this cannot be assumed for individuals) could be at least partially offset by the gonococcus increasing that population's infectiousness or their partners' susceptibility to HIV infection.

So what is the gonococcus telling us to do? We could start with some national leadership and policy structures commensurate with the morbidity, mortality (direct and indirect) and controllability of the other STIs. Though they are inter-related it is naïve to assume that good HIV control is synonymous with good STI control. Each STI is different.[1]

To use the gonococcus as an example, education programs – for health professionals, policy makers, key communities and community leaders – which include the role of the gonococcus in enhancing the transmission of HIV may be timely. The much greater infectiousness of the gonococcus and its wide clinical spectrum need to be better understood.

New combined gonorrhoea/chlamydia polymerase chain reaction (PCR) screening tests are already being used among indigenous populations. PCR testing has the advantages of being able to use urine or other self-collected specimens, overcoming many of the cultural and logistic barriers to case-finding. PCR testing is likely to become standard in most clinical settings but, in its current form, it does not yield antibiotic sensitivity information. Modifications to the Medical Benefit Schedule (MBS) will need to be negotiated to ensure that this vital clinical information remains available from large representative samples.

The MBS also has structural barriers to screening for the gonococcus among gay men with HIV. A standard HIV

monitoring visit to a GP (full blood count, biochemistry, T-cell subsets, viral load test) already exceeds the "3-test rule", under which the GP's pathologist(s) is only rebated for the three most expensive tests. Concurrent multi-site tests for gonorrhoea and chlamydia may be more than the pathologist can afford to absorb financially and is likely to be discouraged. Tests deemed to be of public health importance, such as the Pap smear, are already exempted from the 3-test rule. STIs could also be deemed to be of public health importance, at least in selected populations, if screening is to be encouraged.

The exclusion of sex workers from MBS rebates for STI testing is not only questionable from public health and economic perspectives, it is also legally dubious: all recent studies among sex workers have found their private risk greatly exceeds their professional risk of STIs.

As highlighted in the current report[5] Australia no longer has access to a reliable oral treatment for gonorrhoea. Cefixime is such a drug. It is recommended and used by the U.S. Centers for Disease Control and Prevention. Unfortunately the small market and licensing fees in Australia make it uneconomic for the drug's manufacturers to bring cefixime to this country. This too needs to be negotiated.

Finally, because a substantial proportion of people with gonorrhoea develop symptoms within weeks of becoming infected, it has proven to be the model disease for studying how STIs move through populations. Insights into the epidemiology of gonorrhoea informed the study of HIV epidemiology and are likely to continue to do so as our ability to discriminate between gonococcal strains improves. Rarely manifesting as point-source outbreaks, the epidemiology of gonorrhoea and HIV is predominantly based on multiple small clusters of infections which reflect broader social trends and system failures. The AGSP has begun the task of translating the message. Are we prepared to listen?

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Editor's column

This issue

This issue of *CDI* has a focus on Neisseria with the publication of the 1997 Annual Reports of the Australian Meningococcal Surveillance Programme (p 205) and the Australian Gonoccocal Surveillance Programme (p 212). While the meningococcus and the gonococcus have different means of transmission and cause very different diseases, each is showing changes in epidemiology and patterns of antimicrobial resistance which have important implications for treatment and for public health management and control. In his thought-provoking editorial, Donovan (p 216) draws out these implications for the gonococcus and challenges us to respond to prevent the organism from again becoming hyperendemic. We continue our series on important issues in immunisation with the article by Botham et al (p 218) on the immunisation of preterm infants. Media reports will have made many readers aware of the current outbreak of Newcastle disease (ND) in poultry in NSW. The outbreak report (p 222) provides useful information for reassuring the public about the very low risk to human health from the ND virus. The report of an outbreak of Q fever in an abattoir in NSW (p 222) provides a reminder of the importance of vaccination for abattoir workers throughout Australia.

More changes in the editorial team

Since my last column, the *CDI* editorial team has farewelled another member, our deputy editor, Corrine Rann. Corrine worked with us for 15 months and was the person of first contact for many of our readers, contributors and reviewers. She implemented many improvements to the *CDI* layout and her editorial skills ensured that each issue of *CDI* was produced to a high standard. Her skills and contributions are missed. This is my last editor's column as I am moving shortly to a new position within the Department of Health and Family Services. Pending the appointment of a new Editor and Deputy/Assistant editors, the remaining members of the *CDI* editorial team will continue to publish national surveillance data and outbreak information. However, publication of the 1997 Annual report of the National Notifiable Diseases Surveillance System will be delayed, as will a number of the articles currently in preparation. Please be patient as it will not be long before *CDI* will be back to normal.

Bronwen Harvey

Immunisation of Preterm Infants

S J Botham, D Isaacs, M A Burgess

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS), Royal Alexandra Hospital for Children, Westmead, New South Wales

Importance of immunising preterm infants

In 1993, there were 16,207 preterm births (37 weeks gestation) in Australia, representing 6.3% of all confinements. The majority of preterm births were delivered at 32 - 36 weeks (5.1% of all confinements), but 0.7% and 0.6% of all confinements occurred at 28-31 weeks and 20 -27 weeks respectively.[1] Improvements in neonatal care have resulted in the survival of more infants of lower gestational age.[2] All preterm infants are prone to infection, and infants of lower gestational age with underlying lung disease from long term ventilation are especially susceptible to respiratory pathogens. These infants can benefit greatly from immunisation, and health care providers have a responsibility to ensure that they receive appropriate and timely immunisations.

This brief report provides a summary of current knowledge about the safety, efficacy and duration of immunity of the Australian standard schedule vaccines in preterm infants.

Diphtheria-tetanus-pertussis

Pertussis is a major problem in Australia.[3,4] Hospitalisation and deaths relating to pertussis are more likely to occur in preterm infants.[5,6] Initial studies in preterm infants born at 26 - 36 weeks (mean 31 - 33 weeks) using a combined diphtheria, tetanus and whole cell pertussis (DTPw) vaccine showed that immunogenicity and reactogenicity were similar in preterm and term infants.[7,8,9,10] Recent studies have shown that infants of lower gestational age (range 24 - 34 weeks, mean 27 -28 weeks) do not tolerate DTP immunisation as well as the infants in the earlier studies.[11,12,13] New or increased episodes of apnoea and bradycardia were reported in the 24 - 72 hour period following immunisation for some infants born at 31 weeks gestation. The majority of the apnoeic episodes were minor and self limiting. The authors recommend that all hospitalised preterm infants be commenced on cardiorespiratory monitoring prior to immunisation and that monitoring be continued for at least 48 hours.[12,13] However, preterm infants well enough to be discharged home can safely start their immunisations at 2 months of age (i.e. 2 months after birth), in line with current National Health and Medical Research Council (NHMRC) recommendations that preterm infants be immunised at the same chronological age as term infants.[14]

The studies of reactogenicity in very low birth weight infants did not examine immunogenicity, but a satisfactory antibody response to tetanus toxoid after three immunisations has been reported in a study of 16 preterm infants (<29 weeks and <1000g at birth).[15] The immune responses to diphtheria and pertussis were not measured at this time but the children were followed up 3 - 4 years later (see under Duration of immunity, p 219).[16]

Acellular pertussis vaccines, now available in Australia and licensed for all age groups, may be better tolerated than whole cell vaccines, but few data are available on their use in preterm infants. A 1995 report of a three component acellular vaccine trial in Italy showed the vaccine to be safe and immunogenic in 87 preterm infants (gestation 26 - 37 weeks) but did not state whether these infants were monitored following vaccination.[17]

Haemophilus influenzae type b (Hib)

Hib conjugate vaccines have been available in Australia since 1992 and are routinely commenced at 2 months of age. PRP-OMP, which produces an early antibody response, is preferred for groups at high risk of early Hib disease and is the recommended vaccine for Aboriginal and Torres Strait Islander infants on a 2, 4,12 month schedule. However, several studies have shown that after 2 doses, preterm infants (mean gestational age 28 weeks) do not respond as well to PRP-OMP as term infants. Antibody levels indicative of long term protection were obtained in only 53 - 55% of infants[18,19] PRP-T given on a 2,4,12 month schedule also produced a reduced response in preterm infants after 2 doses, although no difference was seen after the third dose.[20] A study of HbOC vaccine, which has a 4-dose schedule at 2,4, 6 and 18 months, showed that, after 3 doses of HbOC, preterm infants (mean gestational age 26 weeks) had a comparable response to that of term infants.[15] Therefore, the NHMRC recommends that preterm infants receiving PRP-OMP be given an additional dose at 6 months, but that there be no change to the schedule for preterm infants receiving HbOC.[14]

Polio

Protection against polio is achieved by giving 3 doses of oral polio vaccine (OPV) commencing at 2 months of age. After administration of OPV the virus is excreted in the stool for about 6 weeks. Because of the theoretical risk of transmission to other infants, the vaccine should not be given to preterm infants until they are discharged from hospital. Inactivated polio vaccine (IPV) may be used for long term hospitalised infants. It is also the vaccine of choice for infants who have a disease, or are receiving treatment, which lowers immunity. Limited data are available on the use of polio vaccines in preterm infants. Two studies showed that OPV and IPV produce a satisfactory response to all serotypes after 2 doses,[21,22] while a third study showed a poor response to serotype 3.[14] Sick preterm infants given 2 doses of IPV showed a poor response to all 3 serotypes.[23]

Hepatitis B

Although the NHMRC recommends that all infants receive hepatitis B vaccine, universal vaccination will not be incorporated into the standard vaccination schedule until a combination vaccine has been licensed.[14] Currently, hepatitis B vaccine is only routinely given to infants of women who are hepatitis B surface antigen (HBsAg)

positive or who come from communities with carrier rates over 2%. Vaccination of these infants is recommended at birth, 1 month and 6 months. A number of studies have examined the response of preterm infants to hepatitis B vaccine with conflicting results. Several studies have shown a lower seroconversion rate and reduced antibody response in preterm infants immunised at birth.[24,25,26] An improved response was seen if immunisation was delayed until the infants reached a weight of 2kg, or if it was given at about 1 month of age.[25,26,27] Other studies have demonstrated a satisfactory antibody response in preterm infants.[28,29,30] However, the studies showing a reduced response had more infants of lower gestational age and birth weight.

The NHMRC currently suggests two options for hepatitis B immunisation in preterm infants:[14]

1. Give 1st dose at birth, then at 1, 6 and 12 months (4 dose schedule)
2. If birthweight is <2kg, delay immunisation until 2 months of age and give at 2, 3 and 8 months (3 dose schedule)

Option 1 is recommended if the mother is HBsAg positive, when immunoglobulin must also be given, ideally within 12 hours of birth. For both options it is advisable to measure antibody levels 1 month post immunisation.

Duration of immunity

An early British study of 69 preterm infants (gestation 26 – 35 weeks, mean 32 weeks) given DTPw and OPV showed that all had antibody concentrations consistent with protection for diphtheria, tetanus and polio to the age of 19 months (minimal protective levels for pertussis have not been established).[10] There is only one published study of immunisation in extremely preterm infants.[16] This followed 16 neonates born at <29 weeks and <1000g at birth. At 3 - 4 years of age their geometric mean antibody titres (GMT) were similar to a control group of term infants for diphtheria, tetanus and pertussis, and were consistent with protection for diphtheria and tetanus. However, these preterm infants had a lower GMT for Hib and polio serotype 3 than the control group. Twelve of these infants had also received hepatitis B vaccine, and protection was comparable to that of term infants. A study of hepatitis B vaccine in Native Alaskan infants showed that only 8.1% of the preterm infants had protective levels at 3 years of age but a similar decline was also seen in the term infants, in whom only 15% had protective levels.[31]

A longitudinal study of Hib immunity is currently in progress in preterm infants in Adelaide and the results will soon be available. Longitudinal studies of antibody responses in preterm infants receiving vaccines according to the Australian immunisation schedule will be important in determining the immunogenicity and duration of immunity of new vaccines in this group of infants.

Conclusion

Well preterm infants can be safely immunised at the same time as term infants. Full doses of all vaccines should be given. The response of sick and very low birth weight infants appears to be reduced for Hib and hepatitis B vaccines and schedule modifications may be needed. The response of sick infants to diphtheria, pertussis (whole and acellular) and polio vaccines is uncertain and further studies in this population may be warranted.

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The NCIRS was established by the National Centre for Disease Control, Commonwealth Department of Health and Family Services. The Centre analyses, interprets, and evaluates national surveillance data on immunisation coverage and vaccine preventable diseases. NCIRS also identifies research priorities, and initiates and coordinates research on immunisation issues and the epidemiology of vaccine preventable diseases in Australia.

Measles Control Campaign Update

During the three month period of the Campaign, the uptake of measles-mumps-rubella (MMR) vaccine given at primary school clinics and the number of adverse events following MMR vaccination are being monitored. Data are forwarded to the National Centre for Disease Control for collation and publication in *CDI*.

Measles Control Campaign activity data, cumulative to 25 September 1998

Sum total students	661,194
Total forms returned	612,989
Consents to vaccinate	519,068
Total students immunised	487,174
Percentages are:	
Of total students	93% returned their forms
Of total forms returned	85% consented to vaccination
Of total consents to vaccination	94% have been vaccinated
Of total students	74% have been vaccinated.

Adverse events

Faints/syncopy	14
Syncopal fits	10
Hyperventilation	3
Anaphylaxis	3
Rash	2
Local allergic reaction	1
Local reaction	1
Arthropathy	1
Fever	1
Myalgia	1
Lymphadenapathy/ headache/stiff neck/rash	1

Enquiries can be directed to Sue Campbell-Lloyd, National Manager of the Measles Control Campaign, Sydney Office, Commonwealth Department of Health and Family Services.

Global eradication of polio

World Health Organization: Global action plan and timetable for safe handing and maximum laboratory containment of wild polioviruses and potentially infectious materials.

The goal of global eradication of poliomyelitis (polio) is in sight. The number of reported cases has been reduced by more than 90% since the initiative began in 1988 and at least 155 countries are now reporting zero cases annually. The date of onset of the last case of polio associated with wild poliovirus infection in the Western Pacific Region (which includes Australia) was 19 March, 1997.

Once polio is eradicated, the laboratories of the world will be the only remaining source of the virus. Safe handling and, ultimately, maximum containment of poliovirus and potentially infectious materials in the laboratory is crucial.

In July 1998, the World Health Organization (WHO) released the above document for public comment.

The proposed plan is posted on the Internet and can be accessed at:
Http://www.who.ch/gpv-documents/DocsPDF/www9829.pdf

Access to this file requires the use of Adobe Acrobat Reader Software which can be downloaded free of charge at:
http://www.adobe.com/prodindex/acrobat/readstep.html

Comments on the plan should be submitted to

Dr. David Featherstone
Global Programme for Vaccines
World Health Organization
CH-1211 Geneva 27
SWITZERLAND
Email: erstoned@who.ch
by 1 November, 1998.

For copies of the WHO document or further information on polio eradication, the Plan, or polioviruses please contact:

Margery Kennett,
Director,
Western Pacific Regional and Australian National Polio Reference Laboratory
Victorian Infectious Diseases Reference Laboratory
Locked Bag 815
Post Office
CARLTON SOUTH, VIC, 3053

Fax: (03) 9342-2665
Telephone: (03) 9342-2607
Email: margery.kennett@nwhcn.org.au

Final report of the Food Regulation Review

Food: a Growth Industry

The difficulties of balancing public health and consumer protection against the needs of business to be competitive and efficient are nowhere better illustrated than in the area of food regulation. The recently completed Food Regulation Review, chaired by Dr Bill Blair, has spent 12 months considering these issues, while undertaking extensive consultation with government, consumers and all sectors of the food industry (primary industry, manufacturing, retail and export). The final report of the Food Regulation Review, *Food: a Growth Industry*, was released in late August 1998. It makes recommendations for major legislative, procedural and structural reforms to improve the efficiency and effectiveness of the food regulatory system in Australia. The report will be considered by governments, ministerial councils and the Council of Australian Governments.

The report (ISBN 0 642 34518x) is available for sale for $16.95 from **AusInfo** (see below).

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*Formerly known as Commonwealth Government Bookshops

Outbreak of Newcastle disease in commercial poultry flocks, NSW

An outbreak of Newcastle disease (ND) has occurred, involving two commercial poultry flocks in western Sydney (Blacktown and Glenorie) and linked to a third flock at Rylstone160 km north-west of Sydney.

ND is a highly contagious virus disease affecting poultry, cage and aviary birds, and wild birds. There are a number of strains of the disease, which differ in the severity of their clinical signs, ranging from inapparent to a rapidly fatal condition. In its highly virulent form, ND can rapidly cause up to 100 per cent mortality in bird flocks and poses a devastating threat to the poultry industry. Strains are found in most countries but the virulent form has not previously occurred in Australia.

The virus belongs to the family Paramyxoviridae and causes digestive, respiratory and/or nervous signs in birds.

ND does not present any public health risk to consumers of poultry products, including poultry meat, eggs and other chicken products. People exposed to high levels of the virus, for example laboratory workers, may experience conjunctivitis and/or mild influenza-like symptoms.

Control measures have been implemented in accordance with the Australian Veterinary Emergency Plan (Ausvetplan) for ND and include quarantine, surveillance, destruction of poultry, and disinfection of the affected properties. The eradication program is funded jointly by all State and Federal Governments.

More information about Newcastle disease and the outbreak can be found on the NSW Department of Agriculture website at:
http:/www.agric.nsw.gov.au/news/nd/
and on the website of the Office of the Chief Veterinary Officer at:
http://www.dpie.gov.au/ocvo.
A pictorial account is at:
http:/www.agric.nsw.gov.au/news/nd/pictures.htm.

Q fever outbreak in an abattoir in Cooma, NSW

An outbreak of Q fever in workers at an abattoir in Cooma, in the Monaro District of New South Wales, is currently under investigation. To 28 September 1998, out of an estimated workforce of approximately 100, there have been 18 confirmed cases and 12 suspected cases. So far, any suspected cases that have occurred outside the abattoir environment have tested negative. In the majority of suspected and confirmed cases the clinical onset of disease occurred in late August or the first week of September, 1998. Infected stock from outside the Monaro District have been implicated as the source of the epidemic. A screening and vaccination programme of all abattoir employees has been implemented and is nearing completion.

Revision of the infection control guidelines

The Communicable Diseases Network Australia New Zealand (CDNANZ) is reviewing the current infection control guidelines, *Infection Control in the Health Care Setting: Guidelines for the Prevention of Transmission of Infectious Diseases 1996* and *Creutzfeldt-Jakob Disease and Other Human Transmissible Spongiform Encephalopathies: Guidelines on patient management and infection control 1995.*

These guidelines outline the principles of infection control and provide a rationale against which practitioners and health care establishments can develop detailed protocols and systems for infection control that are relevant to their own area of health care. A constantly changing health care environment requires that the guidelines be reviewed to ensure that they continue to provide best practice technical and ethical information.

A Steering Committee has been established to oversee the review, with scientific and administrative support from the National Centre for Disease Control (NCDC).

As part of the review, the Steering Committee is seeking comments from users of the current documents on their utility, and on ways in which they might be improved. To assist the provision of feedback comments, a questionnaire has been developed and is available on the Public Health Division website **http://www.health.gov.au/pubhlth/strateg/communic/review/**. The guidelines can also be viewed on this website.

Completed questionnaires and suggestions should be forwarded to the NCDC at the following address by close of business on 13 November 1998:

Email address: icgreview@health.gov.au

Postal address:

Review of Infection Control Guidelines
National Centre for Disease Control (MDP6)
Department of Health and Family Services
GPO Box 9848
CANBERRA, ACT, 2601

National reporting of *Shiga*-like toxin (verotoxin) producing *Escherichia coli* infections and associated syndromes

Infection with *Shiga*-like toxin (verotoxin) producing strains of *Escherichia coli* (SLTEC or VTEC)♦ has the potential to cause severe and life threatening illness, including haemorrhagic colitis (HC), haemolytic uraemic syndrome (HUS) and thrombotic thrombocytopaenic purpura (TTP). Such strains are found in some animals, and are transmitted to humans through ingestion of undercooked meat (especially minced beef), or other food or water contaminated with animal faeces. Swimming in faecally contaminated lakes has also resulted in cases. Secondary transmission from person to person can occur. While cases are usually sporadic in Australia, two outbreaks, resulting in one death, have been documented. Large outbreaks have occurred overseas and reports of food borne illness resulting from SLTEC strains are increasing world wide.

To improve the understanding of the epidemiology of these infections and facilitate the public health response to cases, a number of States (Queensland, New South Wales, South Australia, Western Australia and Tasmania) have already made SLTEC/VTEC infections and/or HUS/TTP notifiable conditions, and the other States and the Territories are in the process of, or considering, doing so.

On 22 July 1998, members of the Communicable Diseases Network Australia New Zealand (CDNANZ) endorsed case definitions for national reporting of SLTEC (VTEC) infections, HUS and TTP in Australia.♦♦ The Network agreed to report cases to the National Notifiable Diseases Surveillance System (NNDSS) for publication in *CDI*. Cases are reported for the first time in this issue (page 224). The case definitions and explanatory notes are provided in the box below.

National surveillance of HUS is also being undertaken by the Australian Paediatric Surveillance Unit, who are conducting an active surveillance study of HUS in children under the age of 16 years.

♦ also known as enterohaemorrhagic *E. coli* (EHEC)

♦♦ VTEC is already nationally reported in New Zealand.

CDNANZ case definitions for the national surveillance of SLTEC (VTEC), HUS and TTP

***Shiga*-like toxin (verotoxin) producing *E. coli* (SLTEC, VTEC)**

In a clinical specimen from a person with bloody diarrhoea, haemolytic uraemic syndrome (HUS) or thrombotic thrombocytopaenic purpura (TTP):

- isolation of *Shiga*-like toxin (verotoxin) producing *E. coli*;

OR

- identification of Shiga toxin (verotoxin) in *E. coli* OR the gene associated with the production of *Shiga* toxin (verotoxin) in *E. coli*.

Note: The SLTEC/VTEC case definition is for the reporting of confirmed cases to the National Notifiable Diseases Surveillance System. States and Territories may use more sensitive case definitions to identify possible cases for public health follow up. The case definition is not intended to prescribe the laboratory tests that should be done to screen for SLTEC/VTEC infections.

Haemolytic uraemic syndrome (HUS)

A case diagnosed as haemolytic uraemic syndrome (HUS) by a specialist physician, paediatrician, or paediatric nephrologist.

Note: The diagnosis of HUS will generally require the following:

- microangiopathic haemolytic anaemia (Hbg/dl and microscopic evidence of fragmented red cells);

AND

- acute renal impairment (oliguria or anuria, and elevated serum urea, and elevated serum creatinine);

AND

- thrombocytopaenia (platelets d,000/mm^3)

However, the platelet counts may be normal or even high, particularly later in the disease.

Thrombotic thrombocytopaenic purpura (TTP)

A case diagnosed as thrombotic thrombocytopaenic purpura (TTP) by a specialist physician, paediatrician, or paediatric nephrologist.

Note: The diagnosis of TTP will generally require the following:

- microangiopathic haemolytic anaemia (Hbg/dl) and microscopic evidence of fragmented red cells);

AND

- acute renal impairment (oliguria or anuria, and elevated serum urea, and elevated serum creatinine);

AND

- thrombocytopaenia (platelets d,000/mm^3)

Communicable Diseases Surveillance

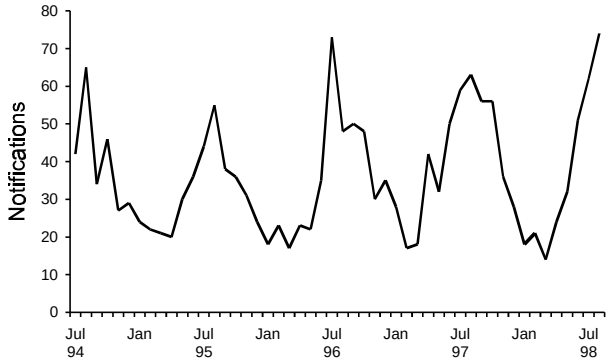
Highlights

Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The CDI Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. The Australian Sentinel Practice Research Network (ASPREN) is a general practitioner-based sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Meningococcal disease

There were 59 reports of meningococcal disease in this period, slightly less than the 64 cases reported for the same period in 1997. Of the 316 cases reported to date in 1998, 311 had onset dates between 1 January 1998 and 16 September 1998, compared with 337 cases and 270 cases in the same periods in 1993 and 1994 respectively. The number of cases in August (74) was higher than in July (62), following the usual seasonal trend (Figure 1). The male:female ratio of 1998 cases to date is 1.17:1 and the age groups with the highest numbers of cases continue to be 0-4 years, 15-19 years and 20-24 years. The State and Territory distribution of cases with onset in 1998 is similar to that seen in the same period in 1997.

Figure 1. Notifications of meningococcal disease, Australia, July 1994 to August 1998, by month of onset



Vaccine preventable diseases

The number of notifications of pertussis infection continues to fall. Although there is a slight increase in the number of reports during this period, examination by date of onset shows that the numbers have fallen in each successive month since December 1997. Most notifications with onset in 1998 are in children aged 5 to 9 (17%), 10 to 14 (15%) and 0 to 4 (11%). The decrease is reflected in the reports from the LabVISE system (Table 3).

A small increase in the number of rubella notifications may represent the start of the seasonal variation expected in the spring.

SLTEC infections, HUS and TTP

With this issue we commence the reporting of *Shiga*-like toxin (verotoxin) producing *Escherichia coli* (SLTEC, VTEC) infections, and the associated syndromes, haemolytic uraemic syndrome (HUS) and thrombotic thrombocytopaenic purpura (TTP). The case definitions for national reporting of these conditions are provided on page 223.

While these conditions are not yet notifiable in all States and Territories, the rarity, severity and public health importance of HUS ensures that most cases are voluntarily reported to State and Territory health authorities. The level of voluntary reporting of SLTEC infections is not known. South Australia is currently the only State in which TTP is notifiable as a separate condition.

Although national reporting only commenced in this reporting period, most States and Territories have provided information about all cases in their records for the 1998 year to date.

In this reporting period, 3 sporadic cases of HUS have been recorded by New South Wales and 1 case of SLTEC infection by South Australia. To date in 1998, the total number of HUS cases reported has been 10 (NSW 5, South Australia 3 and Western Australia 2) and the total number of SLTEC infections has been 14 (South Australia 13 and New South Wales 1).

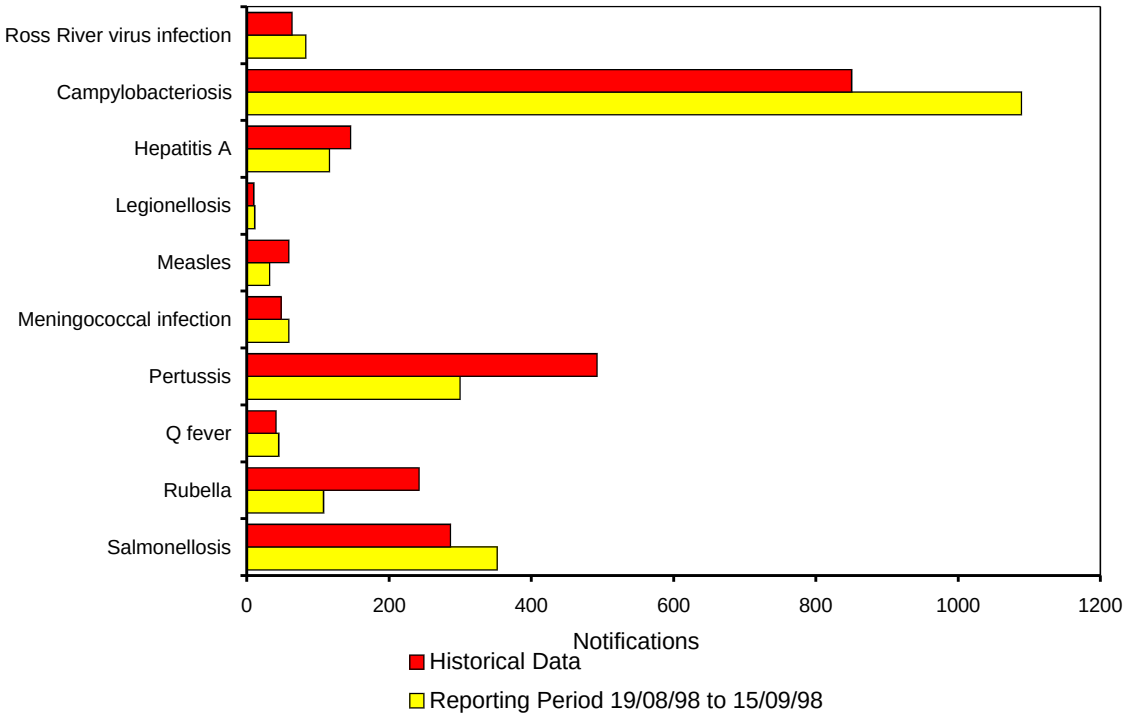
Tables

There were 4,339 notifications to the National Notifiable Diseases Surveillance System (NNDSS) in the four week period, 18 August to 16 September 1998 (Tables 1 and 2). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 2).

There were 2,868 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the four week period, 13 August to 9 September 1998 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 32 to 35, ending 6 September 1998, are included in this issue of *CDI* (Table 5).

Figure 2. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in the corresponding 4 week periods of the last 3 years and the 2 week periods immediately preceding and following those.

Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 18 August to 16 September 1998

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
H. influenzae type b infection	0	0	0	0	0	1	0	0	1	3	22	36
Measles[3]	1	8	0	0	0	7	9	7	32	49	270	420
Mumps	0	4	1	10	2	0	2	10	29	19	140	144
Pertussis	5	111	1	69	32	6	70	6	300	827	4,955	5,688
Rubella[4]	4	4	0	66	1	0	29	4	108	116	598	984
Tetanus	0	0	0	0	0	0	0	1	1	0	4	7

NN. Not Notifiable

1. No notification of poliomyelitis has been received since 1986.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. The total number of measles notifications for 1998 has been revised downwards because of a reclassification of 79 cases previously notified as measles by Victoria. These cases have been reclassified as not measles following results of serology.
4. Includes congenital rubella.

Table 2. Notifications of diseases received by State and Territory health authorities in the period 18 August to 16 September 1998

Disease[1,2,3,4]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998[5]	Year to date 1997
Arbovirus infection (NEC)[6]	0	0	0	1	0	0	1	0	2	4	60	108
Barmah Forest virus infection	1	5	1	21	0	0	1	1	30	23	442	538
Brucellosis	0	0	0	2	0	0	0	0	2	8	30	27
Campylobacteriosis[7]	31	-	14	358	223	37	276	150	1,089	798	7,838	7,986
Chlamydial infection (NEC)[8]	15	NN	57	406	88	12	184	147	909	715	7,669	6,615
Cholera	0	0	0	1	0	0	0	0	1	0	4	2
Dengue	1	4	2	14	0	0	1	2	24	2	380	195
Donovanosis	0	NN	2	0	NN	0	0	0	2	0	27	23
Gonococcal infection[9]	1	60	105	79	12	2	75	73	407	300	3,873	3,192
Hepatitis A	0	31	1	68	7	1	3	5	116	168	2,099	2,331
Hepatitis B incident[5]	0	2	2	4	1	1	5	0	15	27	164	185
Hepatitis C incident[10]	0	9	0	-	4	0	-	-	13	2	124	51
Hepatitis C unspecified[5]	29	NN	34	295	NN	18	5	80	461	746	5,878	6,832
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	0	4	14
Haemolytic uraemic syndrome[11]	NN	3	NN	0	0	0	NN	0	3	NA	10	NA
Hydatid infection	0	0	0	4	0	0	3	0	7	11	33	39
Legionellosis	0	2	0	1	3	1	2	2	11	6	174	110
Leprosy	0	0	0	0	0	0	0	0	0	2	2	10
Leptospirosis	0	1	0	3	0	0	2	0	6	9	115	89
Listeriosis	0	1	0	0	0	0	1	1	3	2	41	58
Malaria	1	5	0	12	3	0	4	0	25	76	568	612
Meningococcal infection	0	20	0	11	5	3	8	12	59	64	316	337
Ornithosis	0	NN	0	0	0	0	1	0	1	2	28	39
Q Fever	0	7	0	36	2	0	0	0	45	35	397	430
Ross River virus infection	1	8	4	64	1	1	0	4	83	66	2,436	6,362
Salmonellosis (NEC)	2	72	25	123	26	4	69	31	352	310	5,751	5,149
Shigellosis[7]	0	-	8	5	6	0	9	6	34	37	448	590
SLTEC infections[12]	NN	0	NN	NN	1	0	NN	NN	1	NA	14	NA
Syphilis[13]	1	36	37	91	1	0	0	5	171	108	1,071	929
Tuberculosis	3	25	2	13	8	2	23	5	81	75	725	725
Typhoid[14]	0	1	0	0	0	0	1	1	3	2	56	57
Yersiniosis (NEC)[7]	1	-	0	3	3	0	3	0	10	11	169	190

1. For diseases preventable by routine childhood immunisation, see Table 1
2. For HIV and AIDS, see Tables 6 and 7.
3. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
4. No notifications have been received during 1998 for the following rare diseases: botulism (foodborne), lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers. There have also been no cases of thromotic thrombocytopaenic purpura (TTP), which became nationally reportable in August 1998.
5. Data from Victoria for 1998 are incomplete.
6. NT: includes Barmah Forest virus.
7. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
8. WA: genital only
9. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
10. Qld, Vic and WA incident cases of Hepatitis C are not separately reported.
11. Nationally reportable from August 1998
12. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC) became nationally reportable in August 1998.
13. Includes congenital syphilis
14. NSW, Qld, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified

\- Elsewhere Classified.

NA Not applicable, as reporting for this condtition did not commence until 1998.

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 13 August to 9 September 1998, and total reports for the year

	State or Territory[1]									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Total reported in *CDI* in 1998
Measles, mumps, rubella										
Measles virus		1						4	5	51
Mumps virus								4	4	31
Rubella virus				11	1		2	1	15	90
Hepatitis viruses										
Hepatitis A virus	4	4	1	9	3			2	23	311
Hepatitis D virus				1					1	4
Arboviruses										
Ross River virus		1	1	20	1			4	27	581
Barmah Forest virus							2		2	26
Dengue not typed								3	3	28
Flavivirus (unspecified)				3			4		7	56
Adenoviruses										
Adenovirus type 2							3		3	18
Adenovirus type 3					1		3		4	30
Adenovirus type 8							1		1	4
Adenovirus type 37							1		1	2
Adenovirus type 40								4	4	9
Adenovirus not typed/pending	4	36		4	15		17	13	89	560
Herpes viruses										
Cytomegalovirus		10		10	5		31	11	67	559
Varicella-zoster virus	3	18	1	32	5		22	20	101	919
Epstein-Barr virus		43		48	21		16	8	136	1,245
Other DNA viruses										
Parvovirus				3	2		20	12	37	166
Picorna virus family										
Echovirus type 2							1		1	1
Echovirus type 4	1								1	3
Echovirus type 11		1					1		2	26
Echovirus type 22		1							1	6
Echovirus not typed/pending							1		1	1
Poliovirus type 1 (uncharacterised)		2							2	5
Poliovirus type 2 (uncharacterised)		3					3		6	11
Poliovirus type 3 (uncharacterised)		2							2	3
Rhinovirus (all types)	3	28			2		5	6	44	353
Enterovirus type 71 (BCR)							1		1	1
Enterovirus not typed/pending	2	20		4		1	5	28	60	368
Ortho/paramyxoviruses										
Influenza A virus	1	409	2	42	88	4	134	34	714	2,259
Influenza B virus		1			7		4	3	15	140
Parainfluenza virus type 1	1				16		2	2	21	265
Parainfluenza virus type 2								1	1	30
Parainfluenza virus type 3		4			4		5	8	21	244
Respiratory syncytial virus	107	142	1	25	79	13	489	142	998	3,079
Other RNA viruses										
HTLV-1								1	1	14
Rotavirus	1	49			21	9	52	21	153	686

Table 3. Virology and serology laboratory reports by State or Territory[1] for the reporting period 13 August to 9 September 1998, and total reports for the year (continued)

	State or Territory[1]									
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Total reported in *CDI* in 1998
Other										
Chlamydia trachomatis not typed		21	6	54	17		2	49	149	2,704
Chlamydia psittaci							3		3	36
Chlamydia species	10	6		1					17	52
Mycoplasma pneumoniae		14	1	27	8		25	3	78	983
Coxiella burnetii (Q fever)		3		4	1		3		11	90
Bordetella pertussis		4		11			16	3	34	796
Cryptococcus species		1							1	12
TOTAL	137	824	13	309	297	27	874	387	2,868	16,858

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.

Table 4. Virology and serology laboratory reports by contributing laboratories for the reporting period 13 August to 9 September 1998

State or Territory	Laboratory	Reports
Australian Capital Territory	The Canberra Hospital	166
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	471
	New Children's Hospital, Westmead	188
	Royal Prince Alfred Hospital, Camperdown	48
	South West Area Pathology Service, Liverpool	68
Queensland	Queensland Medical Laboratory, West End	331
	Townsville General Hospital	12
South Australia	Institute of Medical and Veterinary Science, Adelaide	295
Tasmania	Northern Tasmanian Pathology Service, Launceston	24
Victoria	Monash Medical Centre, Melbourne	85
	Royal Children's Hospital, Melbourne	572
	Victorian Infectious Diseases Reference Laboratory, Fairfield	215
Western Australia	PathCentre Virology, Perth	280
	Princess Margaret Hospital, Perth	113
TOTAL		2,868

Table 5. Australian Sentinel Practice Research Network reports, weeks 32 to 35, 1998

Week number	32		33		34		35	
Week ending on	16 August 1998		23 August 1998		30 August 1998		6 September 1998	
Doctors reporting	58		46		51		41	
Total encounters	8029		6670		7048		5964	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	174	21.7	136	20.4	123	17.5	79	13.2
Rubella	1	0.1	0	0.0	0	0.0	2	0.3
Measles	0	0.0	0	0.0	0	0.0	1	0.2
Chickenpox	8	1.0	2	0.3	11	1.6	9	1.5
Pertussis	4	0.5	0	0.0	3	0.4	2	0.3
HIV testing (patient initiated)	7	0.9	8	1.2	9	1.3	9	1.5
HIV testing (doctor initiated)	6	0.7	6	0.9	2	0.3	2	0.3
Td (ADT) vaccine	68	8.5	58	8.7	51	7.2	34	5.7
Pertussis vaccination	38	4.7	19	2.8	37	5.2	28	4.7
Reaction to pertussis vaccine	0	0.0	0	0.0	0	0.0	0	0.0
Ross River virus infection	2	0.2	1	0.1	2	0.3	0	0.0
Gastroenteritis	69	8.6	51	7.6	66	9.4	69	11.6

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of more than 40 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1998;22:4-5.

LabVISE is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence every four weeks. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1998;22:8.

ASPREN currently comprises about 100 general practitioners from throughout the country. Up to 9,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance in 1998. CDI reports the consultation rates for all of these. For further information, including case definitions, see CDI 1998;22:5-6.

Additional Reports

National Influenza Surveillance, 1998

Three types of data are included in National Influenza Surveillance, 1998. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network, Department of Human Services (Victoria), Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health (Northern Territory); laboratory surveillance data from the Communicable Diseases Intelligence Virology and Serology Laboratory Reporting Scheme, LabVISE, and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. For further information about these schemes, see CDI 1998; 22:83.

Sentinel General Practitioner Surveillance

Consultation rates for influenza like illness recorded by the New South Wales, Victorian and ASPREN Schemes have declined in the last 4 weeks (Figure 3). The highest consultation rates of 17 per 1,000 have been reported by the New South Wales Sentinel Practitioner Scheme. A late seasonal peak in reports of influenza-like illness was reported by the Tropical Influenza Surveillance Scheme in early August, but over the last month these rates have declined from 19.7 to 10.4 per 1,000. The winter peak for reported consultation rates for influenza-type illness across all schemes has been less than the 50 per 1,000 consultations reported in late July and early August of last year.

Figure 3. Sentinel general practitioner consultation rates, Australia, 1998, by week and scheme.

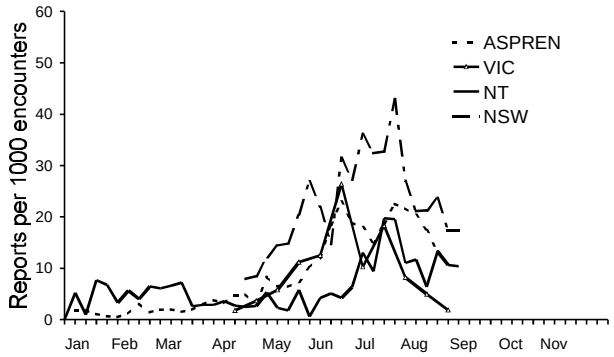


Figure 4. Influenza laboratory reports, Australia, 1998, by virus type and week of specimen collection

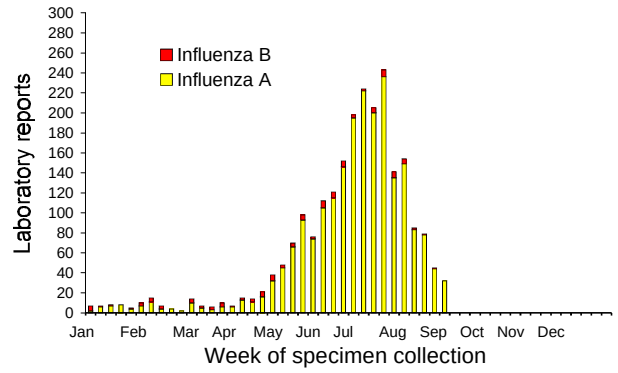
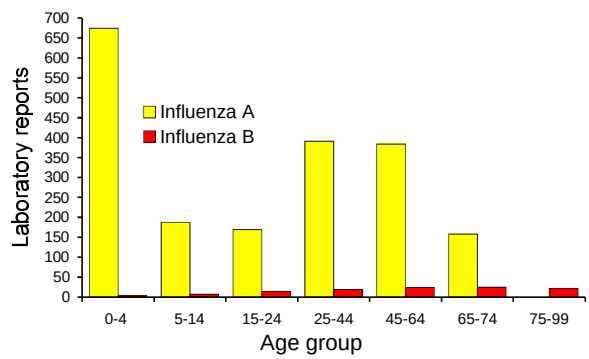


Figure 5. Influenza A and B laboratory reports, Australia, 1998, by age group



Laboratory Surveillance

There have been 2288 laboratory reports of influenza for the year to date. Of these, 2175 (95%) were influenza A and 113 (5 %) influenza B (Figure 4). The number of influenza A reports for this year is greater than those reported over the same period for all years dating back to 1993, reflecting an increase in the level of laboratory testing in 1998. The proportion of influenza A cases is almost as high as was reported in 1996, when 98% of all laboratory reports over the same time period were for influenza A. Of the laboratory reports of influenza A, 674 (31%) were in children less than 4 year of age (Figure 5).

Absenteeism surveillance

Rates of absenteeism in Australia Post employees for three consecutive days of each week have been reported since late April. Absenteeism rates to 23 September 1998 have averaged 0.24% per week. There has been no definite peak in absenteeism rates coinciding with the peak levels of influenza reported by the Sentinel General Practitioner or LabVISE schemes.

Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents on a quarterly basis. The antibiotics which are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. When in vitro resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level resistance to the tetracyclines. Tetracyclines are however not a recommended therapy for gonorrhoea. Comparability of data is achieved by means of a standardised system of testing and a programme-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented.

Reporting period 1 January to 31 March 1998

The AGSP laboratories examined 877 isolates of *Neisseria gonorrhoeae* for sensitivity to the penicillins, ceftriaxone, quinolones and spectinomycin and for high level resistance to the tetracyclines in the March quarter of 1998.

Penicillins

Resistance to this group of antibiotics (penicillin, ampicillin, amoxycillin) was present in a high proportion of isolates examined in Adelaide (40%), Melbourne (39%) and Sydney (37%). In Brisbane and Perth the proportion of penicillin-resistant strains was 15% and 8% respectively. Figure 6 shows the proportion of isolates fully sensitive, less sensitive or relatively resistant to the penicillins by chromosomal mechanisms and the proportion of penicillinase-producing gonococci (PPNG) in different regions and as aggregated data for Australia. PPNG and

relatively resistant isolates usually fail to respond to therapy with the penicillins. Those in the fully sensitive and less sensitive categories (minimal inhibitory concentration, MIC, ≤ 0.5 mg/L) usually respond to a regimen of standard treatment with the above penicillins.

There were 57 PPNG identified in this reporting period (6.5% of all isolates). These were distributed widely with 12 PPNG reported from Melbourne, 21 from Sydney, 10 each from Perth and Brisbane and 4 from the Northern Territory. Infections with PPNG were most frequently acquired overseas, particularly in the South East Asian countries often visited by Australians. Among the counties where infections with PPNG were acquired were the Philippines, Thailand, Singapore, Iceland, Indonesia, Vietnam, and Vanuatu. Local acquisition was also recorded in Sydney.

Of relatively greater importance than PPNG were the 164 (19%) of all isolates resistant to the penicillins by separate chromosomal mechanisms. These so called CMRNG were most often seen in Sydney (97 strains, 30%), Melbourne (39 strains, 30%), Brisbane (14 strains, 9%) and Adelaide (10 strains, 40%). Four relatively resistant isolates were seen in the Northern Territory.

Ceftriaxone and spectinomycin.

Although all isolates from all parts of Australia were sensitive to these injectable agents, a small number of isolates showed some decreased sensitivity to ceftriaxone.

Quinolone antibiotics (Ciprofloxacin, norfloxacin and enoxacin)

Fifty five isolates (6.3%) throughout Australia had altered resistance to this group of antibiotics (QRNG) with 40 of these showing high level resistance. Thirty one QRNG (10%) were detected in Sydney, 11 (7%) in Brisbane and 8 (6%) in Perth, with smaller numbers in the other centres.

An increase in rates of isolation of QRNG has been noted in AGSP reports in 1997. Additionally the appearance of QRNG in infections acquired locally, especially in Sydney but also in Melbourne, was specifically mentioned. The high rate of locally acquired high level resistance to quinolone antibiotics was maintained in Sydney in this quarter but was not confirmed in any other centre. Patients infected with QRNG overseas acquired the infections in Indonesia, Iceland, Vanuatu,Thailand, and the Philippines.

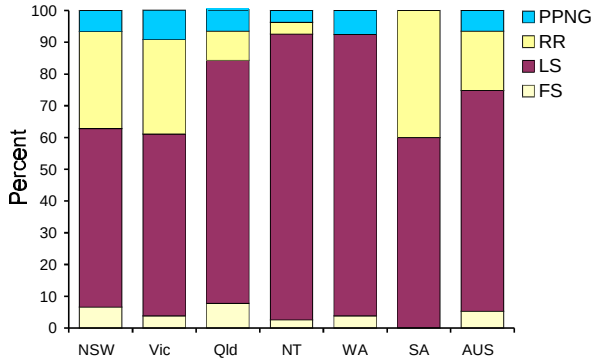
In the corresponding period of 1997, there were 49 QRNG comprising 7.2% of all Australian isolates.

The quinolone agents are the oral agents most often used in centres where penicillins are ineffective. The appearance of quinolone resistance reduces options for successful treatment of gonorrhoea.

High level tetracycline resistance (TRNG)

Forty eight TRNG were detected throughout Australia (5.5% of all strains) with isolates of this type again present in most centres. The highest proportion of TRNG was found in Perth where the 11 TRNG represented 8.3% of all isolates. TRNG were also prominent in Brisbane (7 isolates, 4.6%) , Sydney (18 isolates, 5.7%), Melbourne (9 isolates, 7%) and the Northern Territory (3 isolates). Indonesia was the overseas source of acquisition most often identified, but TRNG strains were also acquired in Thailand, the Philippines, Vietnam, Singapore and the USA. Local acquisition was also recorded.

Figure 6. Penicillin resistance of gonococcal isolates, Australia, 1 January - 31 March 1998, by region



FS Fully sensitive to penicillin, MIC = 0.03 mg/l.
LS Less sensitive to penicillin, MIC 0.06 - 0.5 mg/l
RR relatively resistant to penicillin, MIC ,= 1 mg/l
PPNG Penicillinase producing *Neisseria gonorrhoeae*

HIV and AIDS Surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648 Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for March 1 to March 31 1998, as reported to 30 June 1998, are included in this issue of CDI (Tables 6 and 7).

Table 6. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 30 April 1998, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
HIV diagnoses	Female	1	3	0	2	0	0	0	1	7	5	25	28
	Male	2	22	1	14	0	0	9	3	51	69	232	266
	Sex not reported	0	3	0	0	0	0	0	0	3	2	6	9
	Total[1]	3	28	1	16	0	0	9	4	61	77	263	384
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	4	2	13
	Male	1	0	0	3	0	0	1	0	5	31	36	119
	Total[1]	1	0	0	3		0	1	0	5	35	38	132
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	2	5
	Male	0	2	0	0	1	0	0	0	3	13	24	89
	Total[1]	0	2	0	0	1	0	0	0	3	14	26	94

1. Persons whose sex was reported as transgender are included in the totals.

Table 7. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 30 April 1998, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	21	549	7	125	52	4	193	87	1,038
	Male	182	10,288	94	1,805	628	77	3,719	856	17,649
	Sex not reported	0	262	0	0	0	0	28	0	290
	Total[1]	203	11,118	101	1,936	680	81	3,950	946	19,015
AIDS diagnoses	Female	7	157	0	45	19	2	64	23	317
	Male	81	4,331	31	766	320	41	1,527	337	7,434
	Total[1]	88	4,499	31	813	339	43	1,598	362	7,773
AIDS deaths	Female	2	112	0	28	14	2	45	16	219
	Male	62	3,042	23	529	217	27	1,204	241	5,345
	Total[1]	64	3,161	23	559	231	29	1,255	258	5,580

1.559 Persons whose sex was reported as transgender are included in the totals.

Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease Australian encephalitis in humans. Currently 26 flocks are maintained in the north of Western Australia, seven in the Northern Territory, nine in New South Wales and ten in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information see CDI 1998;22:7

AK Broom[1], J Azuolas[2], L Hueston[3], JS Mackenzie[4], L Melville[5], DW Smith[6] and PI Whelan[7]

1. Department of Microbiology, The University of Western Australia
2. Veterinary Research Institute, Victoria
3. Virology Department, Institute of Clinical Pathology and Medical Research, New South Wales
4. Department of Microbiology, The University of Queensland
5. Berrimah Agricultural Research Centre, Northern Territory
6. PathCentre, Western Australia
7. Department of Health and Community Services, Northern Territory

Sentinel chicken serology was carried out for 17 of the 28 flocks in Western Australia in July 1998, and 21 of the 28

flocks in August 1998. There was one new seroconversion in the Kununurra flock to flavivirus only and this was confirmed at a later bleed. There were no other seroconversions in July or August which is what would be expected at this time of the year.

Sentinel chickens from the Northern Territory were also tested in our laboratory; three of 7 flocks in July 1998 and all 7 flocks in August 1998. There was one new seroconversion in the Leanyer flock to flavivirus only which does not appear to be MVE or Kunjin virus. This was confirmed at a later bleed. The seroconversion, to flavivirus only, in the Gove flock in June 1998 was not confirmed at a later bleed and has been removed from the results.

Childhood Immunisation Coverage

Tables 8 and 9 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised according to the Australian Standard Vaccination Schedule at age 12 months, for the cohort born between 1 January and 30 March 1997, and at age 24 months, for the cohort born between 1 January and 31 March 1996 . The assessment date for both cohorts was 31 March 1998. The 12 months age data for the 1996 cohort, and a full description of the methodology used can be found in CDI 1998;22:36-37.

Table 8. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1997; assessment date 31 March 1998.

	State or Territory								
Vaccine	ACT	NSW	NT[1]	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,036	21,809	896	11,648	4,619	1,567	15,273	6,371	63,219
DTP (%)	84.7	80.9	64.3	85.1	82.7	84.0	84.5	78.7	82.4
OPV (%)	84.6	80.7	65.5	85.4	82.9	84.5	84.7	79.1	82.5
Hib (%)	81.9	80.2	69.3	85.9	80.8	83.2	84.3	78.6	82.1
Fully Immunised (%)	81.0	78.5	59.2	83.2	79.3	82.3	83.0	77.0	80.2
Change in fully immunised since last quarter (%)	-0.9	+2.8	-2.4	+0.7	+0.7	+0.6	+1.5	+1.9	+1.6

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Family Services. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone 02 6203 6185

Table 9. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1996; assessment date 31 March 1998.[1]

	State or Territory								
Vaccine	ACT	NSW	NT[1]	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,065	21,784	954	12,292	4,867	1,623	15,735	6,455	64,775
DTP (%)	78.8	75.6	59.4	78.5	76.3	76.6	76.9	72.5	76.0
OPV (%)	83.9	80.4	69.8	85.9	84.6	84.6	87.2	73.5	82.7
Hib (%)	74.4	76.0	64.0	78.9	76.7	76.9	77.5	72.7	76.5
MMR (%)	86.4	80.2	70.5	86.6	82.3	84.4	85.2	76.3	82.5
Fully Immunised (%)[2]	69.0	62.3	48.8	68.3	62.8	63.4	66.9	54.8	63.8

1. The 12 month age datea for this cohort was published in *CDI* 1998,22:36-37.
2. These data relating to 2 year old children should be considered as preliminary. The proportions fully immunised appear low compared with the proportions for individual vaccines . HIC are checking these calculations.

Serious Adverse Events Following Vaccination Surveillance Scheme

The Serious Adverse Events Following Vaccination Surveillance Scheme (SAEFVSS) is a national surveillance scheme which monitors the serious adverse events that occur rarely following vaccination. More details of the scheme were published in CDI 1997:21;8. Acceptance of a report does not imply a causal relationship between administration of the vaccine and the medical outcome, nor that the report has been verified as to the accuracy of its contents.. It is estimated that 250,000 doses of vaccines are administered every month to Australian children under the age of six years.

Results for the period 2 July to 1 September 1998.

There were 322 reports of serious adverse events following vaccination for this reporting period (Table 10). Onset dates were from 1995 to 1998, the majority (78%) being in 1998. Reports were received from the Australian Capital Territory (12), New South Wales (45), the Northern Territory (3), Queensland (78), South Australia (39), Victoria (17) and Western Australia (128). No reports were received from Tasmania for this period.

The most frequently reported events following vaccination were persistent screaming (205 cases, 63%), followed by other reactions (50 cases, 15.5%), temperature of 40.5^{o} C or more (26 cases, 8.1%) and hypotonic/hyporesponsive episodes (24 cases, 7.5%). One death within 30 days of immunisation was reported from Western Australia. The baby was two months old, and the cause of death was determined to be sudden infant death syndrome (SIDS) by the coroner.

South Australia reported 34 adverse events associated with BCG vaccination of which 20 had lymphadenitis, 11 had a local abscess and 3 had skin lesions around the vaccination site. Onset dates were missing for 2 cases. There was 1 case with an onset date in 1995, 4 cases with onset dates in 1996, 15 cases in 1997 and 12 in 1998. Of the 12 cases in 1998, 10 occured in the January to April period.

Of the 19 reactions associated with MMR vaccine, 12 occurred after the commencement of the Measles Control Campaign in August 1998. Although the incidence of adverse events is being monitored closely and reported regularly throughout the Campaign (see the Measles Campaign Update on p 220 of this issue of *CDI*), there will be a time lag before all the adverse events associated with the Campaign are reported through the SAEFVSS

Thirty-one of the 322 cases were hospitalised. There was incomplete follow-up information for the 34 cases associated with BCG vaccination in South Australia. All other cases had recovered at the time of reporting.

Two hundred and sixty-five cases (82%) were associated with diptheria-tetanus-pertussis (DTP) vaccine, either alone or in combination with other vaccines. Of these, 66% were associated with the first dose of DTP and 26% with the second.

Table 10. Adverse events following vaccination reported in the period 2 July to 1 September 1998.[1]

Event	Vaccines									Reporting States or Territories	Total reports for this period
	DTP	DTP/Hib	DTP/OPV/Hib	DTP/OPV/other	DTP/OPV	DTP/OPV/Hib/ Hep B	MMR	Hep B	Other[2]		
Persistent screaming	151	0	46	1	2	2	3	0	0	ACT, NSW, QLD, Vic, WA	205
Hypotonic/hyporesponsive episode	13	0	6	0	2	0	3	0	0	ACT, NSW, QLD, Vic, WA	24
Temperature of 40.5°C or more	17	1	6	0	1	0	1	0	0	ACT, NSW, QLD, WA	26
Convulsions	4	1	2	0	1	0	5	0	0	NSW, QLD, SA, Vic, WA	13
Anaphylaxis	0	0	0	0	0	0	0	0	0		
Shock	0	0	0	0	0	0	1	0	0	WA	1
Death	0	0	1	0	0	0	0	0	0	WA	1
Other[3]	4	0	4	0	0	0	6	1	35	ACT,NSW, NT, QLD, SA, Vic, WA	50
TOTAL	189	2	65	1	6	2	19	1	35		322[4]

1. Events with onset dates from 1995 to 1998 were reported in this period.
2. Includes influenza, DTPa, CDT, OPV, Hepatitis B, pneumococcal, BCGand ADT vaccines and rabies immunoglobulin (HRIG)
3. Includes lymphadenitis, local reations, fever less than 40.5° and non-specific events such as vomiting.
4. Total incldes two reports where the type of event was not stated.

Bulletin Board

CRC for Water Quality and Treatment, Water Services Association of Australia and Australian Water and Wastewater Association
Cryptosporidium in Water Conference
5-6 October 1998
Carlton Crest Hotel, Melbourne
Phone: 02 9413 1288
Fax: 02 9413 1047
Email: http://www.med.monash.edu.au/epidemiology/crc/CONFER/crypconf.htm

The Australian Institute of Environmental Health
25th National Conference
25-30 October 1998
Ana Hotel, Surfers Paradise, Queensland
Phone: 07 334 2299

The Public Health Association of Australia Inc.,
6th National Conference on Immunisation
Immunisation; Beyond 2000
4-5 November 1998
Hilton on the Park, Melbourne
Phone: 02 6283 2373
Email: conference@pha.org.au

Centers for Disease Control (USA) and the World Health Organization
2nd International Conference on Emerging Zoonoses
5-9 November 1998
Strasbourg, France
Phone: +33 1 474 22016
Fax: +33 1 426 51725
Email: trgt@netvision.net.il

Communicable Diseases Network Australia New Zealand
Conference: *Control of Communicable Diseases in Australia*
10 November 1998
Australian National University, Canberra
Phone: 02 6289 8245
Fax: 02 62897791
Email: ccd.conf@health.gov.au

National Centre for Epidemiology and Population Health
Conference: *Developing Health*
11-12 November 1998
Canberra
Phone: 02 6249 5627
Fax: 02 6249 0740
Email: dev.health@nceph.anu.edu.au

The Australasian Society for HIV Medicine
19th Annual Conference
18-21 November 1998
Newcastle, venue to be advised
Phone: 02 9382 1656
Fax: 02 9382 3699
Email: B.Pearlman@unsw.edu.au

The Australian Society for Microbiology Inc.
The 11th International Conference
International Congress of Virology
9-13 August 1999
International Congress of Bacteriology and Applied Microbiology
9-13 August 1999
International Congress of Mycology
16-20 August 1999
Sydney, New South Wales
Fax: 03 9262 3135
Email: tourhosts@tourhosts.com.au

The Public Health Association of Australia Inc.
31st Annual Conference
26-29 September 1999
Carlton Hotel
Darwin, Northern Territory
Details: PO Box 319
Curtin ACT 2605
Email: conference@pha.org.au

Advance notice

Royal North Shore Hospital
Conference: *Outpatient Parental Therapy - beyond 2000*
17-22 September 2000
Fairmont Resort
Luera, New South Wales
Phone: 02 9956 8333
Fax: 02 0056 5154
Email: confact@conferenceaction.com.au

Health education resource

Talking about HIV/AIDS in the Kimberley, written by clinical psychologist, Pat Lowe, and illustrated by Carol Tang Wei, is a health education and counselling guide for use by health professionals working with Kimberley Aboriginal people. It can be purchased for $60 (postage and packing included) through Ms Ros Cain, of the Kimberley Public Health Unit, PMB 912, Derby WA 6728. Phone 08 9191 1144 or fax 08 9193 13 78

The CDI Bulletin Board is provided as a service to readers. Every effort has been made to provide accurate information, but readers are advised to contact the relevant organisation for confirmation of details. Information about the availability of resources is included when space allows. Inclusion of a resource on the Bulletin Board does not imply endorsement of the resource by either the Communicable Diseases Network Australia New Zealand or the Commonwealth Department of Health and Family Services.

Contributions to the Bulletin Board are invited from those organisations with forthcoming events relevant to communicable disease control.

Overseas briefs

Source: World Health Organization (WHO)

Cholera

Bhutan

Between 15 August 1998 and 28 August 1998, a total of 13 cases were officially notified to WHO. Bhutan has suffered previous cholera outbreaks in 1992 (494 cases) and in 1995 (25 cases).

Afghanistan (update)

As of 2 September 1998, the national outbreak of acute gastroenteritis and suspected cholera, which began in July, was reported as continuing and worsening. Six hospitals in Kabul reported 1400 cases of acute severe diarrhoea, gastroenteritis, and suspected cholera in one week. Between 19 - 26 August 1998, Bamyan reported 328 cases of acute severe diarrhoea and suspected cholera, including 28 deaths. In Herat, 1 died and 86 were hospitalised with acute gastroenteritis. Other areas of Afghanistan involved in the outbreak included Baghlan, Laghman, Takhar, Samangan, Kunduz, Badakhshan, Uruzgan and Ghazni. Control of the outbreak, and city-wide and nation-wide efforts to provide long-term improvements in water and sanitation, are being hampered by the continuing civil war and isolation of many of the areas affected.

Comoros Islands (update)

The country, which consists of 4 islands (Grande Comore, Moheli, Anjouan and Mayotte) has been experiencing a cholera outbreak since February 1998. By mid-August, the cumulative total for 1998 had reached 3199 cases and 40 deaths. Most cases have occurred in a number of districts on the island of Grande Comore, including the capital city, Moroni. However, at least one case has occurred on the island of Mayotte. Poor basic sanitation, very limited personal and environmental hygiene and poor access to safe water are the major contributors to the outbreak.

Armenia

An outbreak of cholera has occurred in a village, about 60 km from Yerevan. As of 6 September, a total of 25 cases had been reported. Cholera was confirmed by laboratory analysis as *Vibrio cholerae* El Tor.

Uganda (update)

As of 21 September 1998, a total of 43911 cases and 1777 deaths (case fatality rate 4%) had been reported in Uganda since the beginning of the epidemic in late 1997. There was a stable or a downward trend in most areas, but the districts of Arua and Moyo in the Northern Region, Kamuli in the Eastern Region and Bushenyi in the Western Region were still reporting high numbers of cases.

Sierra Leone

As of 16 September 1998, the outbreak of cholera affecting Sierra Leone had resulted in 1770 cases and 55 deaths. The organism has been identified by the Pasteur Institute in Abidjan, Cote d'Ivoire, as *Vibrio cholerae* O1 El Tor.

Typhoid fever

Kyrgyzstan

A outbreak of typhoid fever has occurred in Osh Oblast, beginning on 7 September 1998. As of 21 September 1998, 458 patients had been hospitalised and 46 cases had been confirmed as typhoid fever.

Editor: Bronwen Harvey

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Canberra Mailing, PO Box 650, Fyshwick ACT 2609, fax (02) 6269 1212

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Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereeing process. Instructions to authors can be found in *CDI* 1998;22:9.