Annual report of the Australian Gonococcal Surveillance Programme, 1999

The Australian Gonococcal Surveillance Programme
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Abstract

The primary aim of the Australian Gonococcal Surveillance Programme (AGSP) is to monitor the antibiotic susceptibility of *Neisseria gonorrhoeae*. In 1999 the AGSP examined 3,740 isolates of gonococci from all States and Territories. The rates and sites of infection and antibiotic susceptibility patterns varied considerably between regions, reflecting the considerable differences between non-urban and urban gonorrhoea in Australia. Resistance to the penicillin and quinolone groups of antibiotics was highest in urban centres. Although penicillins remained suitable for use in many parts of non-urban Australia, enhanced surveillance is required as levels of resistance increase. Endemic transmission of quinolone-resistant gonococci (QRNG) in homosexually active men increased substantially in New South Wales and Victoria where more then 90% of all QRNG were found. QRNG in other centres continued to be isolated mostly from overseas travellers and at a low frequency. All isolates remained sensitive to spectinomycin and ceftriaxone. A further increase in the number of gonococcal isolates from homosexually active men was recorded in New South Wales and Victoria. 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 of cases was lower, and most isolates were from the genital tract in rates similar to those occurring in previous years. The impact of non-culture based detection methods will adversely affect the ability of the AGSP to monitor trends in gonococcal disease in future years. *Commun Dis Intell* 2000;24:113-117.

Keywords: gonorrhoea, surveillance, AGSP, antimicrobial resistance

ISSN 0725-3141
Volume 24
Number 5
May 2000

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Introduction

The Australian Gonococcal Surveillance Programme (AGSP) is a collaborative programme conducted by reference laboratories in each State and Territory and its primary aim is to monitor the antibiotic susceptibility of isolates of *Neisseria gonorrhoeae.* The gonococcus has a well-recognised capacity to develop resistance to anti-microbial agents used for treatment of gonorrhoea. This ability has seen the progressive emergence and spread of antibiotic resistant gonococci in Australia with a commensurate reduction in the efficacy of cheap and/or oral treatment options. Australia's location, close to many countries where there is a high proportion of resistant isolates,[1] poses particular problems, although the appearance and spread of antibiotic resistant gonococci differs considerably throughout the country.[2] The data from this programme are used to assist in the formulation of treatment regimens appropriate to proper management of gonorrhoea.

There is a close correlation between the likely outcome of treatment and the *in vitro* susceptibility of the gonococcus. However, treatment is increasingly syndromically based and given as a single dose. Even when an aetiological agent is identified, therapy usually must be provided before results of susceptibility tests on individual isolates can be performed. Standardised treatment regimens are therefore formulated using knowledge of the *in vitro* sensitivity of prevalent gonococci.[3] 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.[3]

Treatment of gonorrhoea is essential to prevent recognised and serious complications of the disease itself. It is now also accepted that gonorrhoea, when untreated, serves to significantly amplify the transmission of HIV. Further, it has been shown that this amplification effect of gonorrhoea on HIV transmission can be greatly reduced if gonorrhoea is appropriately managed. While control of gonorrhoea requires an integrated approach addressing behavioural, educational and treatment issues, there are compelling reasons to ensure that proper antibiotic treatment of gonococcal disease is in place as part of this approach.

Continuing and long-term surveillance is required to monitor and respond to changes in antibiotic resistance that may occur in a short space of time. The AGSP has provided quarterly reports to *Communicable Diseases Intelligence (CDI)*since antibiotic sensitivity data were first produced by the AGSP in 1981.[4] Monitoring of resistance to other antibiotics was added as newer therapeutic agents became available and as penicillin treatment was replaced with other agents. Currently, the emergence and spread of gonococci resistant to the quinolone antibiotics, agents widely used in Australia, is a particular concern. This is the fourth annual summary of AGSP data in *CDI* and provides information on antibiotic sensitivity data and trends in gonococcal disease.

Methods

The AGSP comprises participating laboratories in each State and Territory (see acknowledgments). It is a collaborative network of laboratories which seeks to obtain isolates for examination from as wide a section of the community as possible and both public and private sector laboratories refer isolates to regional testing centres. The sources of isolates remained relatively unchanged in 1999. However, the introduction of a rebatable item for nucleic acid amplification assays for the detection of gonococci in the pathology services table of Medicare will see a reduction in the number of cultures available for susceptibility testing as this technology is progressively introduced. Gonococci isolated in and referred to the participating laboratories were examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins and for high level resistance to the tetracyclines, by a standardised methodology.[5] The AGSP also conducted a programme-specific quality assurance (QA) programme.[6] Antibiotic sensitivity data were submitted quarterly to a co-ordinating laboratory which collated the results and also conducted the QA programme. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains to analyse certain trends in disease patterns. The geographic source of acquisition of resistant strains was ascertained whenever possible.

Results

Numbers of isolates

There were 3,740 gonococcal isolates referred to or else isolated in AGSP laboratories in 1999. The distribution and site of infection of these isolates are shown in Table 1. Of these, 3,658 (97.8%) remained viable for susceptibility testing in 1999. One-thousand five-hundred and twenty-three gonococci (42% of the Australian total) were isolated and remained viable for testing in New South Wales (NSW),

Table 1. Gonococcal isolates, 1999, Australia, by sex, site and region (excluding those from the ACT and Tasmania)

	Site	New South Wales	Victoria	Queensland	South Australia	Western Australia	Northern Territory	Australia
Male	Urethra	1,133	568	369	65	226	178	2,552
	Rectal	195	83	26	5	5	0	316
	Pharynx	80	55	7	0	0	0	143
	Other/NS	6	3	23	5	1	62	100
	Total	1,414	709	425	75	232	240	3,111
Female	Cervix	103	32	148	13	75	174	548
	Other/NS	11	3	16	5	6	29	80
	Total	114	35	164	18	81	213	628
Total		1,528	744	589	93	313	453	3,740

744 (20%) in Victoria, 552 (15%) in Queensland, 428 (12%) in the Northern Territory (NT), 302 (8%) in Western Australia (WA), and 93 (2.5%) in South Australia (SA) with small numbers in Tasmania and the Australian Capital Territory (ACT).

Compared with data from the same sources in recent years, there were further increases in the number of isolates in NSW (from 902 in 1997 and 1,386 in 1998), Victoria (from 362 in 1997 and 565 in 1998) and Queensland (from 516 in 1998). There was a decrease in the number of isolates available from the NT (from 555 in 1998 to 453) and WA (from 452 in 1998 to 313). The numbers of isolates in SA were only slightly different from the previous year and those from other centres were low. The increase in the number of isolates in NSW was particularly evident in the first 6 months of 1999, but in the second half of the year numbers were comparable to those obtained in 1998.

Source of isolates

There were 3,111 strains from men and 628 from women, with a male to female (M:F) ratio of 5:1. (The sex of one patient was not specified.) The number of strains from men increased from 2,233 in 1997 and 2,886 in 1998, and strains from women numbered 594 in 1997 and 697 in 1998. The M:F ratio was 3.7:1 in 1997 and 4.1:1 in 1998. The M:F ratio was highest in Victoria (20.2:1) and NSW (12.2:1) where more strains were obtained from urban populations than from non-urban populations, but lower in SA (4.2:1). The lower ratios in WA (2.8:1), Queensland (2.6:1) and the NT (1.1:1), reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in NSW and Victoria (19.4% of isolates in both States). This pattern is similar to that noted in 1997 and 1998. Approximately 5% of isolates are shown as being isolated from 'other' sites. These included 13 cases of disseminated gonococcal infection, 9 in men and 4 in women. Isolates from urine samples were regarded as genital tract isolates. Not all sites were designated. There were a small number of isolates from the eyes of both new-born and older infants.

Antibiotic susceptibility patterns

In 1999 the AGSP reference laboratories examined 3,658 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin, and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed greatly between the various States and Territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

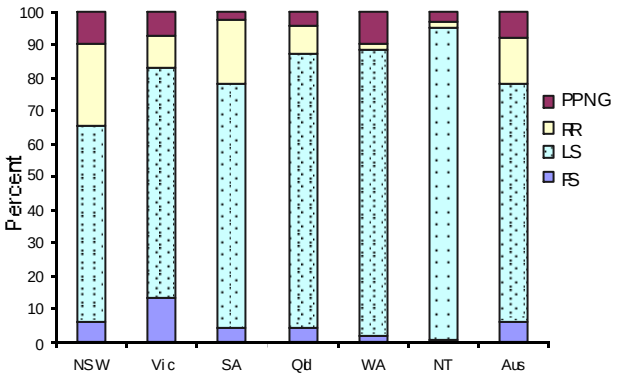
Resistance to the penicillin group (penicillin, ampicillin, amoxycillin) may be mediated by the production of beta-lactamase (penicillinase-producing *N. gonorrhoeae* – PPNG) or by chromosomally-controlled mechanisms (CMRNG).

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 categorisation of strains in Australia in 1999 by penicillin MIC is shown in Figure 1. 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 less sensitive or fully sensitive categories usually respond to therapy with standard treatment regimens with the penicillins. Infections with strains which are PPNG or in the relatively resistant category (CMRNG) usually fail to respond to the penicillins.

The 525 (14.3%) isolates resistant to penicillin by chromosomal mechanisms, CMRNG, in 1999 was lower than the 782 (21.8%) recorded in 1998. Strains of this type were concentrated in NSW (375 CMRNG, 24.6% of all isolates), Victoria (72 CMRNG, 9.7% of all isolates), SA (19 CMRNG, 19%) and Queensland (47 CMRNG, 8.5%). In contrast there were 6 (2%) CMRNG amongst WA isolates and 7 (1.6%) in NT strains.

PPNG increased in 1999 both numerically (to 269 from 206 in 1998), and as a proportion of all isolates (to 7.4% from 5.3% in 1998). Again the distribution of PPNG differed by region. NSW had the highest number, 148, and proportion, 9.7%, of PPNG. The 22 PPNG in WA represented 9.6% of strains in that State. PPNG were also prominent in Victoria (54, 7.2%). In Queensland about 4% of strains were PPNG and in the NT 2.8% were PPNG. One or two PPNG were found in Tasmania, the ACT and SA. Geographic acquisition details were available in about two thirds of cases of PPNG infection. Acquisition of PPNG through local contact was more common than through overseas contact in NSW but not in other centres. Indonesia, the Philippines, Thailand, Vietnam and China were the most frequently nominated countries of PPNG acquisition. PPNG acquisition was also reported from contact in Korea, Singapore, Hong Kong, Papua New Guinea, Timor, Japan, Brazil, Malaysia, Cambodia, the United States of America and the United Kingdom.

Figure 1. Penicillin resistance of gonococcal isolates, 1999, Australia, 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 *N. gonorrhoeae*.

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 so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered resistance as an MIC of 0.06 mg/L or more. Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with this less developed resistance in approximately 90% of cases, but lower doses of the antibiotic will more often result in treatment failure. The proportion of treatment failures increases exponentially as MICs rise. Treatment failure occurs in approximately 60% of infections with strains with MICs of 1 mg/L or more, even when higher doses are used. Currently gonococci with MICs up to 16 and 32 mg/L are being seen in Australia. Newly released quinolone agents would not be expected to offer any significant advantage over ciprofloxacin for the treatment of gonorrhoea.

In 1999 a total of 628 (17.2%) of gonococcal isolates displayed altered sensitivity to the quinolones (QRNG). This is more than three times the number of QRNG seen in 1998 (186, 5.2%) and is attributable to the high rate of QRNG in homosexually active men in NSW and Victoria. Rates of QRNG have been high in NSW since an increase in the number and proportion of QRNG in heterosexuals was noted in NSW in the December quarter of 1996. This rate of isolation was sustained throughout 1997 and the early part of 1998, but declined in the latter part of that year. In NSW in 1998, QRNG appeared in homosexually active males for the first time. In 1999 QRNG increased in NSW to 26% and in Victoria to 24% of all isolates examined. More than 90% of all QRNG identified in Australia in 1999 were found in these two States. QRNG were found in all centres except the ACT. Queensland had 43 (7.8%) QRNG, WA 9 (3%), with smaller numbers in SA, Tasmania and the NT. The spread of QRNG in NSW and Victoria was mainly by local as opposed to overseas contact, but in most other centres cases were imported from overseas contact from sources similar to those described for PPNG acquisition.

High level tetracycline resistance

Two-hundred and eighty-eight high level tetracycline resistant *N. gonorrhoeae* (TRNG, 7.9 % of isolates) were detected throughout Australia in 1999, a slight increase over the 1998 numbers. Most TRNG were found in NSW (168), representing 11% of all isolates. There were 33 (11%) TRNG in WA, 32 (6%) in Queensland, 47 (6%) in Victoria, and 6 in the NT. Infections with TRNG were mainly acquired overseas in Indonesia, Thailand and Singapore. However, an increasing number of isolates were acquired through local contact, especially in NSW.

Discussion

Two typical features of the antibiotic susceptibility patterns of gonococci isolated in Australia were again evident in 1999; major regional differences continued and there was considerable volatility in resistance especially to quinolone antibiotics. As a point of reference, the World Health Organization recommends that an antibiotic should no longer be used for treatment of gonorrhoea when 5% or more of isolates are resistant to its action.

A high proportion of the gonococci isolated in urban centres has been resistant to the penicillins for many years and this trend was maintained in 1999. Between one eighth and one third of isolates in NSW, Victoria, Queensland and SA were resistant to this group of antibiotics. Most of this resistance was chromosomally mediated (CMRNG) and in locally acquired strains. CMRNG increased in Queensland to 8.5% of all isolates. The decline in the rate of PPNG noted over the past few years was arrested, and local transmission of PPNG was evident in NSW and Victoria. Although the proportion of CMRNG in the NT and WA remains low, there has been a continuing shift upwards in MICs to the point where close surveillance needs to be maintained if penicillins are to remain the preferred treatment option.

Patterns of resistance to the quinolone antibiotics also showed further volatility in 1999, especially in NSW and Victoria. The high levels of endemic transmission of QRNG observed in these two centres indicate that use of this group of therapeutic agents should be discontinued there. The proportion and patterns of QRNG in other centres has altered little in recent years and the QRNG isolated were nearly all from imported infections.

The quinolone group of antibiotics, with the penicillins, are the only oral treatments for gonorrhoea available in Australia. The continuing presence of QRNG in numbers shown in these data remains a cause for concern, especially as Australia is located in a region where the prevalence of QRNG is high.

All gonococcal isolates were susceptible to the third generation cephalosporin ceftriaxone. Oral third generation cephalosporins are not available in Australia. Earlier generation cephalosporins are less active in gonococcal disease than ceftriaxone. They should be used with caution as overseas studies have indicated that where CMRNG are present in high numbers, (as is the case in Australia), these agents represent suboptimal therapy.

In 1998 the number of TRNG was about 50% more than the 1997 figure and this figure and proportion increased again in 1999. Sustained domestic transmission of TRNG was evident especially in NSW. The spread of TRNG is examined as an epidemiological marker and tetracyclines are not a recommended treatment for gonorrhoea.

The AGSP has until now been able to confirm other findings on rates of gonococcal disease in Australia with its sample of isolates obtained from relatively unchanging sources. Additionally, AGSP data record site of isolation which is not always available in other data sets. This has allowed the AGSP to comment on trends in gonococcal disease in Australia as a by-product of its prime role in antibiotic susceptibility surveillance. This situation has altered as the use of non-culture based methods (such as nucleic-acid-based amplification assays - NAA) has increased and the availability of cultures and accompanying clinical data has decreased. It is not possible to estimate the effect of NAA testing on the data set available to the AGSP. NAA testing became available as a Medicare rebatable item late in 1999, but was widely used across Northern Australia prior to this. Any decline in AGSP numbers to date has been, ironically, in those areas where enhanced susceptibility surveillance is required as penicillin resistance emerges. In other parts of

Australia the impact of NAA testing on isolate availability is yet to be felt.

There was a further increase in the number of isolates in NSW in 1999, although this was not maintained in the second half of the year. There has been a continuing and, until 1998, accelerating increase in the number of gonococcal isolates in NSW since 1994.[2,7] A significant increase in rates of isolation of gonococci was again noted in Victoria in 1999. Numbers had increased by about 50% in 1998 and a further substantial rise was noted in 1999. In both NSW and Victoria the increase in disease appeared to be in homosexually active males. In NSW the number of rectal and pharyngeal isolates in males increased from 124 in 1997 to 221 in 1998 to 275 in 1999. In Victoria the corresponding figures were 68 to 85 to 138. An increasing incidence of gonorrhoea in homosexually active men has been reported from London.[8] The M:F ratio of disease increased in Victoria from 14:1 in 1998 to almost 20:1 in 1999. In NSW this ratio increased from 9:1 in 1998 to 12:1 in 1999. The M:F ratio of disease altered less in WA and Queensland in 1999 and was lowest (1.1:1) in the NT.

In previous reports it was noted that 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 NSW and Victoria. The different pattern of gonococcal disease in Northern Australia is shown in the lower male to female ratio and the high rate of genital tract isolates in data from Queensland, WA and the NT. This pattern continued in 1999.

Gonococcal disease is again increasing in some developed countries and this has also been evident in parts of Australia for some time. The increased number of cases is becoming more difficult to treat as the choice of suitable therapy is becoming more restricted by antibiotic resistance. Continued monitoring of resistance patterns is required to optimise treatment regimens. Although non-culture based diagnostic techniques decreases the number of isolates for susceptibility surveillance in gonococci, the sample base currently available is sufficient for this purpose. However, assessment of trends in gonococcal disease by the AGSP will be compromised by these changes.

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.

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

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Report of the Australian National Polio Reference Laboratory

1 July to 31 December 1999

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Abstract

Since 1994, as part of the global eradication of poliomyelitis, the Australian National Polio Reference Laboratory (NPRL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL) has been responsible for virological testing to confirm the absence of poliomyelitis in Australia. Samples from patients with acute flaccid paralysis are transported to VIDRL for viral culture. Polio and enteroviruses are referred for intratypic differentiation as wild or Sabin (vaccine) strains. A total of 23 faecal specimens from 17 patients were processed for ente rovirus culture in the period 1 July to 31 December 1999. Since 1995, 1,078 enterovirus isolates from six states have been tested for the presence of wild poliovirus. To date, 562 strains were confirmed as Sabin vaccine-like, one non Sabin-like strain was identical with a laboratory control virus and the other strains were non-polio enteroviruses or other viruses. A World Health Organization (WHO) workshop in diagnostic polio polymerase chain reaction techniques was held at VIDRL in November 1999. The laboratory was reaccredited as a regional polio reference laboratory for the WHO Western Pacific region and a national laboratory for Australia, the Pacific Island countries and Brunei Darussalam. Planning is proceeding for the polio-free certification and containment of laboratory stocks of wild poliovirus infectious materials in Australia. *Commun Dis Intell* 2000;24:118-121.

Keywords: poliovirus, surveillance, acute flaccid paralysis, enterovirus

Introduction

This is the third report of the activities of the National Polio Reference Laboratory (NPRL). Earlier reports for the year 1998[1] and the first half of 1999[2] summarised information on acute flaccid paralysis surveillance, the terms of reference of the laboratory and their implementation. Ongoing activities of the NPRL include the culture of faecal samples from patients with acute flaccid paralysis referred from all Australian states and the characterisation of polioviruses and enteroviruses. The NPRL is also attempting to locate and test all the polioviruses and untyped enteroviruses reported to the Serology and Virology Surveillance Scheme (LabVISE) since 1997. A workshop for selected national polio laboratories in the region served by NPRL was held at the Victorian Infectious Diseases Reference Laboratory (VIDRL) in November 1999 to train staff from these laboratories in polymerase chain reaction (PCR) techniques for the identification and characterisation of polioviruses. NPRL was also successfully reaccredited at that time as a World Health Organization (WHO) regional polio reference laboratory, for a further year. The task of the containment of wild poliovirus infectious materials in Australia was contracted to VIDRL and an inventory of laboratories which may contain these materials, and a national plan to contain the materials are being developed. Further detail on each of these activities is presented in this report.

Methods

Collection and culture of samples from patients with acute flaccid paralysis

Adequate specimens are defined as 2 stool samples collected 24 to 48 hours apart within 14 days of onset of paralysis arriving at the laboratory with ice present. It is recommended that they are transported to the laboratory within three days of collection.

Neutralisation tests to detect poliovirus antibodies were performed on single serum samples from 3 patients with suspected paralysis.

Polymerase chain reaction workshop for selected national laboratories in the region

Prior to September 1999, all poliovirus isolates regardless of their source, were referred from national laboratories for characterisation in an accredited regional reference laboratory. In November 1999, a workshop was organised to provide training in diagnostic PCR techniques for the identification and intratypic differentiation of entero and polioviruses for selected national laboratories in the region. Three NPRL polio laboratory staff members and one each from the national laboratories in Singapore and Hong Kong, and the regional reference laboratory in China (Beijing) participated.

Two staff members from the WHO specialised polio reference laboratory at the Centers for Disease Control and Prevention (CDC), Atlanta, Georgia, USA, facilitated the workshop.

A proficiency test of PCR testing will be conducted by CDC in June 2000 to confirm that NPRL has the capability to correctly identify the panel viruses. If the laboratory is successful the diagnostic PCR may then be used routinely to test referred samples from the region and within Australia. At present, the laboratory is accredited to perform nucleic acid probe hybridisation and enzyme immunoassay for the intratypic differentiation of polioviruses.

Table 1. Cumulative summary of identification of enteroviruses and intratypic differentiation of polioviruses from Australian laboratories from 1995 to 30 June 1999

State	Year	Polio Sabin-like	Non-polio enterovirus	Non-enterovirus/ negative	Total
NSW	1994	4			4
	1995	74	5		79
	1996	24			24
	1997	10			10
	1998	19			20#
	1999				^
Qld	1995	41	5	8	54
	1996	99	4	9	112
	1997	41			41
	1998	8	15	2	25
	1999	2			2
SA	1997	3			3
	1998	3			3
	1999	1			1
Tas	1995	1			1
	1996	3			3
	1997	4			4
	1998	4			4
	1999	4			4
Vic	1995	9			9
	1996	17			17
	1997	5			5
	1998	7			7
	1999	16			16
WA	1995/6	133	384	5	522
	1997	30	76		106
	1998				0*
	1999		2		2
Total	1995-99	562	491	24	1,078

* PCR has replaced culture for enteroviruses, so isolates are no longer available.

\# Includes one non-Sabin poliovirus type 2.

^ A batch of polioviruses isolated in NSW and ACT from 1994 to 1999 were received at VIDRL in April 2000.

Laboratory accreditation

It is a requirement for each country's certification as 'polio-free' that all AFP samples and intratypic differentiation be performed in a WHO-accredited laboratory. An on-site inspection and review of work carried out in the previous year was conducted by a representative from WHO and verified that the laboratory had fulfilled all the criteria for accreditation as a national and regional polio reference laboratory.

Containment of wild poliovirus

The Western Pacific region of the World Health Organization has included containment of wild poliovirus infectious or potentially infectious materials as one of the criteria for each nation's certification as 'polio-free'. Currently, a regional pilot project is underway to facilitate the implementation of a draft plan for poliovirus containment. In order that Australia may meet this criterion the Australian National Certification Committee has appointed a National Coordinator of Poliovirus Containment who is located at VIDRL and approved the development of a national plan for containment of wild polioviruses. As part of this national plan a list of laboratories with stored wild poliovirus infectious materials will be prepared as a part of a national inventory to be submitted to the WHO regional office and included with the national certification documents.

Results

Characterisation of referred entero/polioviruses

One hundred and nineteen polio or untyped enteroviruses were referred to the laboratory between July and December 1999. Ninety-eight of these were viruses isolated in Western Australia between 1996 and 1999. The remaining isolates were from Victoria, Tasmania and Queensland. Forty-nine (41%) of the original 119 referred were recovered in L20B cells (cell selective for polioviruses) and were identified as Sabin vaccine-like polioviruses. Thirty-five (29%) were recovered in rhabdomyo sarcoma (RD) but not in L20B cells, so were non-polio enteroviruses. Thirty-five (29%) not recovered in RD or L20B cells were non-polio enteroviruses or were no longer viable.

The cumulative results of testing on entero and polioviruses submitted from all States and Territories are summarised in Table 1. Since 1995, 1,078 virus isolates have been transported to NPRL from laboratories in five Australian States. Five hundred and sixty two (52%) have been confirmed as Sabin vaccine-like polioviruses, 491 (46%) were non-polio enteroviruses and 24 yielded no virus or viruses other than enteroviruses. One poliovirus characterised in March 1999 as non Sabin-like was described in an earlier report.[2]

Acute Flaccid Paralysis

During the second half of 1999, 23 specimens were received from 17 patients with acute flaccid paralysis (AFP) (Table 2). Samples were received from 7 patients in Queensland, 4 in Victoria, 3 in Western Australia, 2 in New South Wales and one in Tasmania.

Onset dates were only available for 2 patients, both of whom had faeces collected within 14 days of onset of symptoms. Samples from 5 patients were dispatched to NPRL within three days of collection, while 4, 6 and 2 were sent after three to seven, eight to 14 and greater than 14 days respectively. No information was given on storage of the samples before transport. No enteroviruses were isolated from samples from any AFP patient in this reporting period.

One Sabin-like poliovirus type 1 was isolated from faecal samples from a 59 year-old woman, who had received Sabin (oral polio vaccine) prior to travel to Indonesia and developed fever and rigors, possibly vaccine-related, three days later.

Table 2. Specimens processed from Australian patients with AFP 1 July to 31 December 1999

State	District/city	Specimen date	Result
Qld	Brisbane	1-05-99	No virus isolated
Qld	Brisbane	23-06-99	No virus isolated
Qld	Brisbane	29-06-99	No virus isolated
Qld	Brisbane	3-07-1999	No virus isolated
NSW	Jerrabomberra	23-07-1999	No virus isolated
		24-07-1999	No virus isolated
Vic	Omeo	10-08-1999	No virus isolated
Vic	Dandenong	15-08-1999	No virus isolated
Vic	Dandenong	18-08-1999	No virus isolated
Vic	Dandenong	23-08-1999	No virus isolated
		23-08-1999	No virus isolated
Qld	Middlemount	3-09-1999	No virus isolated
Qld	Toombul	12-09-1999	No virus isolated
Qld	Samford	9-10-1999	No virus isolated
		13-10-1999	No virus isolated
WA	Girrawheen	14-10-1999	No virus isolated
		15-10-1999	No virus isolated
NSW	Wallsend	3-11-1999	No virus isolated
		4-11-1999	No virus isolated
		8-11-1999	No virus isolated
WA	Glen Forest	4-11-1999	No virus isolated
		TS 4/11/99	No virus isolated
WA	Kalgoorlie	15-11-1999	No virus isolated
		16-11-1999	No virus isolated
Tas	Gravelly Beach	24-12-1999	No virus isolated
		25-12-1999	No virus isolated

Two patients had elevated antibody levels to poliovirus types 1, 2 and 3, suggestive of past immunisation. The third patient had antibodies to poliovirus types 2 and 3 only, suggestive of a failed type 1 response to vaccination.

Discussion

Samples from AFP patients

Although still below the target of 78 samples from 39 children in Australia aged less than 15 years (one per 100,000), there was a further improvement in the numbers of samples referred to NPRL from patients with acute flaccid paralysis. During 1999, 41 faecal and 1 respiratory sample were processed from 27 patients. In 1997 and 1998, samples were referred from 4 and 11 patients with AFP respectively.[1]

Most of these patients with AFP were also reported to the AFP study group of the Australian Paediatric Surveillance Unit[3] (APSU) and reviewed by the Polio Expert Committee. The absence of wild polioviruses in these samples was used to classify these patients as non-poliomyelitis.[4] There were several patients from whom faecal samples were received but details of their histories have not yet been forwarded to APSU by the clinicians involved in their management. More information is being sought on these patients so they may be included in the final analysis of AFP cases in Australia in 1999.

Characterisation of polioviruses

As a requirement for Australia's certification as a polio-free country, all polioviruses isolated regardless of source must be characterised as Sabin (vaccine) or wild types. The *Communicable Diseases Intelligence (CDI)* publishes virology and serology laboratory reports received through the LabVISE programme. Over 60 uncharacterised polioviruses and nearly 800 untyped enteroviruses were reported in 1999. To date, only 23 polioviruses and two non-polio enteroviruses isolated in 1999 have been referred and tested at NPRL. The other strains received in 1999 were isolated from 1996 to 1998.

Western Pacific Regional certification

The last case of locally acquired poliomyelitis due to wild poliovirus in the Western Pacific occurred in Cambodia in March 1997. In November 1999, a strain of wild poliovirus type 1 was isolated in China. However, the case was epidemiologically and virologically linked to a virus possibly imported from the Indian sub-continent. There has been no evidence of re-established indigenous transmission in China.[5]

The Americas were certified in 1994, three years after their last case was detected in Peru. The Western Pacific Regional Certification Commission is meeting in July 2000 to examine evidence to prepare a case for certification. If the decision is favourable, the Western Pacific Region will be the second of the six WHO regions to be certified polio-free.

Containment of wild poliovirus

As a part of certification each country is required to provide evidence of three years of high immunisation rates, quality AFP surveillance, no wild polioviruses isolated from any source and a plan developed in the event that wild poliovirus is imported.[1] The last indigenous wild poliovirus in Australia was most likely in the mid-1960s.[2]

For certification in the Western Pacific Region, an additional criterion has recently been added. Since circulation of wild poliovirus has ceased, the only sources of wild polioviruses or wild poliovirus infectious materials are from importations from countries where endemic poliomyelitis still occurs or in laboratories. The region has developed an action plan, which includes a national search of all medical/biological laboratories which may have wild poliovirus infectious or potentially infectious materials, and the preparation of a national inventory system for laboratories which contain such materials.[6] Once containment issues have been addressed in the Western Pacific region, a detailed plan will be available which may be adapted in other regions. A more comprehensive report on laboratory containment of wild polioviruses is being prepared.

Acknowledgements

We would like to thank the staff in Australian hospitals and reference laboratories for their continued cooperation in this effort to certify Australia as wild poliovirus free.

The Australian National Polio Reference Laboratory is funded by the National Centre for Disease Control, Department of Health and Aged Care and by the Victorian Department of Human Services.

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Outbreak of echovirus 30 meningitis in Wingecarribee Shire, New South Wales

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Abstract

An outbreak of aseptic meningitis due to echovirus 30 occurred in the Wingecarribee Shire, NSW, during October to November 1994, with 30 cases fitting the clinical case definition. Cases were ascertained from attendees of the local hospital. Medical files were reviewed and a standard questionnaire administered. Viral cultures were performed on CSF, throat swabs and stool specimens. The clinical presentation and laboratory findings were typical of viral meningitis. Cases were aged 8 months to 51 years; 26 were admitted to hospital. Headache was present in 93%, photophobia in 86%, vomiting in 69%, fever in 72%, and neck stiffness in 62%. In spite of temporal clustering, the mode(s) of transmission in this outbreak remain speculative. Although the route of transmission was not established, general hygiene measures to stop transmission were implemented when a common water source was excluded on epidemiological grounds. *Commun Dis Intell* 2000;24:121-124.

Keywords: echovirus, meningitis, disease outbreak

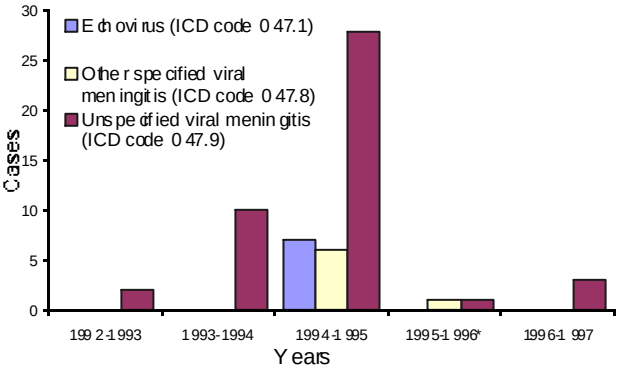
Introduction

Echoviruses (enteric cytopathogenic human orphan viruses) are a type of enterovirus, small RNA viruses responsible for many diseases including aseptic meningitis.[1,2] Transmission of enteroviruses is generally regarded as faecal-oral, and humans are the only known reservoir of human enteroviruses.[2,3] Close contact appears to be the primary avenue of spread, through faecal contamination of fingers, table utensils and food.[3] Young children are the usual reservoir, and abundant circulation of enteroviruses amongst them has been demonstrated.[2] It is possible that respiratory spread occurs but this seems less likely given that faecal shedding lasts longer than oropharyngeal shedding.[2] Transmission via insect vectors such as flies may also occur.[3]

Transmission has occurred in families, nurseries and institutions.[3] Waterborne outbreaks of enterovirus infection have occurred and the source is often difficult to establish, although contamination with sewage is considered likely.[2,3] Enteroviruses are hardy in fresh and salt water, particularly in the presence of organic matter, survive sewage treatment and chlorination, and can travel many miles downstream from the source.[2,3]

There are several reports of large outbreaks of aseptic meningitis due to echovirus 30,[4] including one involving

Figure 1. Separations from Bowral Hospital due to viral meningitis, 1992-1997



* PCR for enterovirus introduced

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47 cases in Western Australia.[5] An average of 4 cases of viral meningitis are seen per year at Bowral Hospital (Figure 1). Nine cases presented between 2 November and 10 November 1994, prompting notification of an outbreak of aseptic meningitis to the South Western Sydney Public Health Unit (SWSPHU). This report describes the clinical and public health investigation of this outbreak in the Southern Tablelands of New South Wales. Echovirus 30 was subsequently shown to have caused the outbreak.

Materials and Methods

Case ascertainment

The medical superintendent of Bowral Hospital notified SWSPHU of persons who presented to the hospital's emergency department and were diagnosed as having viral/aseptic meningitis during the period 4 October to 28 November 1994, the dates of the first and last notifications.

Case classification

The medical records of the above patients were reviewed by staff of the SWSPHU . The diagnosis of aseptic meningitis was accepted where the person had a headache associated with fever, photophobia and/or neck stiffness, the cerebrospinal fluid (CSF) microscopy and culture were negative for bacteria (when performed) and where no other cause for the headache was evident.

The Centers for Disease Control, Atlanta, case definition for aseptic meningitis is a syndrome characterised by the acute onset of meningeal symptoms, fever, and cerebrospinal fluid pleocytosis, in the presence of bacteriologically sterile cultures.[6] The SWSPHU modified this to the following:

Definite case: Isolation of echovirus 30 from the CSF by culture;

Probable case: Clinical diagnosis of viral meningitis and isolation of echovirus 30 from throat swabs or stool culture;

Possible case: Clinical diagnosis of viral meningitis without virological confirmation.

Polymerase chain reaction (PCR) only became available after the outbreak had occurred and was performed retrospectively. Unfortunately, this meant that it was performed on available stored CSF supernatants, compromising its sensitivity. Accordingly, PCR was not included in the above definitions.

Epidemiological investigation

The cases were interviewed by telephone using a structured questionnaire. Clinical histories were confirmed, and epidemiological links were sought. Patients were asked about their recent travels within Wingecarribee Shire, contacts with others with symptoms of viral illness, especially with prominent headaches, attendance at institutions (schools or child care centres), the source of their drinking water, and if and where they had been swimming.

A chart review was performed and the following data were obtained: name, medical record number, age, sex, underlying illness, date of onset of symptoms (fever, headache, photophobia, neck stiffness, drowsiness, focal neurological signs, gastrointestinal, cardiovascular or respiratory symptoms), signs (temperature, photophobia, neck stiffness, Kernig's sign, focal neurological signs) and laboratory data (full blood count, differential white cell count, biochemistry including liver function tests).

Microbiological investigations

CSF microscopy, bacterial culture and biochemical analysis were performed by the microbiology laboratory at Bowral District Hospital. Protein and glucose were determined. Cell counts and Gram stains were performed. CSF was inoculated onto horse blood and chocolate agar and examined daily for one week.

CSF was initially sent to Westmead Hospital for viral culture, but was subsequently processed by the Virology Laboratory at the South Western Area Pathology Service. CSF was inoculated into viral culture tubes containing primary monkey kidney (Edward Keller, Australia) or MRC-5 (Edward Keller, Australia) cells. The tubes were examined periodically for cytopathic effect (CPE) by inverted microscopy.

Throat gargles and faecal specimens were obtained and sent to the Virology Laboratory at South Western Area Pathology Service. Throat gargles were incubated in a viral transport medium containing penicillin, gentamicin and amphotericin B for 30 minutes prior to inoculation. Stool specimens were prepared by washing in phosphate buffered saline and centrifuging at 2,800g for 10 minutes. The supernatants were incubated in a solution of penicillin, gentamicin and amphotericin B for 30 minutes prior to incubation. Prepared throat gargles and stools were inoculated into primary monkey kidney and MRC-5 cell lines.

Cultures were observed for a total of three weeks. Those cultures which exhibited CPE suggestive of enterovirus were referred to the reference laboratory (Institute of Clinical Pathology and Medical Research, Westmead, Sydney, Australia) for typing by neutralisation using pooled antisera.

The Amplicor Polymerase Chain Reaction Kit (Roche Molecular Systems, Basel, Switzerland) was used to retrospectively perform PCR on 11 CSF specimens which had been stored at -80^{o}C. Kit insert instructions were followed, as published previously.[7]

Blood cultures were performed with the BACTEC NR 960 system.

Results

Clinical details

A total of 30 cases were detected; 7 definite, 7 probable and 16 possible. Of the 7 definite cases, 6 were the result of infection with echovirus 30 and one with echovirus 3. Twenty-six patients were admitted to hospital for an average of 2.8 days (range 1-9 days). Their ages ranged from 8 months to 51 years (median 25 years). The male:female ratio was 2:3. Headache occurred in 26/28 cases (93%), photophobia in 24/28 (86%), vomiting in 20/29 (69%), temperature >37.4^{o} C in 21/29 (72%), neck stiffness in 18/29 (62%), upper respiratory symptoms on presentation in 7/27 (26%), and gastrointestinal symptoms on presentation in 5/28 (18%). No patient had a rash.

Laboratory findings

The mean peripheral white cell count was $11x10^9$/L (range $4.4\text{-}20.2x10^9$/L, reference range $4\text{-}11x10^9$/L). The mean CSF parameters were: white cell count $239x10^6$/L (range $0\text{-}1160x10^6$/L, reference range $<2x10^6$/L), neutrophils $202x10^6$/L (range $0\text{-}1136x10^6$/L, reference range $<1x10^6$/L) and protein 0.32g/L (range 0.21-0.47g/L, reference range 0.15-0.45g/L). Virology results are presented in Table 1.

Epidemiology

The results of the epidemiological investigation are given in Table 2. Wingecarribee Shire experienced a crude attack rate (AR) of 0.8 per 1,000 with the highest AR recorded in the Mittagong postal area (1.5 per 1,000). The Mittagong postal area reported 21 of the 29 cases of acute viral meningitis attributed to the outbreak. (One case did not have the postcode recorded). Drinking water was not sampled for viral studies as the multiple, separate sources ruled out drinking water as a source.

Discussion

An outbreak of aseptic meningitis due to echovirus 30 occurred in the Wingecarribee Shire during October to November 1994, with 30 cases fitting the clinical case definition. Several outbreaks of meningitis due to echovirus 30 have been reported, including one in Australia.[5]

The clinical presentations were fairly typical of viral meningitis.[1] Most patients had fever, headache and meningism. The onset was often short (a few hours), and patients were often debilitated, requiring admission to hospital. Other systemic features of myalgia and arthralgia were also typical.[1]

The laboratory findings were also consistent with viral meningitis. CSF neutrophil pleocytosis is well recognised in viral meningitis, especially in the first 1-2 days,[1] and has been documented with echovirus 30.[5,8-12] CSF proteins were within the reference ranges, but may be slightly elevated,[5,8,11-13] and CSF glucose may be slightly depressed as occurred in some cases.[12,13] Peripheral leukocytosis may occur.[8] Unfortunately, PCR for enterovirus became available two months after the outbreak finished, and was performed on CSF supernatant stored at –80ºC. Not surprisingly, as the viral RNA is mainly found in the cells and the supernatant is largely acellular, the PCR positivity rate was low.[14]

Table 1. Virology results for 18 patients from whom CSF and/or stool was submitted for viral culture

Case No.	CSF viral culture	CSF enterovirus PCR	Stool viral culture
9	No growth	Negative	No growth
18	No growth	Negative	Not tested
20	No growth	Negative	Not tested
24	No growth	Positive	Not tested
13	Echo 30	Positive	No growth
23	Echo 30	Positive	Echo 30
3	Echo 30	Positive	Not tested
4	Echo 30	Positive	Not tested
15	Echo 30	Negative	Not tested
22	Echo 30	Negative	Not tested
27	Echo 3	Negative	Not tested
7	Not tested	Not tested	No growth
10	Not tested	Not tested	No growth
11	Not tested	Not tested	No growth
14	Not tested	Not tested	No growth
12	Not tested	Not tested	Echo 30
16	Not tested	Not tested	Negative
19	Not tested	Not tested	Echo 30

The mode(s) of transmission in this outbreak remain speculative. There was no obvious geographical clustering. Cases were distributed throughout Wingecarribee Shire although the majority lived in the Mittagong postal area. However, this area is large and isolated, and has multiple sources of drinking water, including four reticulated town water supplies drawing water from different dams, and several independent rainwater tanks. Under the circumstances water samples were not submitted for virological testing. Only one patient swam in a public pool or other body of water during September to November 1994.

Table 2. Epidemiological investigations

Investigation	Comment
Time period	4 October to 21 November 1994
Geographical clustering	Wingecarribee Shire; 21 of 29 cases where the postcode was recorded, resided in Mittagong postal area.
Attack rates	Wingecarribee Shire = 0.8 per 1,000 population.
	Mittagong postal district = 1.5 per 1,000population.
	OR 1.94, 95% CI 1.07-3.51
	Expected 4 cases per year in the Wingecarribee Shire ie 0.01 per 1,000 in a similar 48 day period.
Source of drinking water	Four reticulated town water supplies drawing water from different dams.
Swimming	Only 1 case swam in a public pool during September to November 1994.
Day care centres	No clustering of cases was observed.
Person-to-person transmission	One family had 3 members fall ill (one laboratory confirmed) several days apart, suggesting transmission within the family rather than co-primary infection.
	A nurse caring for a case fell ill 5 days later.
	The other cases were not epidemiologically linked.

Enteroviruses are usually transmitted by the faecal-oral route, except for respiratory tract infections where transmission may be by respiratory secretions, and conjunctivitis where person to person spread can occur.[1,2,3] The occurrence of some person to person transmission was suggested by the 3 clinical cases in one family (one confirmed by culture) and a health care worker who became ill after nursing a case. However, the other cases were not epidemiologically linked to a confirmed case suggesting other means of transmission.

The echovirus 3 case is probably a sporadic case that occurred in the midst of the echovirus 30 outbreak. We cannot determine if the outbreak involved more than one type of echovirus.

Although the route of transmission was not established, general measures to stop transmission were implemented when a common water source was excluded on epidemiological grounds. These measures included information on personal, food and domestic hygiene publicised through preschools, schools and the local media. Education of patients and doctors was instituted early. Nonetheless, the collection of the appropriate specimens was suboptimal. Throat swabs and stool specimens (or rectal swabs) are easily obtained, and were the only positive specimens in some of our cases.

This is the second outbreak of enterovirus-related aseptic meningitis reported in Australia.[5] Although recognised early because most of the cases presented to one hospital, bacterial meningitis was difficult to exclude initially as many of the patients had a neutrophil pleocytosis both peripherally and in the CSF. In addition, the source of the outbreak remains unknown despite prompt and thorough investigation.

Acknowledgments

Ms Laura Baird and Dr Gregory Stewart, Public Health Unit, South Western Sydney Area Health Service, Mr George Blackwell, General Manager, Wingecarribee Health Services, Dr Simon Grant, Directory of Medical Services, Bowral and District Hospital, Mr Peter Tomlinson, Hospital Scientist, Pathology Department, Bowral and District Hospital, Department of Virology, Institute of Clinical Pathology and Medical Research, Centre for Infectious Diseases and Microbiology, Westmead Hospital.

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National Communicable Diseases Data Management Meeting

The Surveillance and Management Section of the National Centre for Disease Control, Commonwealth Department of Health and Aged Care, held a National Communicable Diseases Data Management Meeting in Sydney on 17 and 18 May 2000. The aim of the meeting was to improve the quality and transfer of data to the National Notifiable Diseases Surveillance System (NNDSS) and the process of incorporating the expanded data collections for tuberculosis (TB), hepatitis C and foodborne diseases.

Thirty-two participants attended the meeting comprising representatives from each State and Territory health department, the Commonwealth Department of Health and Aged Care, the Department of Defence, the Public Health Laboratory Network (PHLN), the National Centre in HIV Epidemiology and Clinical Research, and the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases.

During the meeting, State and Territory representatives presented details of the notifiable diseases surveillance systems for their jurisdiction, field definitions, and their similarity to those proposed in the revised NNDSS data specifications. Other participants presented details of their organisations and the utilisation of national communicable diseases data. Participants also discussed the possible expanded disease data collections and some national disease-specific surveillance needs.

The outcomes of the meeting include:

- a greater working knowledge of each notifiable diseases system, with a move towards consistent definitions to feed into the revised NNDSS;
- an opportunity to address IT and data management issues which are common between States and Territories;
- the proposed development of disease detail coding protocols by PHLN;
- proposed preparation of proceedings which will include a summary of each State and Territory notifiable diseases system to be available through the Department of Health and Aged Care's Internet site; and
- a report to be prepared on the issues involved in implementing the expanded TB data collection.

A follow-up teleconference will be held in June, to finalise the recommended revised NNDSS core fields and definitions for endorsement by the Communicable Diseases Network of Australia and New Zealand.

Communicable Diseases Surveillance

Presentation of NNDSS data for April 2000

In the March 2000 issue an additional summary table was introduced. Table 1 presents 'date of notification' data, which is a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health unit. Table 2 presents data by report date for information only. In Table 2 the report date is the date the public health unit received the report.

Table 1 now includes the following summary columns: total current month 2000 data; the totals for previous month 2000 and corresponding month 1999; a 5 year mean which is calculated using previous, corresponding and following month data for the previous 5 years (MMWR Weekly Feb 25, 2000:49(07);139-146); year to date figures; the mean for the year to date figures for the previous 5 years; and the ratio of the current month to the mean of the last 5 years.

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'.

Bloodborne diseases

There were 1,233 notifications of hepatitis C in April 2000. This was a decrease from March 2000 (2,015), April last year (1,782), and the mean of the last 5 years (1,333). A total of 7,194 notifications of hepatitis C have been received for the year to date 2000. This was an increase from the year to date mean of the last 5 years (5,224). Of the notifications for April 2000, 15 were reported as hepatitis C incident cases. Seventy-five per cent of incident case notifications were in the 20 to 39 years age range. The male to female ratio was 1:1.1.

Gastrointestinal diseases

There were 444 notifications of salmonellosis in April 2000. This was a decrease from March 2000 (711), April last year (741) and the mean of the last 5 years (715). Forty-four per cent of cases (194) were in the 0-5 years age group. The male to female ratio was 1.1:1.

There were 4 notifications of typhoid in April 2000 and the ages ranged from 9 to 36 years. Four States currently report SLTEC/VTEC. There were 3 cases reported in April 2000, all from South Australia. There was also one case of HUS from New South Wales (NSW) in a male aged 2 years.

Quarantinable diseases

There were no cases of cholera, plague, rabies, yellow fever or viral haemorrhagic fever in April 2000.

Sexually transmissible diseases

There were 984 notifications of chlamydial infection in April 2000, which was a decrease from March 2000 (1,412) and April last year (1,242) but greater than the mean of the last 5 years (851). A total of 4,864 notifications of chlamydial infection have been received for the year to date 2000, which was a 47% increase from the year to date mean of the last 5 years (3,300). Most cases of chlamydial infection were reported from Queensland (30%) and Western Australia (25%). Eighty-six per cent of the cases were aged 15 to 34 years. The male to female ratio was 1:1.5.

There were 385 notifications of gonococcal infection in April 2000, which was a decrease from March 2000 (536), April last year (517) and the mean of the last 5 years (423). Most cases were reported from the Northern Territory (24%) and Western Australia (24%), with Queensland reporting 21% and Victoria 18%. The ages of cases ranged from 14 to 69 years, with 85% of gonococcal notifications aged 15 to 39 years. The male to female ratio was 2.6:1.

A total of 98 syphilis notifications were received in April 2000, which was a decrease from March 2000 (169), April last year (169) and the mean of the last 5 years (149). The year to date 2000 figure (531) was also lower than the year to date mean of the last 5 years (577). Most of the notifications were reported from Queensland (66%) and New South Wales (25%). Fifty per cent of syphilis cases were aged 20 to 34 years. The male to female ratio was 1.2:1.

Vaccine preventable diseases

There was a continuing decrease in the total number of vaccine preventable disease notifications with a total of 169 notifications in April 2000. This was mainly the result of the continuing decrease in notifications of pertussis.

There were no notifications of diphtheria, tetanus or poliomyelitis.

Two cases of *Haemophilus influenzae* type b were reported from Queensland. Both cases were male with unknown immunisation status: one case was a child aged under 1 year and the other an adult aged 40 years.

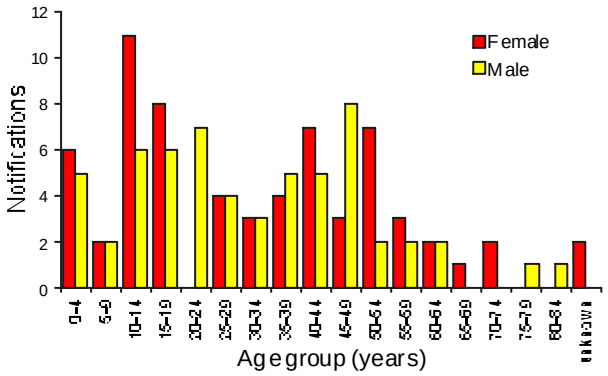
The number of notifications of measles, mumps and rubella were lower than for the same period in 1999 and for the mean of the last 5 years. However, an increase in the number of notifications of measles occurred in April 2000 (20) compared with March 2000 (11). Most measles notifications were from South Australia (30%), Victoria (30%) and NSW (13%). Measles notifications were most common in those aged under 5 years (10, 50%), with 3 cases aged under 1 year, 3 cases aged 1 year, 2 cases aged 3 years and 2 cases aged 4 years. The male to female ratio in this age group was 12.3:1. The immunisation status for this age group was reported as unknown for all but 3 cases: one child under 1 year was not immunised and 2 children aged 3 and 4 years were reported as partially immunised.

A similar pattern was seen for rubella notifications with 15 cases in April 2000 and 11 cases in March 2000. Most rubella notifications were from Queensland (47%) and Victoria (47%). Rubella notifications were most frequent in those aged under 5 years (5, 33%) and in those aged 15 to 39 years (5, 33%). Amongst those aged under 5 years, there were 4 cases aged under one year and 1 case aged 2 years. The male to female ratio in this age group was 1.5:1. For all of these rubella notifications the immunisation status was recorded as unknown, but cases under 1 year are age-ineligible for vaccination. Of concern females predominated (4, 80%) amongst those aged 15 to 39 years.

Pertussis cases for April 2000 (124) had decreased when compared with March 2000 (208) and the mean of the last 5 years (331). Pertussis notifications were most frequent in NSW (44%), Victoria (19%) and Queensland (17%). Cases of pertussis occurred in all age groups with peaks in the 0-4 (11), 10-19 (31) and 40-49 (23) years age ranges, with an overall male to female ratio of 0.9:1 (Figure 1). Immunisation status was reported for 11% of all pertussis notifications.

A total of 38 reports of meningococcal infection were received for April 2000, higher than the number of notifications for March 2000 (25), for April last year (33), and for the mean of the last 5 years (27). Most meningococcal cases were from NSW (39%), Victoria (29%) and Western Australia (18%). Meningococcal notifications were most frequent in those under 30 years of age with a predominance in the 0-4 and 15-24 years age ranges. The overall male to female ratio was 1.3:1. Serotype information was provided for 34% (13/38) of cases. Forty-six percent were serotype B and 54% were serotype C.

Figure 1. Notifications of pertussis, April 2000, by age group and sex



Vectorborne diseases

There were 14 notifications for dengue in April 2000, which was a decrease from March 2000 (33), but an increase from April last year (7) and the mean of the last 5 years (13). The majority of cases were from the Northern Territory (71%, all imported). A total of 167 notifications of dengue were received for the year to date 2000. This was an increase from the year to date mean of the last 5 years (85).

There were 422 notifications of Ross River virus infection in April 2000, which was a decrease from March 2000 (748), from April last year (804) and the mean of the last 5 years (854). The notifications decreased for all States and Territories in April 2000, except for the Northern Territory which reported 15 cases in this period compared with 7 cases in March 2000. The majority of notifications were still from Queensland (33%), Western Australia (21%) and NSW (20%). Forty-nine per cent of cases were aged 30 to 49 years. The male to female ratio was 1:1.

There were 59 notifications of malaria in April 2000, which was a decrease from March 2000 (93) and from the mean of the last 5 years (61), but an increase from April last year (50). The cases were due to *Plasmodium vivax* (33); *P. falciparum* (13); and 1*P. falciparum/P. vivax* co-infection. More than two thirds of the notifications were reported from Queensland (40) and all cases were imported. Seventy-five per cent of notifications were aged 20 to 44 years. The male to female ratio was 4.9:1.

There were 13 notifications of arbovirus infection (NEC) in April 2000, which was a decrease from March 2000 (15), but an increase from April last year (4) and the mean of the last 5 years (6). Most cases were reported from Victoria (46%), the Northern Territory (31%), Western Australia (15%) and Queensland (8%). Eight cases of Murray Valley Encephalitis were reported from Western Australia including 2 cases with onset dates in May 2000. There was one case in a male aged under 1 year with the remainder of the cases aged from 30 to 69 years. The male to female ratio was 3:1.

Other diseases

There were 124 notifications of legionellosis in April 2000, which was an increase from March 2000 (28), from April last year (22) and from the mean of the last 5 years (20).

The ages ranged from 25 to 89 years with a male to female ratio of 1.7:1 (Figure 2). Of these notifications 109 (88%) were due to *L. pneumophila* , 13 (10%) *L. longbeachae*, and 2 (2%) unknown/other (Figure 3).

A total of 207 notifications of legionellosis were received for the year to date 2000. This was an increase from the year to date mean of the last 5 years (78). The majority of the cases notified in April 2000 and for the year to date 2000 were associated with outbreaks in Victoria (87%) (Figures 4 and 5). This included the outbreak at Melbourne Aquarium, which was briefly discussed in *CDI* in April 2000.

Figure 2. Notifications of legionellosis, April 2000, by age group and sex

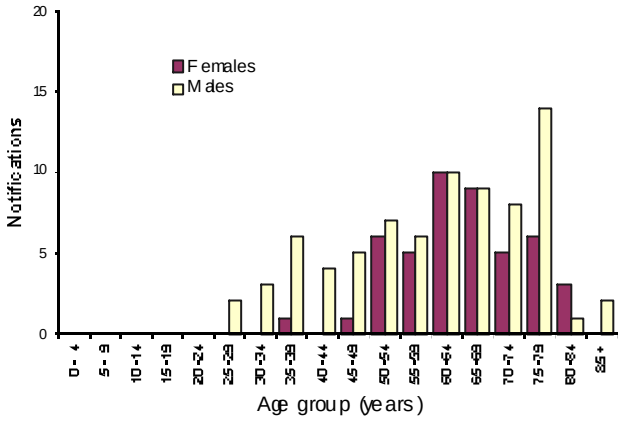


Figure 3. Notifications of legionellosis, January to April 2000, by serogroup

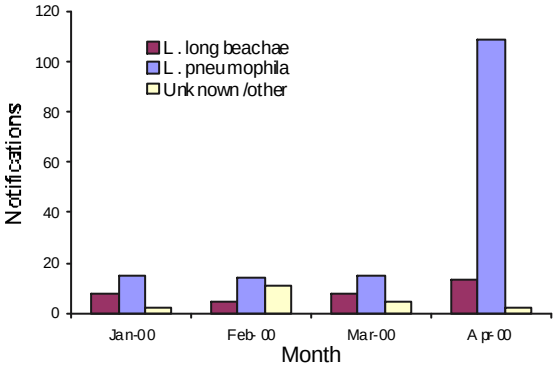


Figure 4. Notifications of legionellosis, 1991 to April 2000, Victoria and Australia, by month of notification

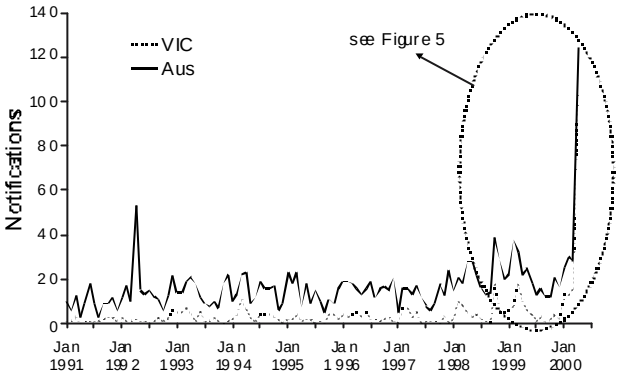
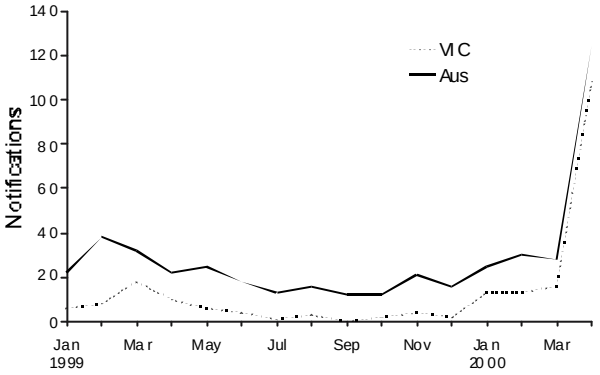


Figure 5. Notifications of legionellosis, January 1999 to April 2000, by month of notification

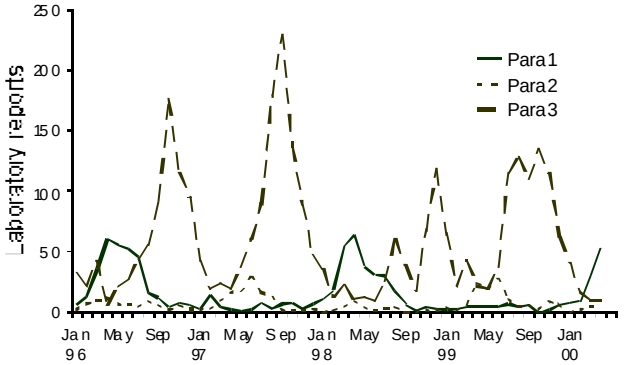


Parainfluenza viruses

The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a voluntary scheme that receives reports from sentinel laboratories around Australia. LabVISE reports showed an outbreak of respiratory illness due to parainfluenza type 1. The outbreak commenced in March 2000 and has continued throughout April. Reports of parainfluenza type 2 are low compared with the same period in 1999 and reports of parainfluenza type 3 are dropping after an outbreak that peaked in late winter and early spring 1999. Ninety-three per cent of parainfluenza type 1 reports were in children in the 0-4 years age group. More males than females were affected, with a male to female ratio of 1.25:1.

Historical data recorded by LabVISE show that outbreaks of parainfluenza virus type 2 and parainfluenza virus type 1 occur in the autumn months of alternate years. The last recorded outbreak of parainfluenza type 1 occurred in autumn 1998. By contrast Australia has recorded outbreaks of parainfluenza type 3 each year during winter and early spring. (Figure 6).

Figure 6. Parainfluenza virus laboratory reports, 1996-2000, by type and month of specimen collection



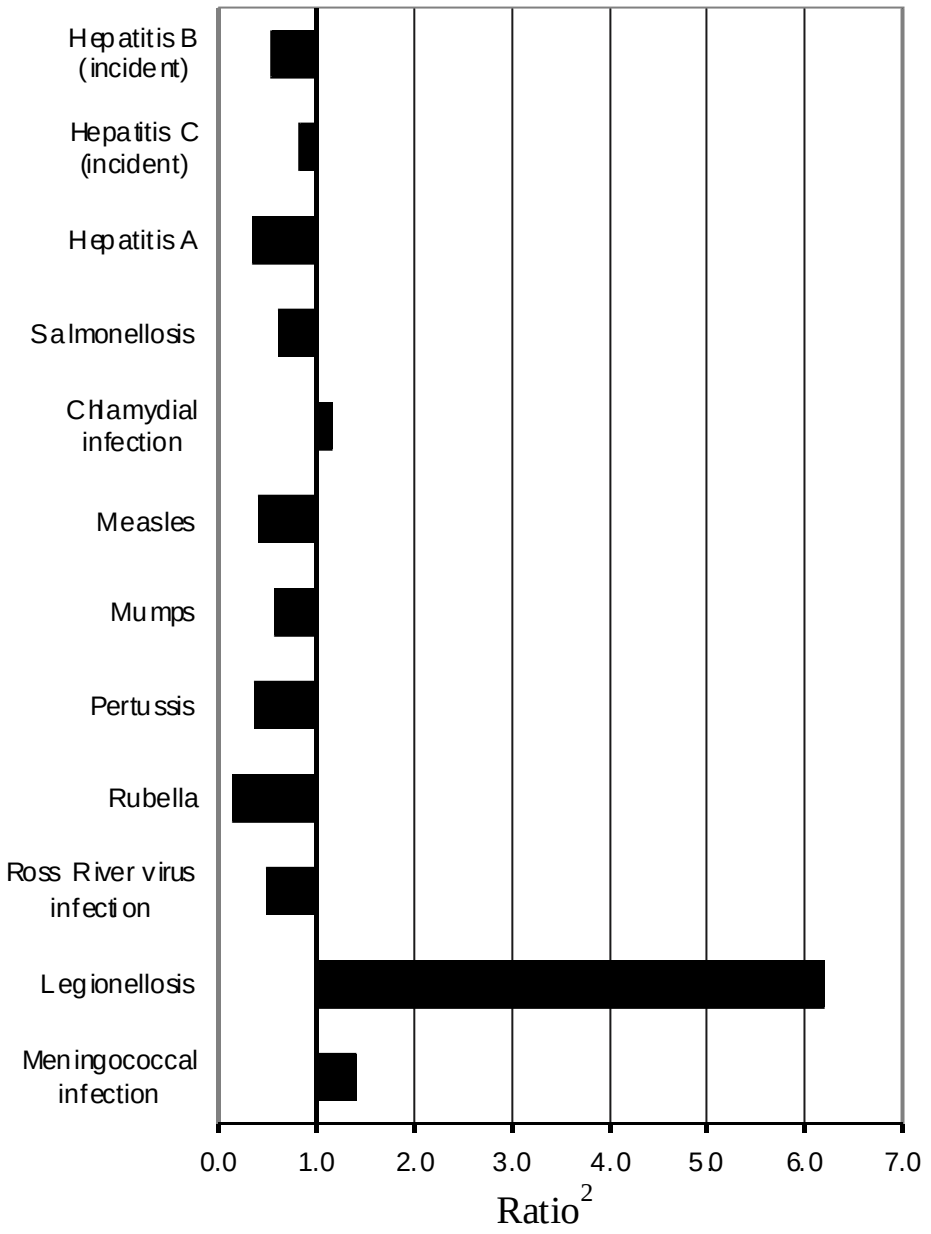
Tables

There were 5,500 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in April 2000 (Table 1). Data by date of report for weeks 13 to 17 ending 30 April 2000, are included in this issue of *CDI* (Table 2). The number of reports for selected diseases[1] have been compared with a 5 year mean, calculated using March to May data for the previous 5 years* (Figure 7).

There were 1,138 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 30 April 2000 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 13 to 17, ending 30 April 2000, are included in this issue of *CDI* (Table 5).

Figure 7. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 30 April 2000 with historical data[2]



1. Selected diseases are chosen each calendar month according to current activity
2. Ratio of current month total to mean of last 5 years as defined above*

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 30 April 2000, by date of notification#

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total April 2000[1]	Total March 2000[1]	Total April 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	1	6	1	1	1	0	0	3	13	27	24	24	91	99	0.5
Hepatitis B (unspecified)[2]	1	75	0	53	0	4	163	46	342	661	607	607	2,304	2,305	0.6
Hepatitis C (incident)	2	3	0	-	0	1	3	6	15	32	37	28	112	69	0.8
Hepatitis C (unspecified)[2]	10	372	6	230	42	16	440	102	1,218	1,983	1,745	1,375	7,082	5,161	0.9
Hepatitis D	0	1	0	1	0	0	1	0	3	3	2	2	6	6	1.5
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0.0
Campylobacterosis[3]	14	-	8	269	121	20	336	133	901	1,025	861	901	4,098	3,814	1.0
Haemolytic uraemic syndrome	NN	1	0	0	0	0	NN	0	1	3	1	2	6	3	0.5
Hepatitis A	0	11	3	8	8	0	15	20	65	82	112	186	376	910	0.3
Hepatitis E	0	0	0	0	0	0	0	0	0	0	0	2	0	2	0.0
Listeriosis	0	1	0	2	1	0	1	2	7	8	3	5	32	26	1.4
Salmonellosis	4	55	19	154	37	12	82	81	444	711	741	715	2,503	3,149	0.6
Shigellosis[3]	1	-	13	14	1	1	1	10	41	41	63	62	172	273	0.7
SLTEC,VTEC[4]	NN	0	0	NN	3	0	0	NN	3	4	3	2	16	5	1.5
Typhoid	0	3	0	0	0	0	0	1	4	7	3	3	26	36	0.7
Yersiniosis[3]	0	-	0	3	0	0	1	0	4	12	10	19	33	102	0.2
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	1	1	1	1	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rabies	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Yellow Fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Sexually transmissible															
Chancroid	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0.0
Chlamydial infection[5]	28	127	67	238	53	19	250	142	984	1,412	1,242	851	4,864	3,300	1.2
Donovanosis	0	0	0	1	NN	0	0	0	1	1	1	3	7	17	0.3
Gonococcal infection[6]	1	38	33	79	12	1	69	92	385	536	517	423	1,905	1,613	0.9
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0.0
Syphilis[7]	0	24	6	35	0	0	0	3	98	169	169	149	531	577	0.7

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 30 April 2000, by date of notification,[#] (continued)

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total April 2000[1]	Total March 2000[1]	Total April 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Vaccine preventable															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Haemophilus influenzae type b	0	0	0	2	0	0	0	0	2	1	5	4	5	17	0.5
Measles	0	4	0	1	6	1	6	2	20	11	25	40	53	218	0.4
Mumps	0	4	0	0	0	0	[1]	3	8	16	23	[1]4	57	53	0.6
Pertussis	5	54	0	21	8	12	24	0	124	208	205	331	1,019	1,698	0.4
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rubella[8]	0	1	0	7	0	0	7	0	15	11	26	102	61	495	0.1
Tetanus	0	0	0	0	0	0	0	0	0	1	0	1	3	2	0.0
Vectorborne															
Arbovirus infection NEC	0	0	4	1	0	0	6	2	13	15	4	3	40	34	2.2
Barmah Forest virus infection	0	9	2	28	0	0	2	2	43	66	109	101	222	366	0.4
Dengue	0	1	10	2	0	0	0	1	14	33	7	[1]3	167	85	1.1
Malaria	2	2	2	40	4	1	3	5	59	93	50	61	316	307	1.0
Ross River virus infection	1	85	15	[1]39	38	2	52	90	422	748	804	854	2,378	3,512	0.5
Zoonoses															
Brucellosis	0	0	0	0	0	0	0	0	0	3	0	3	5	10	0.0
Hydatid infection	0	NN	0	0	1	0	[1]	0	2	5	2	3	14	10	0.7
Leptospirosis	0	1	0	10	0	0	0	0	11	37	56	22	78	77	0.5
Ornithosis	0	NN	0	NN	1	0	[1]	0	2	8	12	7	21	27	0.3
Q Fever	0	4	0	32	0	0	3	0	39	60	37	46	182	165	0.8
Other															
Legionellosis	0	6	0	2	3	2	108	3	124	28	22	20	207	76	6.2
Leprosy	0	0	0	0	0	0	0	0	0	0	0	1	0	3	0.0
Meningococcal infection	0	15	0	2	3	0	1[7]	7	38	25	33	27	130	94	1.4
Tuberculosis	0	5	1	1	0	0	22	3	32	76	57	82	264	331	0.4
Total	70	908	250	1466	343	92	1,612	759	5,500	7,186	7,868	7,043	29,389	29,045	

1. 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.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA, Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella
#. Date of notification = a composite of three components: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health unit.

NN Not Notifiable.
NEC Not Elsewhere Classified.
- Elsewhere Classified.
na Not applicable.
* Ratio = ratio of current month total to mean of last 5 years calculated as described above.

Table 2. Notifications of diseases received by State and Territory health authorities for weeks 13 to 17, by date of report,* April 2000

Week number	13	14	15	16	17	Year to
Week ending on	2 April 2000	9 April 2000	16 April 2000	23 April 2000	30 April2000	date total
Disease[1]						
Bloodborne						
Hepatitis B (incident)	3	4	7	6	1	103
Hepatitis B (unspecified)[2]	137	131	134	108	80	2,533
Hepatitis C (incident)	13	8	6	6	2	128
Hepatitis C (unspecified)[2]	524	419	517	313	222	7,484
Hepatitis D	0	1	1	0	1	5
Gastrointestinal						0
Botulism	0	0	0	0	0	0
Campylobacterosis[3]	227	231	257	187	214	4,166
Haemolytic uraemic syndrome	0	1	0	1	0	5
Hepatitis A	22	17	14	18	20	396
Hepatitis E	0	0	0	0	0	0
Listeriosis	3	4	1	1	2	31
Salmonellosis	169	144	149	103	88	2,587
Shigellosis[3]	16	4	5	11	13	164
SLTEC,VTEC[4]	1	0	1	0	0	17
Typhoid	3	0	1	1	1	30
Yersiniosis[3]	3	4	0	1	0	33
Quarantinable						0
Cholera	0	0	0	0	0	1
Plague	0	0	0	0	0	0
Rabies	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0
Yellow Fever	0	0	0	0	0	0
Sexually transmissible						0
Chancroid	0	0	0	0	0	0
Chlamydial infection[5]	338	267	350	270	218	5,027
Donovanosis	0	0	0	1	0	8
Gonococcal infection[6]	153	102	133	90	101	1,947
Lymphogranuloma venereum	0	0	0	0	0	0
Syphilis[7]	38	31	37	25	18	579
Vaccine preventable						0
Diphtheria	0	0	0	0	0	0
Haemophilus influenzae type b	1	0	0	1	0	5
Measles	2	4	9	3	4	52
Mumps	3	7	4	4	0	61
Pertussis	53	45	44	55	19	1,228
Poliomyelitis	0	0	0	0	0	0
Rubella[8]	4	4	5	1	5	63
Tetanus	1	0	0	0	0	4
Vectorborne						0
Arbovirus infection NEC	6	0	5	3	4	34
Barmah Forest virus infection	17	8	12	21	6	231
Dengue	7	1	9	4	8	173
Malaria	23	28	9	17	9	314
Ross River virus infection	172	164	157	145	93	2,444

Table 2. Notifications of diseases received by State and Territory health authorities for weeks 13 to 17, by date of report,* April 2000 (continued)

Week number	13	14	15	16	17	Year to
Week ending on	2 April 2000	9 April 2000	16 April 2000	23 April 2000	30 April2000	date total
Disease[1]						
Zoonoses						0
Brucellosis	0	0	1	1	0	6
Hydatid infection	2	0	1	1	0	14
Leptospirosis	9	6	7	9	4	86
Ornithosis	3	2	3	1	1	31
Q Fever	19	13	11	14	4	195
Other						0
Legionellosis	5	7	7	8	49	154
Leprosy	0	0	0	0	0	0
Meningococcal infection	7	6	14	3	14	137
Tuberculosis	25	20	15	13	17	342
Total	2,009	1,683	1,926	1446	1218	30,818

1. 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.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with *Shiga*-like toxin (verotoxin) producing *E. Coli* (SLTEC/VTEC)
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella

* Date of report is the date the public health unit received the report.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 3. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 to 30 April 2000 [1]

State or Territory	Laboratory	This period	Total this period[2]
Australian Capital Territory	The Canberra Hospital	0	0
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	69	200
	New Children's Hospital, Westmead	62	67
New South Wales	Repatriation General Hospital, Concord	0	0
	Royal Prince Alfred Hospital, Camperdown	22	11
	South West Area Pathology Service, Liverpool	0	0
Queensland	Queensland Medical Laboratory, West End	366	333
	Townsville General Hospital	0	0
South Australia	Institute of Medical and Veterinary Science, Adelaide	351	365
Tasmania	Northern Tasmanian Pathology Service, Launceston	7	12
	Royal Hobart Hospital, Hobart	0	0
Victoria	Monash Medical Centre, Melbourne	0	3
	Royal Children's Hospital, Melbourne	51	101
	Victorian Infectious Diseases Reference Laboratory, Fairfield	161	238
Western Australia	PathCentre Virology, Perth	0	0
	Princess Margaret Hospital, Perth	49	0
	Western Diagnostic Pathology	0	0
Total		1,138	1,330

1. The complete list of laboratories reporting for the 12 months, January to December 2000, will appear in every report from January 2000 regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.
2. Total reports include both reports for the current period and outstanding reports to date.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 30 April 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	0	3	0	0	0	3	32	15	117
Mumps virus	0	0	0	0	1	0	0	0	1	6	22	21
Rubella virus	0	0	0	3	0	0	0	0	3	9	16	29
Hepatitis viruses												
Hepatitis A virus	0	0	0	2	4	0	0	0	6	30	55	146
Hepatitis D virus	0	0	0	1	0	0	0	0	1	1	2	3
Arboviruses												
Ross River virus	0	4	5	43	62	1	1	0	116	201	642	793
Barmah Forest virus	0	0	1	17	0	0	0	0	18	31	79	75
Flavivirus (unspecified)	0	0	0	2	0	1	0	0	3		34	16
Adenoviruses												
Adenovirus type 3	0	0	0	0	1	0	0	0	1	4	10	12
Adenovirus type 37	0	0	0	0	0	0	2	0	2	1	3	8
Adenovirus not typed/pending	0	8	0	0	28	1	11	5	53	106	312	355
Herpes viruses												
Cytomegalovirus	1	11	0	13	25	2	16	1	69	95	383	411
Varicella-zoster virus	1	4	0	25	17	1	9	0	57	126	458	559
Epstein-Barr virus	0	5	0	63	71	0	4	0	143	125	679	798
Other DNA viruses												
Parvovirus	0	0	0	0	1	0	1	0	2	36	87	131
Picorna virus family												
Echovirus type 7	0	1	0	0	0	0	0	0	1		3	1
Echovirus type 11	0	2	0	0	0	0	0	0	2	12	6	48
Echovirus type 30	0	1	0	0	0	0	1	0	2		66	6
Poliovirus type 3 (uncharacterised)	0	1	0	0	0	0	0	0	1	1	2	2
Rhinovirus (all types)	0	17	0	0	1	0	1	0	19	23	104	107
Enterovirus not typed/pending	0	3	0	6	0	0	115	0	124	62	406	258
Ortho/paramyxoviruses												
Influenza A virus	2	1	0	1	28	0	1	0	33	44	213	160
Influenza B virus	0	0	0	0	6	0	0	0	6	15	31	43
Parainfluenza virus type 1	0	15	0	2	12	0	3	21	53	4	100	13
Parainfluenza virus type 2	0	0	0	0	3	0	1	0	4	24	10	36
Parainfluenza virus type 3	0	0	0	0	9	0	1	0	10	22	77	151
Respiratory syncytial virus	0	38	0	9	10	1	19	16	93	116	316	328
Other RNA viruses												
Rotavirus	0	7	0	0	8	0	1	0	16	58	139	214
Norwalk agent	0	0	0	0	0	0	1	0	1	5	2	17
Other												
Chlamydia trachomatis not typed	6	39	14	54	42	2	12	5	174	242	1,001	996
Chlamydia psittaci	0	0	0	0	0	1	1	0	2	15	26	28
Mycoplasma pneumoniae	0	0	1	14	11	0	1	0	27	84	175	368
Mycoplasma hominis	0	1	0	0	0	0	0	0	1		1	4
Rickettsia australis	0	0	0	0	0	0	1	0	1		1	1
Streptococcus group A	0	2	5	20	0	0	0	0	27	2	136	2
Brucella species	0	1	0	0	0	0	0	0	1		4	2
Bordetella pertussis	0	2	0	3	5	0	3	0	13	26	178	197
Legionella pneumophila	0	0	0	0	0	0	1	0	1		3	12

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 30 April 2000, and total reports for the year[2] (continued)

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Legionella longbeachae	0	0	0	0	1	0	0	0	1	1	17	14
Legionella species	0	0	0	0	0	0	1	0	1		1	
Cryptococcus species	0	0	0	0	1	0	0	0	1	2	2	6
Leptospira species	0	0	0	5	0	0	0	0	5		14	
Treponema pallidum	0	1	15	21	0	0	0	0	37	2	166	8
Toxoplasma gondii	0	1	0	0	0	0	0	0	1	1	4	4
Echinococcus granulosus	0	0	0	0	1	1	0	0	2		5	
Total	10	165	41	304	351	11	208	48	1,138	1,564	6,006	6,500

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. 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.

\- No data received this period.

Table 5. Australian Sentinel Practice Research Network reports, weeks 13 to 17, 2000

Week number	13		14		15	
Week ending on	2 April 2000		9 April 2000		16 April 2000	
Doctors reporting	72		73		77	
Total encounters	9,119		9,272		9,691	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	34	3.7	34	3.7	42	4.3
Chickenpox	5	0.5	14	1.5	9	0.9
Gastroenteritis	68	7.5	92	9.9	92	9.5
Gastroenteritis with stool culture	12	1.3	17	1.8	17	1.8
ADT immunisations	66	7.2	50	5.4	51	5.3

Table 5. Australian Sentinel Practice Research Network reports, weeks 13 to 17, 2000, (continued)

Week number	16		17	
Week ending on	23 April 2000		30 April 2000	
Doctors reporting	70		64	
Total encounters	7,617		5,834	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	31	4.1	33	5.7
Chickenpox	9	1.2	14	2.4
Gastroenteritis	76	10.0	56	9.6
Gastroenteritis with stool culture	13	1.7	12	2.1
ADT immunisations	51	6.7	19	3.3

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 2000;24:6.

LabVISE is a sentinel reporting scheme. Currently 17 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. 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 2000;24:10.

ASPREN currently comprises about 120 general practitioners from throughout the country. Between 7,000 and 8,000 consultations are reported each week, with special attention to 14 conditions chosen for sentinel surveillance in 2000. CDI reports the consultation rates for five of these. For further information, including case definitions, see CDI 2000;24:7-8.

Additional Reports

National Influenza Surveillance, 2000

Three types of data are included in National Influenza Surveillance, 2000. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network (ASPREN), the Department of Human Services (Victoria), the Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health 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 2000;24:9-10.

Sentinel general practitioner surveillance

Reports of influenza-like illness consultations for April 2000 and the earlier part of the year 2000 were available from the Northern Territory and ASPREN surveillance schemes. Victorian and New South Wales sentinel schemes resumed in May 2000.

The Northern Territory showed a characteristic early peak of 20.1 per 1,000 influenza-like illness consultations in late February. There were 20 influenza-like illness consultations in April 2000, a decrease from March 2000 (29) and April last year (110). Influenza-like illness consultation rates from January to April 2000, were 6.6 per 1,000 consultations (97 cases), less than for the same period last year (15.0 per 1,000) (Figure 8).

ASPREN recorded a peak of 5.5 per 1,000 consultations for influenza-like illness by the end of April 2000, greater than for the same period last year (3.5 per 1,000) and an increase from the beginning of the year (1.8 per 1,000) (Figure 8). The age distribution of influenza-like illness consultations reported by ASPREN for January to April 2000 is shown in Figure 9. Most influenza-like illness consultations occurred in the 15 to 44 years age range. (190, 57%). Forty-one (12%) cases were in children under 5 years of age and 14 (4%) in the 65+ years age range (Figure 9). The male to female ratio was 1:1 (172:163). The age distribution pattern for April 2000 was the same as for January to April 2000. However, there were more influenza-like illness consultations for females than males (male to female ratio 1:1.3).

Figure 8. Sentinel general practitioner influenza consultations rates, week 18 1999 to week 17 2000, by scheme

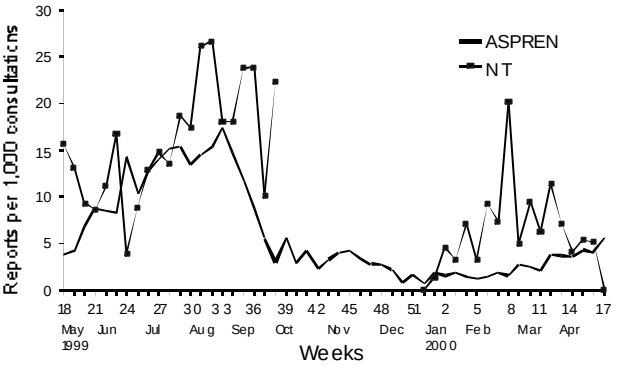


Figure 9. ASPREN influenza-like illness consultation, January to April 2000, by age group and sex

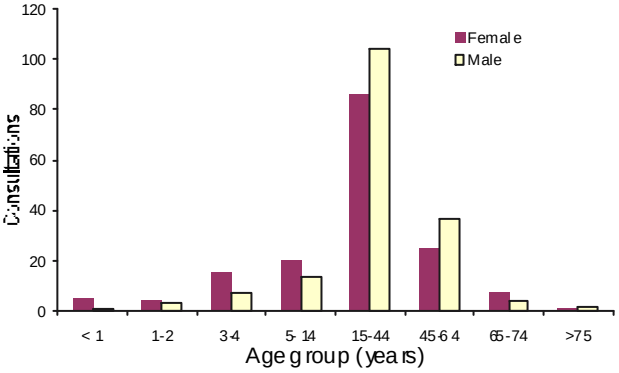


Figure 10. Laboratory reports of influenza, 2000, by type and week of specimen collection

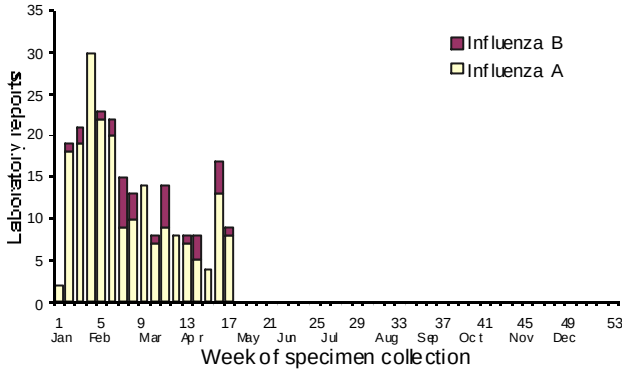
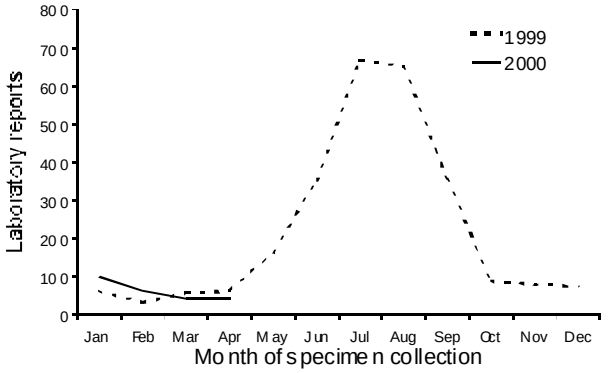


Figure 11. Laboratory reports of influenza, 1999 to 2000, by month of specimen collection



Laboratory Surveillance

LabVISE collects surveillance data all year. There were 244 laboratory reports of influenza virus isolation from January to April 2000. Of these, 213 (87%) were influenza A virus and 31 (13%) were influenza B virus (Figure 10). This was an increase from the same period last year (208 isolations), and comparable with the consultation rate for influenza-like illness for ASPREN. The number of influenza reports from LabVISE was higher between mid-January and the end of February 2000 than for the corresponding period in 1999. From the end of February 2000 the number of reports returned to a similar level to that seen in 1999 (Figure 11). Specifically for April 2000, there were 39 influenza reports, a decrease from March 2000 (42) and April last year (61). Of these, 33 were influenza A virus and 6 were influenza B virus. Age information was only available for 32% of the 244 reports with a peak in the 45 to 64 years age range (27; 34%). There were 12 (15%) reports in children under 5 years of age and 25 (32%) in the 65+ years age range, with a male to female ratio of 1:1.6 (Figure 12).

Absenteeism surveillance

Australia Post reports employees absent if they are not at work for 3 or more consecutive days in 1 week. The weekly rates for April peaked in the 2 week period coinciding with Easter (0.9%) and then declined to 0.5% later in April. Average weekly absenteeism rates for April were 0.8%, more than double the average rate for April 1999 (0.3%) (Figure 13). The increase in weekly absenteeism rate for April was not reflected in the corresponding trends in influenza-like illness consultations and laboratory reports.

Figure 12. Laboratory reports of influenza, 2000, by age group and sex

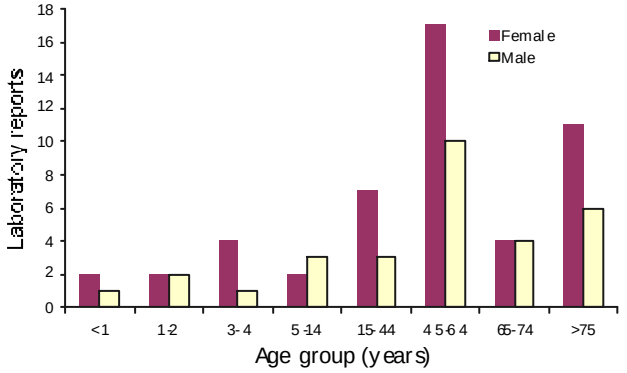
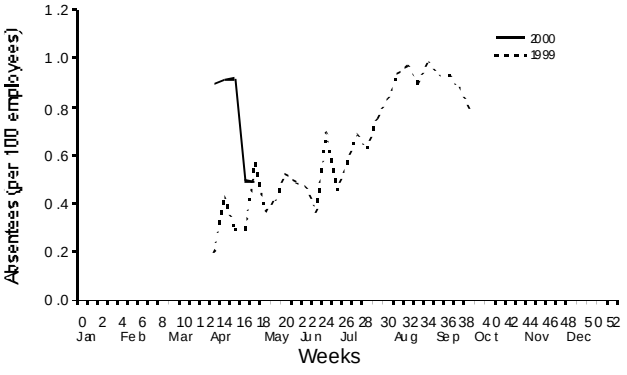


Figure 13. Absenteeism rates in Australia Post, 2000



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 virus which cause the potentially fatal disease Australian encephalitis in humans. Currently 28 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 2000;24:8-9.

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6. PathCentre, Western Australia
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Sentinel chicken serology was carried out for 26 of the 28 flocks in Western Australia in March and April 2000. Widespread activity was detected in the Kimberley, Pilbara, Gascoyne and Murchison regions in March and April 2000 and at one site in the Mid-West during April. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 6. A number of the later seroconversions have not yet been confirmed.

These high levels of MVE virus activity have occurred as a result of high wet season rainfall in the Kimberley region and high cyclonic rains and extensive flooding in the Pilbara, Gascoyne and Murchison regions. MVE virus antibodies were detected in 3, possibly 4, chickens in the Dongara flock (Mid-West) in late April. This is the furthest south the virus has ever been detected. A survey to determine MVE antibody levels in domestic chickens located in this region and areas further south is being carried out to determine the southern limit of MVE virus activity in Western Australia.

A number of media warnings have been issued by the Health Department of Western Australia to warn residents living in the northern areas of Western Australia of the increased risk of disease. Additional warnings were also sent out by the Regional Public Health Units to Aboriginal communities in the regions.

Table 6. Flavivirus seroconversions in Western Australian sentinel chicken flocks in March and April 2000

	March 2000			April 2000		
Location	MVE	MVE/KUN	KUN	MVE	MVE/KUN	KUN
Kimberley						
Wyndham			2			
Kununurra		1		1		1
Halls Creek	1	2	1	4		
Fitzroy Crossing	2			1	1	
Derby*				3#	5#	
Pilbara						
Port Hedland*	3			2	1#	4#
Karratha	4			3#		
Harding Dam*	2	4		8	1	
Marble Bar	3	1		6#	1#	
Pannawonica	3	2		3	1	
Tom Price	1			3#		
Paraburdoo	1					
Onslow	3					
Ophthalmia Dam	2	6				
Newman	1			4		
Exmouth				1	1	
Gascoyne						
Carnarvon		1		2#		
Murchison						
Meekatharra	1					
Mid-West						
Dongara				2#	2	

* 2 flocks of 12 chickens at these sites.

\# Some of these results have not yet been confirmed.

MVE Antibodies to Murray Valley encephalitis virus detected by ELISA.

KUN Antibodies to Kunjin virus detected by ELISA.

Table 7. Flavivirus seroconversions in Northern Territory sentinel chicken flocks in March and April 2000

Location	March 2000			April 2000		
	MVE	MVE/KUN	KUN	MVE	MVE/KUN	KUN
Alice Springs	3#					
Leanyer				1		2
Tennant Creek	1	1	1			3
Katherine				1		

* 2 flocks of 12 chickens at these sites.

\# Some of these results have not yet been confirmed.

MVE Antibodies to Murray Valley encephalitis virus detected by ELISA.

KUN Antibodies to Kunjin virus detected by ELISA.

Eight cases of Murray Valley Encephalitis were reported from Western Australia including 2 cases with onset dates in May 2000.

(It should also be noted that there are now 28 flocks in Western Australia as a new flock at the Curtain Air Base, south of Derby, has now been added to the program).

Serum samples from all seven of the Northern Territory sentinel chicken flocks were tested in the laboratory in March 2000 and from six flocks in April 2000. There were seroconversions to flaviviruses in the flocks located at Alice Springs and Tennant Creek in March and at Leanyer, Katherine and Tennant Creek in April. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 7. A number of media warnings have been issued by the Northern Territory Health Department and to date there have been four cases of Australian encephalitis confirmed from central Australia.

There have been no seroconversions to flaviviruses in the NSW and Victorian sentinel chicken flocks over this period.

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, and annually in HIV/AIDS and related diseases in Australia Annual Surveillance Report. The reports are 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; http://www.med.unsw.edu.au/nchecr.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 31 December 1999, as reported to 31 March, are included in this issue of CDI (Tables 8 and 9).

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 December 1999, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1999	This period 1998	Year to date 1999	Year to date 1998
HIV diagnoses	Female	0	2	0	3	0	0	0	0	5	3	75	90
	Male	0	31	0	11	0	0	8	5	55	37	610	623
	Sex not reported	0	0	0	0	0	0	0	0	0	1	1	7
	Total[1]	0	33	0	14	0	0	8	5	60	41	686	720
AIDS diagnoses	Female	0	2	0	0	0	0	0	0	2	3	16	19
	Male	0	6	0	1	0	0	0	2	9	22	131	279
	Total[1]	0	8	0	1	0	0	0	2	11	26	147	299
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	4	8
	Male	1	0	0	0	1	1	0	3	11	98	146	135
	Total[1]	0	1	0	0	0	1	1	0	3	11	103	154

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

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 December 1999, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	26	603	11	148	61	6	212	113	1,180
	Male	220	10,792	108	1,965	672	79	3,872	907	18,615
	Sex not reported	0	253	0	0	0	0	24	0	277
	Total [1]	246	11,667	119	2,120	733	85	4,121	1,023	20,114
AIDS diagnoses	Female	8	184	0	47	25	3	68	26	361
	Male	86	4,630	36	817	345	44	1,603	347	7,908
	Total [1]	94	4,826	36	866	370	47	1,678	375	8,292
AIDS deaths	Female	3	113	0	31	15	2	48	16	228
	Male	65	3,167	24	564	230	29	1,267	246	5,592
	Total [1]	68	3,288	24	597	245	31	1,321	263	5,837

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

Bulletin Board

Australian School of Environmental Studies
Arbovirus Research in Australia
3-7 July 2000
Couran Cove Nature Resort, Gold Coast, Queensland
Contact Dr Michael Brown
Queensland Institute of Medical Research
PO Box Royal Brisbane Hospital
Herston, Queensland, 4029
Website: http://www.mcaa.org.au

National Centre for Epidemiology and Population Health
International Short Course in Advanced Communicable Diseases Epidemiology
17-28 July 2000
Innovations Building, Mills Road
The Australian National University, ACT
Contact: Ros Hales
Phone: 02 6249 2790
Fax: 02 6249 0740
Email: Ros.Hales@anu.edu.au

Public Health Association of Australia
7th National Public Health Association of Australia Immunisation Conference
2-3 August 2000
Gold Coast International Hotel
Gold Coast, Queensland
Contact: Annette Mellick
Phone: 02 6285 2373
Fax: 02 6282 5438
Email: conference@phaa.net.au
Website: http://www.phaa.net.au

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

The Australasian Society for HIV Medicine
12th Annual Conference
16-19 November 2000
The Carlton Crest, Melbourne, Victoria
Phone: 02 9382 1656
Fax: 02 9382 3699
Email: B.Pearlman@unsw.edu.au

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 Aged Care.

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

A cluster of measles

Rosalind Holland and Robert Hall, Communicable Disease Control Branch , Department of Human Services , South Australia

Over a 4-week period from 10 April to 4 May 2000, 7 cases of serologically confirmed measles were notified to the Communicable Disease Control Branch (CDCB) (Figure 1).

The index case was a 25-year-old female employed as a patient service assistant in a large metropolitan private hospital. She developed a fever on 2 April and a rash on the following day. There was no history of recent travel, contact with a person with a rash, illness, previous measles or measles vaccination. On 13 April her 20-year-old brother (case 3) who resided in the same household presented with a measles-like illness. He had no history of measles or measles vaccination and had therefore received normal immunoglobulin (human) (NIg(H)) and measles-mumps-rubella (MMR) vaccine on 10 April 2000. It could not be established if case 3 was due to a vaccine response or measles infection.

Case 2 was a 33-year-old female ambulance officer whose first symptoms occurred on 6 April and whose rash appeared on 9 April. A second ambulance officer, a 37-year-old male, became ill on 17 April with a rash on 21 April (case 4). Case 2 had no history of contact with a person with an illness with a rash and it could not be determined if she carried patients to or from the same private hospital as the index case. The two ambulance officers did not work together during the infectious period, although they may have been in the same location within 2 hours of each other. Neither case 2 nor case 4 had a history of previous measles infection nor documented evidence of measles vaccination.

On 28 April the 3-year-old daughter of case 4 presented with fever and cough. She developed a rash the following day (case 6). She had documented evidence, including date and batch number, of a single dose of MMR vaccine given at 12 months of age. A 32-year-old female friend visited case 4 at home during his infectious period and developed symptoms on 28 April. A rash appeared on 3 May (case 7). This woman reported that she had not only had measles as a child but had also been vaccinated.

Case 5 was a 41-year-old female with a mild illness and an evanescent rash but positive measles serology. No epidemiological links could be established with the other cases; however, serological tests in a low prevalence community have a low positive predictive value and a thorough assessment of the clinical illness remains an important guide to the correct interpretation of positive IgM results. Serology for other rash illnesses was not diagnostic.

To prevent further transmission, general practitioners and pathology collection centres were asked to identify patients or staff who may have had contact with cases or who may have been in the waiting room up to 2 hours after a case had been present. MMR vaccine or NIg(H) was offered to persons who had been exposed, and a letter describing signs and symptoms of measles was distributed. Local Immunisation Co-ordinators employed by the Divisions of General Practice provided information to GPs, assisted them with control activities and co-ordinated additional supplies of MMR vaccine. As at 25 May 2000 no further cases of serologically confirmed measles have been reported. Serum samples tested by the Victorian Infectious Diseases Reference Laboratory on 3 of the 7 cases were Reverse Transcriptase Polymerase chain reaction negative.

Figure 1. Notifications of measles cases, South Australia, 1 April to 26 May 2000, by day of onset,

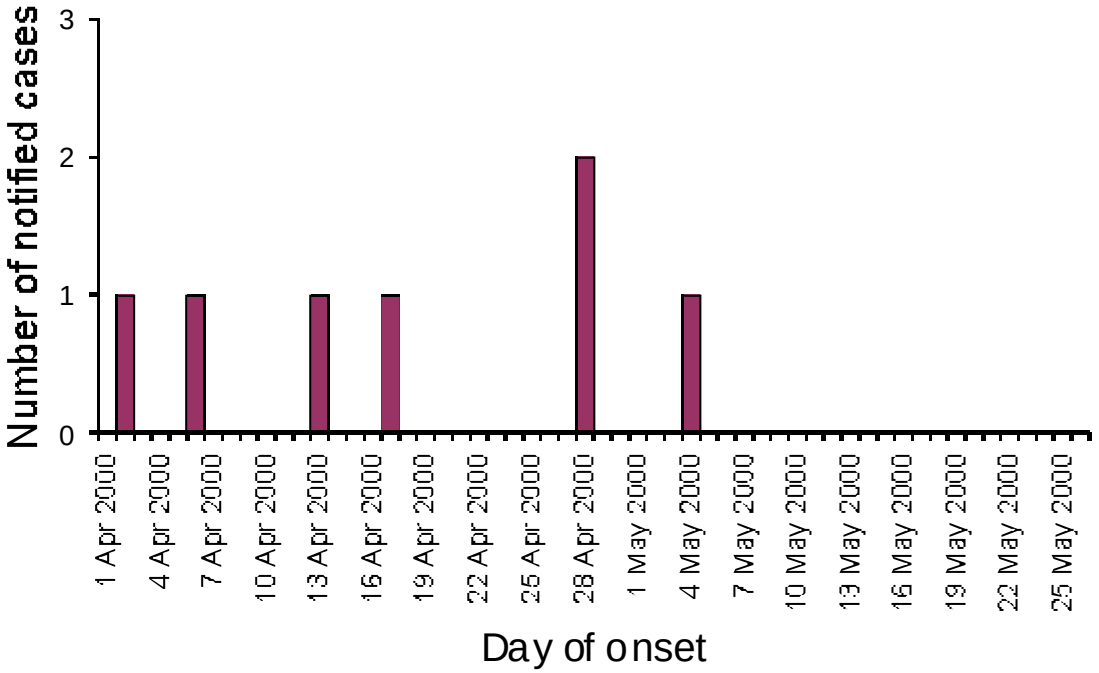

Table 1. Selected features of measles cases, South Australia, April to May 2000

Case	Rash onset	Date of IgM+	Age	Sex	Vaccine	Comment
1	3/4/00	7/4/00	25	F	unknown	Patient assistant in a private hospital, parvovirus IgG detected, rubella negative
2	9/4/00	17/4/00	33	F	? 1 dose	Ambulance officer, parvovirus negative, rubella IgG detected
3	18/4/00	2/5/00	20	M	10/4/00	Brother of case 1, NIg(H) on 10/4/00
4	21/4/00	1/5/00	37	M	unknown	Ambulance officer, parvovirus negative, rubella IgG detected
5	25/4/00	27/4/00	41	F	unknown	
6	29/4/00	1/5/00	3	F	1/5/98	Daughter of case 4, parvovirus negative, rubella IgG detected
7	3/5/00	3/5/00	32	F	1974	Friend of case 4

This cluster of measles cases is the first notified in South Australia (SA) since April 1999 and raises several important issues. The median age of patients in this cluster was 32 with the range of 3-41 years. Measles vaccination was introduced in SA in 1970 and it has been assumed that persons born before then will have immunity to measles from contact with wild disease. Additionally, a serosurvey conducted in 1997 showed that only 3% of persons born before 1975 were seronegative for measles IgG (CDCB, unpublished data). Control of transmission requires a rapid response and these cases were notified only after the diagnosis had been confirmed, not on suspicion as is required by the SA *Public and Environmental Health Act*. There is an apparent lack of appreciation by health care establishments of the infectivity of measles and the need to exclude or isolate people where measles was a possible diagnosis. The episode also illustrates the need for health care workers to be immune to the vaccine-preventable diseases of childhood. Protocols requiring documented evidence of MMR vaccination of their staff members should be instituted.

Cholera in Pohnpei

*Adapted from a report forwarded from the Pacific Public Health Surveillance Network**

The Federated States of Micronesia reported an outbreak of Cholera (serotype Ogawa), which was confirmed by the Public Health Laboratory on Guam on 8 May 2000. The primary symptom was an acute onset of watery diarrhoea with intermittent vomiting and dehydration. The outbreak was exacerbated by the death of the high-ranking traditional chief of Kitti on 27 April 2000. The funeral was followed by several days of feasting and drinking sakau (kava).

The majority of cases were from Kitti or were connected to the funeral. They either attended the funeral or ate food taken from the feast. There have been no fatalities.

As of 11 May 2000, 36 cases were admitted to the Pohnpei State Hospital. The first two cases were admitted on 17 and 19 April. Five cases were admitted on 1 May and since then, the daily admissions fluctuated between one to six cases.

The outbreak was limited to the island of Pohnpei with a strong focus in Kitti Municipality. The preponderance of cases resided in Kitti Municipality and attended the funeral. The rural dispensaries in the other municipalities did not experience an increase in diarrhoea despite increased surveillance activities.

The 36 patients admitted to the hospital were aged from 2 to 81 years with only three under the age of 15 years.

The public health response included closure of all schools and sakau bars, and curtailing of all travel to the outer islands. The hospital established a separated ward for cholera patients with limited family access and with no food allowed in or out of the cholera wards. The Division of Primary Health Care conducted follow-up visits to the homes of all admitted patients. Household members were provided with antibiotics and printed health education materials on water disinfection and safe disposal of faeces. In addition, community leaders were being contacted and their assistance requested. Radio announcements on preventative health measures were broadcast throughout the day.

* Prepared by JP Chaine, PIHOA Regional Epidemiologist

Thanks also to the Pohnpei and Federated States of Micronesia health authorities for sharing this information

Overseas briefs

Source: World Health Organization (WHO)
This material has been summarised from information on the WHO Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the CDI homepage.

Cholera in Somalia

From 1 January to 21 April 2000, 2,232 cases of cholera and 230 deaths have been reported. Cases have been confirmed in Bay, Lower Juba, Lower Shabelle, Mogadishu and Mudug. In areas where international teams are present, the case-fatality rate is lower due to effective case management and the implementation of adequate control measures. It should be noted however, that cholera control in Somalia is complex and difficult as a result of problems of security, accessibility and the recent drought.

WHO continues to carry out cholera control coordination activities through the cholera task force, which collects and compiles data to be shared with partners, assists in processing stool samples, provides cholera supplies as required and gives training support. Agencies involved include: UNICEF, *Action internationale contre la faim*, the International Medical Corps, *Médecins sans frontières*, the Coordinating Committee of the Organization for Voluntary Services and the Somali Red Crescent Society.

Yellow fever in Nigeria

The Federal Ministry of Health has confirmed 2 cases of yellow fever in Kano State.

In collaboration with WHO, UNICEF and Médecins sans Frontières, the Ministry is planning to conduct a mass vaccination campaign in the affected areas of Kano State and Ekiti State.

Meningococcal disease, serogroup W135

This update concerns cases of meningococcal disease associated with international travel and reported previously, as well as confirmed cases of *Neisseria meningitidis* serogroup W135.

New and revised notifications of these cases from 28 February 2000 have been received from the following countries:

Country	Cases	Deaths
Finland	1	
France	18	4
Germany	2	
Iran (Islamic Republic of)	2	
Italy	0	
Kuwait	1	
Morocco	3	1
Netherlands	6	
Oman	18	2
Saudi Arabia	241	59
Singapore	4	
Spain	0	
United Kingdom	31	5
United States	3	

WHO is actively monitoring the situation and requesting countries to send specimens to WHO.

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CDI is produced every four weeks by the National Centre for Disease Control, Department of Health and Aged Care, GPO Box 9848, Canberra, ACT, 2601; Fax: (02) 6289 7791, Phone: (02) 6289 8245; email: cdi.editor@health.gov.au.

This journal is indexed by *Index Medicus* and Medline.