Meningococcal disease in Australia; looking at the past, thinking of the future

Mahomed Patel, National Centre for Epidemiology and Population Health, Australian National University, Canberra, Australian Capital Territory, 0200

Abstract

In 1987 an unexpected change in the epidemiology of meningococcal disease began in Australia. The change was accompanied by an outbreak of serogroup A meningococcal disease among Aboriginal central Australians, and was followed by a progressive rise in notifications of disease caused by both serogroup B and C nationwide. Over the last 4 years, the notification rate has plateaued at 2.1-2.3 per 100,000 population. Virulent clonal groups of serogroup A and C meningococci that have caused outbreaks appear to be identical to strains that have caused large outbreaks in other countries. We cannot predict where and when the next outbreak will occur. However, we can plan to respond swiftly when it does. This report presents an overview of the observed trends, the association between the microbiology and epidemiology of meningococcal disease, and the relevance of this association to outbreaks, with recommendations for management. *Comm Dis Intell* 1997;21:233-236.

Introduction

Neisseria meningitidis is one of the few endemic pathogens in industrialised countries that healthy children and young adults are particularly susceptible to. It can be fatal within a few hours of onset. The threat of meningococcal disease in child-care centres and schools is alarming, and demands swift action by public health authorities. Although outbreaks of the disease attract media and public attention, most cases are sporadic and cannot be linked to another case. However the public health response to sporadic cases is still demanding, with an average of 22 close contacts per case receiving chemoprophylaxis[1]. If the case attended a child-care centre, the number of contacts requiring such attention may be as high as 72[2]. Over the last 4 years, between 350 and 400 cases of meningococcal disease have been notified annually in Australia[3], suggesting that several thousand contacts may have needed follow-up. A cluster of cases, by contrast, is a rare event and historically, very few regions in Australia have experienced this in any one year.

The rising incidence of disease in Australia over the last decade[4-7], as in other industrialised countries[1,8-11] should serve as a warning of possible recurrences. Canada[11,12], the United Kingdom[13], New Zealand[1,14] and the United States of America[8] have had to conduct extensive vaccination programs in response to outbreaks. In Australia, by contrast, vaccination programs have been of a smaller magnitude[7,15-17]. The need may arise for larger

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vaccination programs to control outbreaks in Australia.

The incidence of meningococcal disease in Australia overall rises in June and peaks by October each year. However, it differs from the other types of bacterial meningitis such as *Haemophilus influenzae* type b disease and pneumococcal disease, in its characteristically unpredictable rise and fall in incidence from region to region**.** A close examination of national and global trends suggests that it may be possible to anticipate future outbreaks, enabling us to prepare a response.

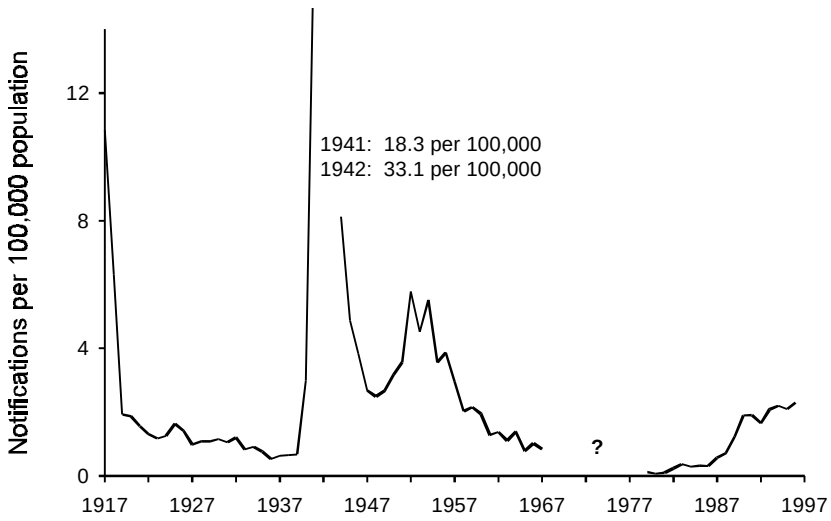
Microbial characteristics and epidemiology

The epidemiology of meningococcal disease is inextricably linked to the microbial characteristics of *N. meningitidis.* The microbe has been subdivided into 13 serogroups. In Australia, over 90% of invasive isolates are serogroups B and C. Further characterisation of invasive isolates is invaluable for guiding public health action, and for following the global spread of invasive strains analogous to that accompanying the antigenic shifts and drifts of the influenza virus.

The three major serogroups of meningococci cause differing patterns of disease in the community. Serogroup A meningococci are associated with explosive epidemics of meningitis, and very high attack rates of disease, up to 500 per 100,000[18]. Serogroup B meningococci are the major cause of sporadic disease in industrialised countries, and may cause outbreaks with lower attack rates than with serogroup A; some invasive strains have persisted in localities at hyperendemic levels for over a decade. Serogroup C meningococci are usually associated with sporadic disease, and have been implicated in both small clusters and large outbreaks, with attack rates between those seen for serogroups A and B.

Endemic disease in industrialised countries is caused by genetically diverse strains, mainly of serogroups B and C[18]. By contrast, outbreaks are usually caused by genetically homogeneous strains of these serogroups, consistent with the expansion of a virulent subtype.

Figure. Notification rate of meningococcal disease in Australia, by year



Data up to 1990[19] were modified with supplementary data, and updated to 1996 from published annual reports of the National Notifiable Diseases Surveillance Scheme.

These strains usually cause sporadic disease, but when and why they cause an outbreak is not clear. Possible risk factors include preceding infections in the population, such as those caused by the influenza virus or *Mycoplasma pneumoniae* [18]. Poor socio-economic conditions also increase the risk, where conditions such as overcrowding facilitate efficient transmission of virulent strains. This was probably an important factor contributing to the large outbreaks among indigenous populations in Canada [18], United States of America[18], New Zealand[14,18] and Australia[7].

While the rising incidence over the last decade has been attributed to genetically heterogeneous strains in many industrialised countries, the emergence of one or two virulent strains also accounted for a large proportion of that increase[1,8,9,11,18]. Research into bacterial population genetics suggests that the temporal variation in incidence is usually associated with clonal replacement of strains, much like influenza epidemics are driven by antigenic variation.

Meningococcal disease in Australia

The notification rate of meningococcal disease has fluctuated this century (Figure). Notifiable diseases data underestimate the true incidence of meningococcal disease[6,20]. For example, the level of under-reporting of meningococcal meningitis and septicaemia in New South Wales was estimated to be 55% in 1989-1990 and 21% in 1991-1992[6]. As the true incidence of meningococcal disease in Australia is not known, the notification rates have been taken to reflect trends in the incidence of meningococcal disease since 1917.

The epidemics of meningococcal disease caused by serogroup A in Australia at the time of the two world wars were part of a pandemic. After another peak of activity in the early 1950's, there was a continuing decline in the notification rate. In 1987, after over a decade of very low level activity nationally (notification rate under 1 per 100,000), a large outbreak of serogroup A meningococcal disease was reported amongst Aboriginal central Australians[7]. Notifications of disease caused by both serogroups B and C started to rise throughout Australia. The overall rate has plateaued at 2.1-2.3 per 100,000 over the last 4 years. The rise in incidence in sporadic cases has been accompanied by an increased frequency of outbreaks of disease caused mainly by serogroup C meningococci [15,16,21-23]. Although we have limited published information on the phenotypic distribution of invasive strains in Australia, it does provide some insights into outbreaks in the global context.

Predictably, the outbreak strains in Australia, although not always fully

characterised, have been closely related to those in other countries. If a virulent subtype of *N. meningitidis* causes a particular pattern of disease overseas, we should be well prepared to respond if the strain subsequently appears in Australia.

Serogroup A meningococci

The number of outbreaks caused by serogroup A have been low in industrialised countries since the Second World War, and concentrated in the poorest socio-economic groups [7,14,18,19] . Large epidemics continue to affect developing countries, and in 1996, more than 150,000 cases and 16,000 deaths were reported, mainly in west Africa.

Pandemic waves of disease caused by serogroup A meningococci since the beginning of this century have been associated with genetically distinct bacterial clones[18]. Of these, subgroup I-1 caused the 1987-1991 outbreak among Aboriginal central Australians[7,18], and has been associated with many outbreaks overseas. These included an outbreak in the Native Americans in Canada, which spread to skid row residents of the Pacific North West of the United States of America[18], and one in New Zealand in 1985[14,18]. Disease caused by this strain has not recurred in central Australia since 1992.

Serogroup B meningococci

Two complexes of serogroup B meningococci have caused increased disease activity in industrialised countries over the last three decades. The ET-5 complex (mainly phenotype B:15:P1.16), whilst being associated with outbreaks overseas, does not appear to have become established in Australia yet.

A second ET complex known as lineage III (B:4:P1.4), has also been associated with disease overseas,and now accounts for the major proportion of serogroup B isolates in the United Kingdom [9]. The number of isolates began to rise in New Zealand in 1991, with a rise in the incidence of meningococcal disease from 1.5 per 100,000 in 1989-1990, to 14.5 per 100,000 in 1996[24]. Vaccine trials are now being planned for this strain *(personal communication, Michael Baker, New Zealand).* It is of concern that this strain was detected in New South Wales, Queensland and Victoria in 1996[25].

Serogroup C meningococci

Two clonal complexes of serogroup C meningococci have caused increased activity in many countries, including Australia. The ET-37 complex, characterised mainly by C:2a:P1.5,2 has a global distribution, and has been the most common of the serogroup C strains causing endemic disease and localised outbreaks in industrialised countries[18]. This phenotype caused two localised clusters of cases in north Queensland between 1993 and 1994[15]. It was also responsible for the cluster of cases that affected mainly young adults in Penrith, New South Wales in 1996[23].

A second phenotype, C:2b:P1.2, has been associated with the ET complex known as the A4 cluster. This phenotype caused localised outbreaks in New Zealand[1] and the United Kingdom[9,13] that were controlled by community-wide vaccination programs. It was also associated with the high incidence of disease and a cluster of cases in south western Sydney in 1991, and was genetically identical to the outbreak strain identified in 1990-1991 in an Aboriginal community [17,22]. This phenotype also caused clusters in two other north Queensland communities over the same time period[15].

Future Management

The National Health and Medical Research Council (NH&MRC) has developed guidelines[26] for detecting, treating and documenting cases of meningococcal disease, and for planning and implementing control measures for sporadic cases and clusters. It also provides information for parents of children who may be at risk of the disease, and informative letters to general practitioners, child-care centres, schools, and the media.

It is not possible to predict exactly where and when the next outbreak may occur, how extensive it may be, or which particular subtype may be responsible. We can however, plan to respond swiftly when it does occur. With a sensitive surveillance system, we can detect an outbreak at the earliest opportunity, and control the incidence of secondary cases and of localised outbreaks in child-care centres, schools and other institutions. Each new case requires a response from the local public health unit to minimise transmission of the invasive strain among close contacts. The case must be documented and notified so that related cases can be detected readily at the local and regional levels.

We need accurate data on the incidence of meningococcal disease in Australia, and the associated morbidity. An assessment of the burden of disease is necessary to determine the cost effectiveness of a possible routine childhood vaccination program. Conjugate vaccines against serogroups A and C, and multivalent vaccines against a range of serogroup B strains are expected to become available within the next few years. The United Kingdom[27] and New Zealand (*personal communication, Michael Baker, New Zealand*) have indicated their intention to incorporate appropriate vaccines into the routine childhood vaccination schedule.

Regional and national surveillance data should be linked to microbiological data, and be interpreted in the context of global trends. They should be used to forecast possible future outbreaks, so that strategies to control the disease can be established.

Australia was caught by surprise by the change in the epidemiology of meningococcal disease that began to emerge in 1987. Outbreaks are unpredictable, and we were not well prepared to control the outbreak in central Australia[7]. The rising incidence elsewhere in Australia in the late 1980's was first suspected after noting the rising frequency of admissions to the Royal Children's Hospital in Melbourne[4]. The first publication that confirmed the new national trend appeared in 1992[5], and in 1993 the first meeting of the NH&MRC Working Party to address Australia's response to this change was convened. Although the incidence appears to have plateaued over the last 5 years, outbreaks of the disease are still likely to recur. This prediction is based on the evolving epidemiology of the disease in other industrialised countries, and serves as a warning for us to be prepared.

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Communicable Diseases Surveillance

Mycoplasma pneumoniae

Atypical pneumonia due to *Mycoplasma pneumoniae* is an acute, febrile illness of the lower respiratory tract. Transmission of the organism is by the inhalation of droplets produced by coughing, or by direct contact with an infected person. The incubation period is between one and four weeks, and the infection may be asymptomatic, particularly in children under five years of age. Clinical manifestations vary from a mild afebrile pharyngitis to atypical pneumonia in up to 30% of cases. Onset is insidious with signs and symptoms including headache, malaise, cough, sore throat and occasionally pleuritic chest pain. Antimicrobial therapy is not required for an upper respiratory tract infection. Whilst pneumonia is usually self-limiting, appropriate treatment with erythromycin or tetracycline can shorten the the course of the illness, which may last from several days to a month or more. However *Mycoplasma pneumoniae* can be cultured from the sputum of infected individuals for weeks to months following effective treatment, and may therefore serve as a source of infection for others. The organism has a worldwide distribution, and whilst most cases appear to be sporadic, epidemics do occur, particularly in closed environments such as in the family setting and in institutions.

The number of reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme has continued to rise since early 1996 (Figure 1). For 1996 a total of 1,010 reports was received. More females were reported than males, with a male:female ratio of 1:1.3 (Figure 2). The predominance of females was particularly marked in the 25 - 44 years age group.

Figure 1. ***Mycoplasma pneumoniae*** **laboratory reports, 1993 to 1997, by month of specimen collection**

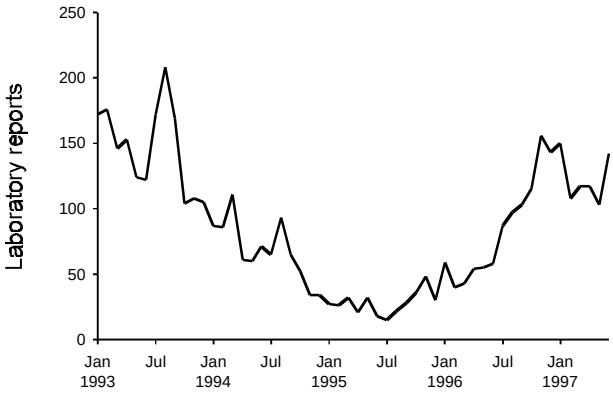
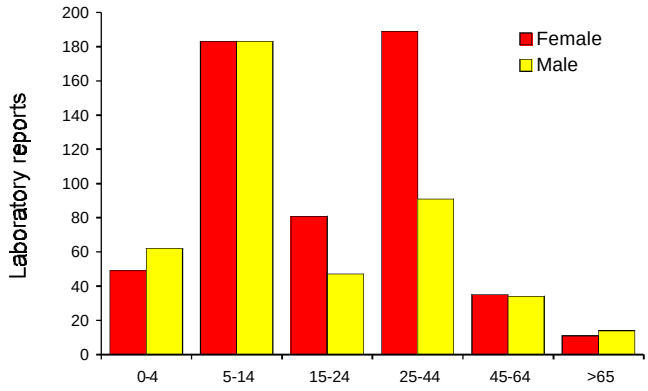


Figure 2. ***Mycoplasma pneumoniae*** **laboratory reports, 1996, by age group and sex**



National Notifiable Diseases Surveillance System

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 1997;21:5.

Correction: Tables 1 and 2 in the previous issue, *CDI* 1997:21:224 were incorrect, and represent data for the period 11 to 24 June 1997.

Reporting period 23 July to 5 August 1997

There were 1,931 notifications received for this two week period (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 4).

A total of 3,618 notifications of pertussis with onset in 1997 has been received so far. This is higher than any corresponding period since the establishment of the scheme

Figure 3. ***Haemophilus influenzae*** **type b infection notifications, 1991 to 1997, by month of onset**

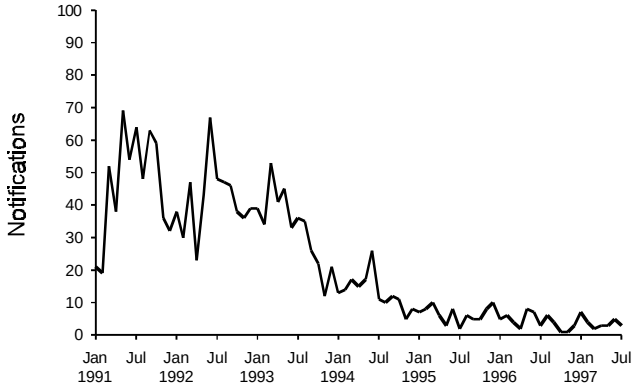


Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 23 July to 5 August 1997

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	0	0	0	0	0	1	1	2	1	32	37
Measles	3	4	0	3	0	1	5	13	29	14	328	272
Mumps	0	1	1	NN	0	0	0	3	5	8	115	68
Pertussis	3	46	1	90	38	2	29	22	231	94	4271	1808
Rubella	5	2	0	14	4	0	12	3	40	54	763	1565
Tetanus	0	1	0	0	0	0	0	0	1	0	7	1

NN. Not Notifiable

1. No notifications of poliomyelitis have been reported 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.

Table 2. Notifications of other diseases received by State and Territory health authorities in the period 23 July to 5 August 1997

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
Arbovirus Infection (NEC)[3]	0	0	1	0	0	0	0	0	1	1	108	40
Barmah Forest virus infection	0	4	-	4	0	0	0	-	8	18	494	668
Campylobacteriosis[4]	3	-	6	227	73	8	107	36	460	451	6818	7065
Chlamydial infection (NEC)[5]	12	NN	12	122	0	24	49	42	261	362	4840	5028
Dengue	0	0	0	0	0	-	0	1	1	2	193	26
Donovanosis	0	NN	6	0	NN	0	0	0	6	2	23	32
Gonococcal infection[6]	1	19	19	33	0	0	16	28	116	174	2713	2490
Hepatitis A	2	13	3	46	1	0	7	3	75	68	2022	1466
Hepatitis B incident	0	0	0	0	0	0	4	0	4	6	134	143
Hepatitis C incident	0	0	0	-	0	0	-	-	0	3	8	32
Hepatitis C unspecified	7	NN	10	128	NN	11	129	6	291	307	5607	5929
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	1	12	12
Legionellosis	0	0	0	0	1	0	0	0	1	7	100	117
Leptospirosis	0	1	0	1	0	0	0	0	2	16	78	155
Listeriosis	0	1	0	0	0	0	1	1	3	6	52	35
Malaria	3	0	0	0	2	1	4	1	11	48	493	529
Meningococcal infection	0	2	0	3	1	0	2	6	14	36	235	213
Ornithosis	0	NN	0	0	0	0	2	0	2	1	37	55
Q Fever	0	3	0	6	0	0	0	1	10	20	355	321
Ross River virus infection	0	6	2	25	1	1	2	4	41	40	6203	7352
Salmonellosis (NEC)	3	16	9	45	13	3	28	13	130	156	4685	3836
Shigellosis[4]	0	-	2	9	4	0	3	4	22	42	533	429
Syphilis	0	8	3	13	0	2	0	1	27	85	726	944
Tuberculosis	0	1	0	8	2	0	17	1	29	29	571	648
Typhoid[7]	0	1	0	0	0	0	2	0	3	3	46	59
Yersiniosis (NEC)[4]	1	-	0	10	1	0	0	0	12	4	173	156

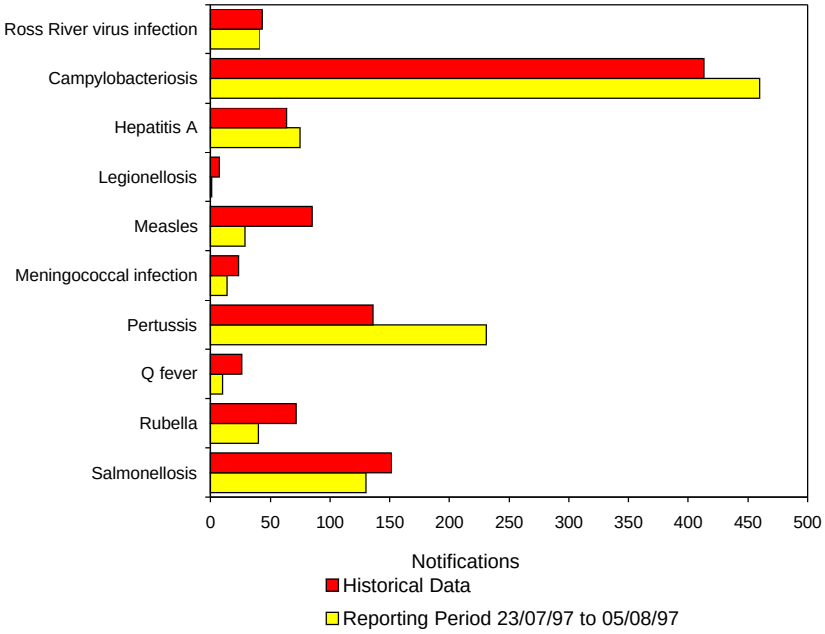
1. For HIV and AIDS, see *CDI* 1997;21:226-227. For rarely notified diseases, see Table 3.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. NT: includes Barmah Forest virus.
4. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
5. WA: genital only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. NSW, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified

\- Elsewhere Classified.

Figure 4. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods, the corresponding perioerds of the last 3 years and the periods immediately preceding and following those.

Figure 5. Syphilis notifications, 1991 to 1997, by month of onset

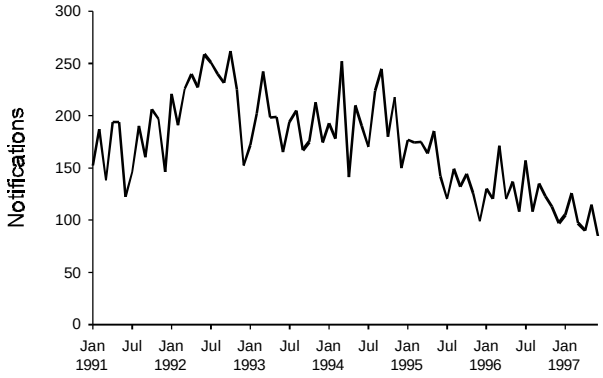


Figure 6. Syphilis notifications, 1997, by age group and sex

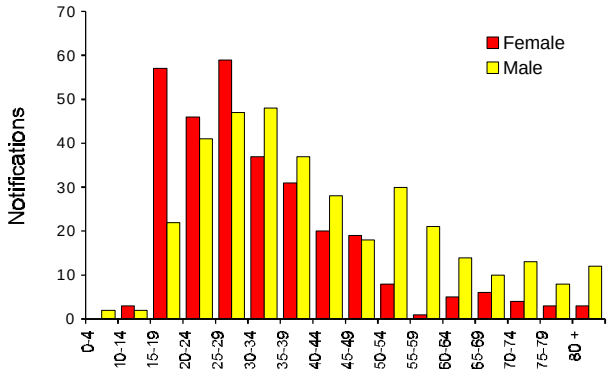


Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 23 July to 5 August 1997

Disease[2]	Total this period	Reporting States or Territories	Total notifications 1997
Brucellosis	2	Qld	20
Chancroid			1
Cholera			2
Hydatid infection	3	NSW, Vic	24
Leprosy			7

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1996.
2. No notifications have been received during 1997 for the following rare diseases: botulism, lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.

A total of 3,618 notifications of pertussis with onset in 1997 has been received so far. This is higher than any corresponding period since the establishment of the scheme in 1990. There were 231 notifications of petussis this period. Notifications are expected to increase over the spring and summer months, and the total notifications for 1997 is likely to be the highest recorded by this scheme.

There have been 32 notifications of *Haemophilus influenzae* type b infection (Hib) for the year to date. The number of notifications has declined following the introduction of conjugate Hib vaccines in 1992 (Figure 3).

A decrease in the number of notifications of syphilis has been observed since 1995 (Figure 5). For 1997 the male:female ratio was 1.2:1. However, there was a predominance of females in the 15 - 19 years age group (Figure 6).

National Influenza Surveillance, 1997

Three types of data are included in National Influenza Surveillance, 1997. 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 Department of Health and Community Services, 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 1997; 21:126.

Overall influenza activity continued to rise this fortnight, although the sentinel general practitioner consultation rate recorded in the Northern Territory declined. Sixty per cent of reports this period were for influenza B. Reports of influenza A have however, increased.

Laboratory Surveillance

A total of 323 reports of influenza virus was recorded by the LabVISE scheme this fortnight (Figure 7). Of these 130 were for influenza A, 177 for influenza B and 16 were untyped. The epidemic of influenza B this season is continuing. The number of reports of influenza B received in June and July are the highest recorded for these months by this scheme in the last five years. A greater number of reports are now being received for the 25 - 64 years age group, possibly as a result of greater awareness and testing generated from the recent high level of media attention. This age group accounted for 44% and 31% of influenza B and influenza A reports respectively.

Sentinel General Practitioner Surveillance

Reports of consultation rates for influenza-like illness from the New South Wales Scheme increased in the latter half of July, having decreased early in the month (Figure 8). The Department of Human Services Victoria, recorded a rate of 28 consultations per 1,000 encounters for the second two weeks of July, and the ASPREN scheme consultation rate also rose, reaching 30 per 1,000 in the last week of July. The Northern Territory data also indicate increased influenza activity for the last two weeks of July.

Absenteeism Surveillance

Australia Post recorded a national absenteeism rate of 3.1%. This has remained stable throughout the season.

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) currently comprises 107 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. Of these, CDI reports the consultation rates for chickenpox, gastroenteritis, HIV testing (doctor initiated), HIV testing (patient initiated), influenza, measles, pertussis, Ross River virus infection and rubella. For further information, including case definitions, see CDI 1997;21:6.

Data for weeks 30 and 31 ending 27 July and 3 August respectively are included in this issue of *CDI* (Table 4). The consultation rate for gastroenteritis has remained at a low level since the beginning of June. The consultation rate for chickenpox increased in week 31 to the level seen in May and June. The consultation rate for measles, pertussis and rubella has remained low for several months.

Figure 7. Laboratory reports of influenza, 1997, by type and week of specimen collection

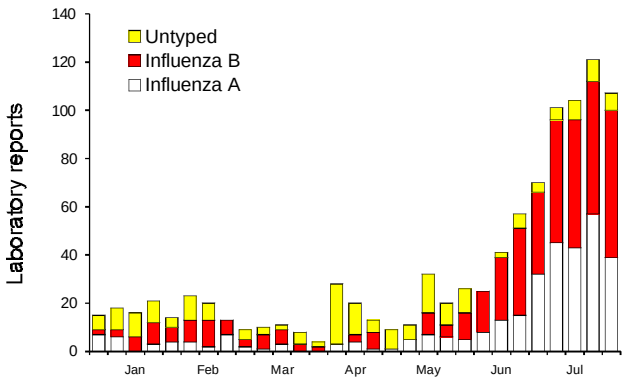


Figure 8. Sentinel general practitioner influenza consultation rates, 1997, by week and scheme

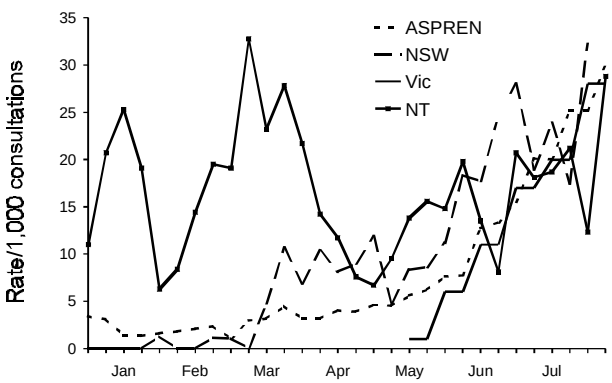


Table 4. Australian Sentinel Practice Research Network reports, weeks 30 and 31, 1997

Condition	*Week 30, to 27 July 1997*		*Week 31, to 3 August 1997*	
	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Chickenpox	5	0.8	17	2.3
Gastroenteritis	51	8.3	58	7.9
HIV testing (doctor initiated)	7	1.1	9	1.2
HIV testing (patient initiated)	9	1.5	11	1.5
Influenza	184	29.9	244	33.1
Measles	1	0.2	2	0.3
Pertussis	1	0.2	1	0.1
Ross River virus infection	1	0.2	6	0.8
Rubella	2	0.3	1	0.1

Table 5. Sentinel Chicken Surveillance Programme seroconversions, Western Australia, June and July 1997

	June				July			
	MVE	Kunjin	MVE & Kunjin	Flavivirus	MVE	Kunjin	MVE & Kunjin	Flavivirus
Kimberley								
Kalumburu	2		1		2			
Kununurra	1							
Fitzroy Crossing	1							
Derby	1		1					
Lombadina	1							
Broome			1		2	1	1	
Pilbara								
Karratha	1							

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 24 flocks are maintained in the north of Western Australia, ten in the Northern Territory, ten 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 1997;21:6

AK Broom[1], JS Mackenzie[2], L Melville[3], DW Smith[4] and PI Whelan[5]

1. Department of Microbiology, The University of Western Australia
2. Department of Microbiology, The University of Queensland
3. Berrimah Agricultural Research Centre, Darwin
4. PathCentre, Perth
5. Department of Health and Community Services, Darwin

Sentinel chicken serology was carried out for all of the 24 flocks in Western Australia in June and July 1997. There were 11 seroconversions to flaviviruses in the Kimberley and Pilbara regions in June, and 6 from the Kimberley region in July (Table 5). Five flocks of sentinel chickens from the Northern Territory were tested in June and July 1997, and during this period there were no seroconversions to flaviviruses.

LabVISE

The Virology and Serology Laboratory Reporting Scheme, 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 each fortnight. 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 1997;21:8-9.

There were 2,131 reports received in the *CDI* Virology and Serology Laboratory Reporting Scheme this period (Tables 6 and 7).

The number of Ross River virus reports has declined after peaking in March. There were 51 laboratory reports of Ross River virus this fortnight with 50% of reports received from Queensland and 43% from Western Australia.

Figure 9. Rhinovirus laboratory reports, 1991 to 1996 average and 1997, by month of specimen collection

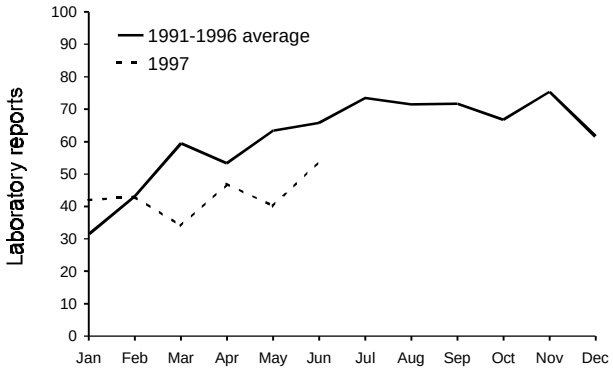
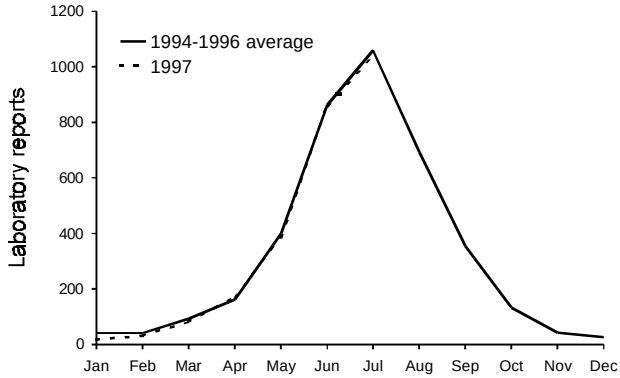


Figure 10. Respiratory syncytial virus laboratory reports, 1994 to 1996 average and 1997, by month of specimen collection



Forty-six reports of rhinovirus were received this period. Ninety-one per cent of reports were for the 1 - 4 years age group. The number of reports received this year is low compared to previous years (Figure 9).

Six hundred and ninety-seven reports of respiratory syncytial virus were received this period. Ninety-three per cent of reports were for children below the age of five years. The number of reports received this year is consistent with that of previous years (Figure 10).

One hundred and four reports of rotavirus were received this period for 54 males and 48 females (2 sex not stated). Eighty-six per cent of reports were for children under five years of age. The number of reports was lower than average for the month of June (Figure 11).

Figure 11. Rotavirus laboratory reports, 1992 to 1996 average and 1997, by month of specimen collection

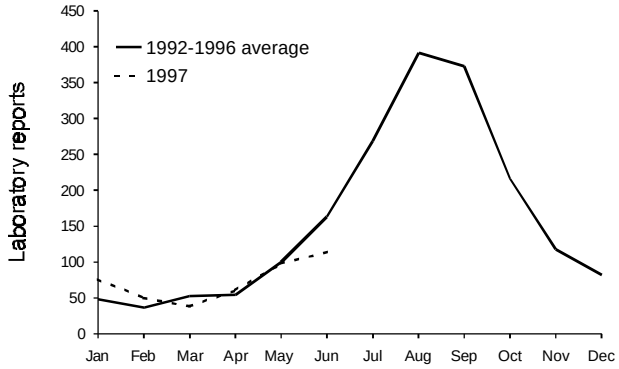


Table 6. Virology and serology laboratory reports by State or Territory[1] for the reporting period 17 to 30 July 1997, historical data[2], and total reports for the year

	State or Territory[1]								Total this fortnight	Historical data[2]	Total reported in *CDI* in 1997
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
Measles, mumps, rubella											
Measles virus				1				2	3	2.3	40
Mumps virus				1				4	5	1.5	28
Rubella virus	3			1			3	1	8	14.8	412
Hepatitis viruses											
Hepatitis A virus	7	1		15	2		2	15	42	11.5	541
Hepatitis D virus					1				1	0.7	15
Arboviruses											
Ross River virus			1	22	1		2	25	51	18.5	2,005
Barmah Forest virus				5				1	6	6.7	193
Dengue not typed				2				10	12	1.2	54
Kunjin virus								1	1	0	7
Adenoviruses											
Adenovirus type 1					1		1		2	1.3	19
Adenovirus type 41								2	2	0	3
Adenovirus not typed/pending	6	2		29	10		2	6	55	47.7	628

Table 6. Virology and serology laboratory reports by State or Territory[1] for the reporting period 17 to 30 July 1997, historical data[2], and total reports for the year, continued

	State or Territory[1]								Total this fortnight	Historical data[2]	Total reported in *CDI* in 1997
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
Herpes viruses											
Herpes virus type 6								1	1	0	4
Cytomegalovirus	5	8		26	7		10	18	74	62.8	788
Varicella-zoster virus	5	2	1	18	9		13	28	76	49.5	936
Epstein-Barr virus	7	12	1	14	30	1	5	24	94	81.3	1,748
Other DNA viruses											
Molluscum contagiosum								1	1	0.2	7
Contagious pustular dermatitis								2	2	0	2
Parvovirus					4	1	10	1	16	8.2	252
Picornavirus family											
Coxsackievirus A9	3								3	0	6
Coxsackievirus A16	2								2	0	10
Echovirus type 4					1				1	0	1
Echovirus type 5	1								1	0	6
Echovirus type 6					1				1	0	1
Echovirus type 9	1								1	0.7	2
Echovirus not typed/pending					1				1	0	3
Rhinovirus (all types)		9		16	2		1	18	46	30.3	414
Enterovirus not typed/pending				24				34	58	29.8	430
Ortho/paramyxoviruses											
Influenza A virus	1	31		25	9		40	19	125	181.8	477
Influenza A virus H3N2				1		1	3		5	7.3	7
Influenza B virus	2	14	1	48	3	3	44	62	177	22.2	530
Influenza virus - typing pending					16				16	0.3	230
Parainfluenza virus type 1			1						1	14.3	42
Parainfluenza virus type 2				7	5			1	13	7.5	93
Parainfluenza virus type 3	1	3	1	14	6		4	8	37	34.7	503
Parainfluenza virus typing pending					4				4	2.2	189
Respiratory syncytial virus	54	189		206	57	15	153	23	697	525.8	2,791
Paramyxovirus (unspecified)							1		1	0.8	13
Other RNA viruses											
HTLV-1			1						1	0.5	10
Rotavirus	6	5			24	2	35	32	104	127.3	712
Norwalk agent							2		2	1	67
Chlamydia trachomatis not typed	52	10		30	16	3	4	129	244	139.7	3,275
Chlamydia psittaci							1		1	3	47
Chlamydia species				1					1	0.7	22
Mycoplasma pneumoniae	4	10	2	23	7	2	8	12	68	27.3	1,140
Coxiella burnetii (Q fever)		5		8				5	18	8.2	232
Rickettsia australis				1					1	1.2	13
Rickettsia tsutsugamushi				3					3	1.3	20
Bordetella pertussis	2	1		10			9	19	41	11	1,134
Legionella pneumophila								1	1	0.7	16
Cryptococcus species	1								1	0.3	14
Leptospira pomona				1					1	0	12
Leptospira species				1					1	2.2	6
Toxoplasma gondi							1		1	2.5	1
TOTAL	163	302	9	553	217	28	354	505	2,131	1,492.80	20,151

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods, the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 7. Virology and serology laboratory reports by contributing laboratories for the reporting period 17 to 30 July 1997

State and Territory	Laboratory	Reports
Australian Capital Territory	The Canberra Hospital, Canberra	187
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	31
	New Children's Hospital, Westmead	226
	Royal Prince Alfred Hospital, Camperdown	18
Queensland	Queensland Medical Laboratory,	172
	West End State Health Laboratory, Brisbane	389
South Australia	Institute of Medical and Veterinary Science, Adelaide	214
Tasmania	Northern Tasmanian Pathology Service, Launceston	23
	Royal Hobart Hospital, Hobart	1
Victoria	Commonwealth Serum Laboratories, Melbourne	9
	Microbiological Diagnostic Unit, University of Melbourne	4
	Monash Medical Centre, Melbourne	51
	Royal Children's Hospital, Melbourne	173
	Victorian Infectious Diseases Reference Laboratory, Fairfield	121
Western Australia	PathCentre, Virology, Perth	512
TOTAL		2131

Overseas briefs

Source: World Health Organization (WHO)

Monkeypox, Democratic Republic of the Congo

The rise in the number of reported cases of monkeypox which began last year, has continued in 1997. From March to May 1997, 170 suspected cases were reported. There were no deaths. Most cases (79%) were in children under 16 years of age. In February 1997 a team of investigators was sent to study the cause of the outbreak. Due to the unstable political and social situation in the country, the team had to be evacuated after 10 days. WHO is planning to resume the investigations in September 1997.

Dengue, Malaysia

For the year to date(to 26 July), health authorities nationwide have received 11,328 notifications of cases of dengue. Of these 10,841 were dengue fever and 487 were dengue haemorrhagic fever. There were 28 deaths. The WHO Collaborating Centre in Kuala Lumpur has confirmed 99 cases of dengue haemorrhagic fever/dengue shock syndrome. For the same period last year, only 43 severe cases were diagnosed. Of the 57 dengue virus isolates investigated by the WHO Collaborating Centre this season, 37 were dengue type 1, 17 dengue type 2 and three dengue type 3. It is expected that the outbreak will peak in the next few weeks. The nation has been put on alert and aggressive integrated control programs have been instigated.

Plague, Mozambique

The Ministry of Health reported 115 cases of plague for the period 7 June to 4 July, in the Mutarara District, Tete Province; a plague endemic zone. No deaths have been reported. The last outbreak in this area occured in late 1994, when 216 cases were reported. Appropriate measures to control this outbreak are being taken.

Yellow fever, Liberia

A case of yellow fever in a 35 year old male in the northern part of Liberia was confirmed on 6 July. A second case is being investigated. Surveillance activity is being increased by Medical Emergency Relief International (MERLIN), a non-government organisation in the area. A mass vaccination campaign is being organised jointly by the Ministry of Health and several other agencies.

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