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CONTENTS

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ISOLATION OF VANCOMYCIN-RESISTANT ENTEROCOCCI IN QUEENSLAND, CASE 1

David Paterson[1], *Anthony Jennings*[1], *Amanda Allen*[1], *Kevin Sherlock*[1] *and Michael Whitby* [2]

Abstract

Vancomycin-resistant enterococci are increasingly being reported from many parts of the world. We describe a case of peritonitis with *Enterococcus faecium* exhibiting the *van A* phenotype. The organism was resistant to vancomycin, teicoplanin, amoxycillin and high levels of streptomycin. Rectal swabs from more than 25 other patients who were in the hospital at the same time were negative. No staff members were found to be colonised. Infection control measures were effective in preventing the spread of the resistant *Enterococcus faecium.* Regular surveillance of enterococcal isolates and faecal specimens or rectal swabs of patients at high risk may be justified to determine the level of vancomycin resistance in Australian hospitals. ***Comm Dis Intell* 1996; 20; 400-40**1.

Introduction

Enterococci are common nosocomial pathogens and are intrinsically resistant to a large number of antibiotics. Amoxycillin is the drug of choice for most infections, with vancomycin being used in cases of amoxycillin resistance or penicillin allergy. If there is amoxycillin and vancomycin resistance, teicoplanin is usually the only readily available alternative. However, enterococci with the *van A* phenotype, resistance to teicoplanin as well as vancomycin exists.

Vancomycin-resistant enterococci (VRE) have been described in Europe and the United States of America since the late 1980s. Three phenotypes (*van A*, *van B* and *van C*) are recognised. The first case in Australia was described at the Australasian Society for Infectious Diseases meeting in Darwin in May 1995[1]. Few cases have been detected in Australia since then.

We describe a case of infection with *van A* phenotype vancomycin-resistant *Enterococcus faecium* in a patient in Queensland. We also report on an investigation into carriage by staff members and other patients.

Case report

A 65 year old man with a history of end-stage renal failure (treated with chronic ambulatory peritoneal dialysis) presented with peritonitis in August 1996. He had a history of hypertension and aortic stenosis requiring prosthetic heart valve replacement. He had not received medical care outside Queensland, nor had he been accommodated near patients from interstate or overseas. Several courses of vancomycin had been required as empiric therapy for suspected peritonitis.

Peritoneal fluid bags grew *Bacteroides fragilis*. A perforated diverticular abscess was suspected and laparotomy performed. The peritoneal fluid obtained during laparotomy grew *Enterococcus faecium*, methicillin-resistant *Staphylococcus aureus, Lactobacillus* species and *Clostridium perfringens.*

Enterococcus faecium was identified according to the following criteria: nonmotile, nonpigmented, catalase negative Gram-positive cocci, Lancefield group D, growth in 6.5% NaCl, pyrrolydonylarylamidase positive, pyruvate negative. Both API Strep and Vitek GPI identified the organism as *Enterococcus faecium*.

The organism was resistant to amoxycillin (no zone with 10 mcg disc using NCCLS methods) and penicillin minimal inhibitory concentration (MIC) >64 mg/L by E test. It was also resistant to vancomycin (MIC >256 mg/L by E test) and teicoplanin (MIC 32 mg/L by E test). Thus the organism meets the description of the *van A* phenotype. High level resistance to streptomycin was demonstrated (MIC >2,000 mg/L). There was no high level resistance to gentamicin. The organism was resistant to ciprofloxacin (no zone with 5 mcg disc) and trimethoprim-sulphamethoxazole (no zone with 1.25/23.75 mcg disc). A 33 mm zone was found with pristinamycin (15 mcg disc).

The patient was transferred to a single room. Disposable gloves and a plastic apron were worn by doctors and nurses entering the room. A stethoscope, sphygmomanometer and thermometer were dedicated to the room.

Rectal swabs were collected from all patients who had been in the same ward as the index patient. These were plated onto blood agar containing vancomycin (3 mg/L), colistin (7.5 mg/L), nystatin (12,500 IU/L) and gentamicin (8 mg/L). None of the six patients tested was positive. Rectal swabs from twenty other renal unit patients were negative for VRE. A rectal swab obtained subsequently from the index patient, plated onto the antibiotic supplemented blood agar grew the resistant *Enterococcus faecium*.

Environmental samples collected from the bed, door handle and drawers around the patient were negative for *Enterococcus faecium*.

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Medical, nursing, paramedical and environmental services staff working in the patient's ward were tested for hand carriage of the resistant enterococcus. A modified glove fluid method was used. Twenty millilitres of trypticase soy broth with 2% Tween 80 was placed in a plastic bag. The hands of the staff member were placed individually in the broth and massaged externally for 30 seconds. The broth was then plated onto blood agar. Thirteen staff members were tested. None had hand carriage of VRE. One registered nurse was found to have hand carriage of a vancomycin-resistant catalase negative Gram-positive coccus which grew on, and blackened, bile aesculin plates. This organism was subsequently identified as *Leuconostoc* species.

Discussion

It is not unexpected that infections with vancomycin-resistant enterococci are being found in Australian patients. It is probable that other patients who have been colonised have not yet been detected. What can be done to identify such patients?

Guidelines produced by the Hospital Infection Control Practices Advisory Committee in the United States of America recommend that even hospitals without known cases should monitor for VRE[2]. This can be done by periodic testing of enterococcal isolates for vancomycin resistance. In addition, periodic screening of rectal swabs (or faecal specimens) from high risk patients can be performed. High risk patients include those in intensive care units, those with end-stage renal failure and those with a history of vancomycin usage. Patients treated with chronic ambulatory peritoneal dialysis are frequently given vancomycin as empiric treatment for peritonitis or catheter exit site infections. They become exposed to low levels of vancomycin for prolonged periods, thus creating an environment for the development of vancomycin resistance.

As *van A* or *van B* phenotypes can be induced by vancomycin use, there is logic in restricting vancomycin use to prevent the development of VRE. Vancomycin use should be discouraged in the following circumstances: *Clostridium difficile* colitis, routine surgical prophylaxis, treatment in response to a single blood culture positive for coagulase negative *Staphylococcus*, initial empiric treatment of febrile neutropaenic patients and treatment of Gram-positive infections in renal failure patients purely for dosing convenience[2].

The patient described here probably developed VRE as a result of exposure of his endogenous enterococcal flora to low levels of vancomycin over a prolonged period. The negative results from many other patients indicate that the prompt use of appropriate infection control measures can prevent nosocomial spread to other patients. No further cases have been identified in the hospital.

The American guidelines appear reasonable to consult if a patient colonised or infected with VRE is detected[2]. Patients with VRE should be housed in a single room or in a multibed room with other patients with VRE. Gloves and plastic aprons should be worn when nursing the patient. Ward contacts of the index patient should be screened, and the patient isolated if VRE is detected. Strict hand washing procedures should be observed. Prolonged intestinal carriage of VRE by patients is well described and efforts should be made to add alerts to the patient's chart so that appropriate isolation can be made on readmission.

Acknowledgments

The clinicians and nursing staff involved in staff and patient surveillance are thanked for their cooperation. Dr Joan Faoagali and Mr Adrian Whitfield (Royal Brisbane Hospital) performed the teicoplanin E test.

References

1. Kamarulzaman A, Tosolini FA, Boquest AL *et al*. Vancomycin resistant *Enterococcus faecium* infection in a liver transplant recipient [abstract]. *Aust NZ J Med* 1995;25:560.
2. Centers for Disease Control and Prevention. Recommendations for preventing the spread of vancomycin resistance. *MMWR Morb Mort Wkly Rep* 1995;44:RR-12.

ISOLATION OF VANCOMYCIN-RESISTANT ENTEROCOCCI IN QEENSLAND, CASE 2

Joan Faoagali[1], *Jan Bodman*[1] *and Alanna Geary*[2]

Abstract

A case of vancomycin-resistant enterococcus (VRE) colonisation is reported. The organism was not isolated from other patients sharing a room with the index case or from the environment. The microbiology laboratory plays an important role in the detection of VRE and in alerting the infection control, medical and nursing staff. Nosocomial transmission of VRE can be prevented by adherence to appropriate infection control procedures. The occurrence of VRE can be prevented by the appropriate use of vancomycin. ***Comm Dis Intell* 1996;20:402-403.**

Introduction

A reduction in the morbidity and mortality due to many bacterial diseases has been documented since antimicrobial agents were introduced for general use in the 1940s[1,2,3]. However, due to the widespread use of antimicrobials, drug resistance has emerged as a major public health problem in both community and institutional settings. Increased microbial resistance has resulted in prolonged hospitalisations and higher death rates from infections. In addition it has necessitated the use of more expensive, and often more toxic, drugs or drug combinations resulting in higher health care costs[4].

Case report

In June 1996, vancomycin-resistant *Enterococcus faecalis* (VRE) and methicillin-resistant *Staphylococcus aureus* (MRSA) were isolated from a groin swab. The swab was collected because the patient had a rash. The patient had been hospitalised in Queensland for four months with a chronic illness. During this time the patient had received several courses of vancomycin. The VRE was a possible *van B* phenotype, with minimal inhibitory concentration (MIC) to vancomycin 16 mg/L (intermediate), MIC to teicoplanin <4 mg/L (sensitive), ampicillin MIC <2 mg/L (sensitive), and no high level resistance to gentamicin.

An investigation was set up to determine whether patients who had shared a room with the index case were colonised with VRE and whether the resistant organism was present in the environment. A rectal swab was collected from the patient with VRE (the index case) for screening. This yielded VRE on culture. Rectal swabs were also collected from four patients who had shared a room with the index case and from another patient who had previously been in the same room for a month. These were all screened for VRE and found to be negative. Environmental samples were collected from 20 sites in the patient's room and cultured. No VRE were isolated, although non-VRE *Enterococcus faecalis* was found in some areas including curtain rails.

The index patient was isolated in a single room. The room in which the patient had been nursed was subjected to 'terminal' cleaning and the room closed over the weekend. A second groin swab collected two weeks later yielded VRE and MRSA on culture.

The patients who had shared a room with the index case were nursed together in one area.

Discussion

This appears to have been an isolated case. The laboratory has been screening for VRE routinely since October 1995. Rectal swabs collected weekly from haematology/oncology and intensive care patients were screened for VRE using blood agar with amikacin 8 mg/L and vancomycin 6 mg/L. About 400 rectal swabs have been screened and no VRE detected. Our patient did not fulfil the criteria for routine screening and hence was not screened by this method. However this laboratory method would not have detected VRE in this patient as the isolate did not have high level resistance to aminoglycosides and would therefore not have grown on our selective medium. As a result of this an alternative VRE screening medium is currently being investigated.

The microbiology laboratory plays a fundamental role in the surveillance and control of VRE. This is achieved through the use of good technical procedures and prompt reporting of VRE to the medical, nursing and infection control staff.

Enterococci should be identified to species level. Antimicrobial susceptibility testing on enterococci isolated from blood, sterile body sites and other sites (as clinically indicated) should include determination of vancomycin resistance as well as high level resistance to penicillin and aminoglycosides. The laboratory's method of susceptibility testing should include use of the control organism *Enterococcus faecalis* ATCC 51299. This strain has a moderate level of vancomycin resistance mediated by the *van B*

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gene which, unlike high level resistance mediated by the *van A* gene, is difficult to detect.

Efforts to contain VRE and prevent its spread to others is necessary for the management of patients colonised with this organism. Affected patients should be isolated and standard infection control principles adhered to. Particular attention should be paid to the decontamination and disinfection of the environment around the patient. The patient should remain in isolation while colonised with VRE or if readmitted without VRE 'clearance'. The patient may be 'delisted' if rectal and lesion swabs for VRE are persistently negative (three cultures on consecutive weeks) in hospital.

A record of VRE cases should be kept for the epidemiological tracking of cases including their location, antibiotic history and risk factors. Vancomycin use should be reserved for specific conditions and hospitals should develop guidelines for the proper use of vancomycin[5].

References

1. Neu HC. Antimicrobial agents. Role in preosocomial infections. In Wenzel RP. Prevention and control of nosocomial infections. Baltimore: Williams and Wilkins 2nd ed., 1993.
2. French GL, Phillips J. Antimicrobial resistance in hospital flora and nosocomial infections. In CG Mayhall, ed. *Hospital epidemiology and infection control.* Baltimore: Williams and Wilkins, 1996.
3. Landman D, Quale JM, Oydna E *et al.* Comparison of five selective media for identifying faecal carriage of VRE. *J Clin Micro* 1996;34:751-752.
4. Centers for Disease Control and Prevention. *Addressing emerging infectious disease threats, a prevention strategy for the United States.* Atlanta: United States Department of Health and Human Services, Public Health Service, 1994.
5. HospitaL Infection Control Practices Advisory Committee. Recommendations for preventing the spread of vancomycin resistance. *Infection Control Hospital Epidemiology 1995;16:105-113.*

ACUTE FLACCID PARALYSIS SURVEILLANCE IN AUSTRALIA: THE FIRST YEAR

Ana Herceg[1,4]*, Margery Kennett*[2,4]*, Jayne Antony*[3,4] *and Helen Longbottom*[1,4]

Abstract

Surveillance for acute flaccid paralysis commenced through the Australian Paediatric Surveillance Unit in March 1995. Thirty-five cases were reported in the first year, giving an estimated incidence of 0.90 cases per 100,000 children under the age of 15 years. Nearly half the cases were Guillain-Barre syndrome. No cases of poliomyelitis were identified. This surveillance scheme will assist in the process of certification of the eradication of poliomyelitis in Australia and the World Health Organization Western Pacific Region. ***Comm Dis Intell* 1996;20:403-405.**

Introduction

The World Health Organization (WHO) aims to eradicate poliomyelitis from the world by the year 2000[1]. Poliomyelitis has already been eradicated from the Americas[2]. For a country to be declared polio free it needs to meet a number of requirements, including polio vaccination coverage of more than 80%, no confirmed poliomyelitis cases for three years and adequate surveillance and investigation of suspected poliomyelitis cases.

Australia has not had any poliomyelitis cases reported through the National Notifiable Diseases Surveillance System since one case was reported in 1986, one case in 1978 and two cases in 1977[3]. The WHO however considers the detection and investigation of all cases of acute flaccid paralysis (AFP) as an essential and sensitive method of detecting wild poliovirus.

The differential diagnosis of acute flaccid paralysis includes Guillain-Barre syndrome, transverse myelitis and traumatic paralysis[4]. Other viruses (for example enterovirus types 70 and 71) may mimic polio. All these events are rare and little is known about the incidence, clinical course and outcomes of AFP in Australia.

In March 1995, surveillance of acute flaccid paralysis commenced through the Australian Paediatric Surveillance Unit (APSU). The aims of the study were to describe the incidence, causes and clinical picture of AFP cases in Australia and to determine whether any cases of AFP are caused by paralytic 'wild' poliovirus.

Methods

A case of acute flaccid paralysis was defined as a child aged less than 16 years with:

acute onset of flaccid paralysis in one or more limbs

or

acute onset of bulbar paralysis.

The Australian Paediatric Surveillance Unit (a unit of the Australian College of Paediatrics) conducts active, prospective national surveillance of selected rare paediatric

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conditions by sending a reply-paid report card each month to over 900 paediatricians in Australia. Paediatricians are asked whether or not they have seen any of a number of conditions listed on the card. Over 90 per cent of paediatricians return the card each month. Acute flaccid paralysis was included on the APSU card from March 1995.

In addition to returning the APSU card, paediatricians were asked to report cases by telephone to the principal investigator. This was so that paediatricians could be reminded of the WHO requirements for stool testing and asked to comply with them.

Paediatricians were asked to collect two stool specimens from the case, preferably 24 hours apart, within 14 days of the onset of paralysis. Specimens were then sent for viral culture, in particular looking for poliovirus. If poliovirus was isolated, it was sent to the National Polio Reference Laboratory in Melbourne for characterisation as wild or vaccine-like. This protocol has recently changed slightly (see Box).

A two-page questionnaire on the clinical features, laboratory investigations and final diagnosis of the case was sent to reporting paediatricians and was followed by a second questionnaire at 60 days asking about any residual paralysis.

Data analysis was performed using Epi Info version 6[5].

Cases were classified according to WHO criteria[6] as:

Poliomyelitis: an AFP case with wild poliovirus isolation.

Non-polio AFP: an AFP case with adequate stool specimen testing negative, or with no residual paralysis except if wild virus is isolated, or

Polio-compatible: an AFP case with residual paralysis or who died or was lost to follow-up and for whom stool specimens were either not taken or were inadequate.

Results

There were 35 cases of AFP with onset dates between March 1995 and February 1996, reported by 49 paediatricians. The 35 cases in 12 months corresponded to an incidence of 0.90 per 100,000 children under the age of 15 years.

Nineteen initial reports (54%) were by telephone. Completed questionnaires containing clinical information were provided for 29 cases. The diagnosis alone was provided for four cases and no information at all was available for two cases. Reports were for cases from all States and Territories except the Australian Capital Territory (Table 1).

Of the 33 cases for which information was provided, 18 cases were male and 15 female. Their ages ranged from two months to 12 years. There was no seasonal distribution of cases. Their reported diagnoses are described in Table 2. Forty-eight per cent of cases were diagnosed as having Guillain-Barre syndrome. No cases of poliomyelitis were identified.

One case with a diagnosis of transverse myelitis had been vaccinated with oral polio vaccine seven days prior to developing paralysis; poliovirus type 3 (Sabin-like) was identified from his stool. In addition, the case had a demonstrated seroconversion to poliovirus type 3.

Thirty-two cases were hospitalised and 11 required intensive care admission. Nineteen cases had paralysis of all four limbs, six had paralysis of two limbs, one had paralysis of one limb and no information was reported for seven cases. Six cases had bulbar paralysis and six had respiratory depression. Nine cases had cranial nerve involvement. Eighteen cases had residual paralysis at 60 days. There were no deaths.

Only eight cases had two stool samples taken and tested according to WHO criteria. A further four cases had one stool sample taken. As a result, of the 35 total cases, 17 were classified as non-polio and 18 were classified as polio compatible.

Discussion

The first year of surveillance of acute flaccid paralysis in Australia has supported the presumption that Australia is free of wild poliomyelitis. The estimated incidence of 0.90 cases of AFP per 100,000 children under the age of 15 years is close to the one case per 100,000 expected for a poliomyelitis-free country. Surveillance of AFP in the Americas found an annual incidence of 1.4 cases per 100,000 children

Table 1. Acute flaccid paralysis cases reported by State and Territory, March 1995 - February 1996

State / Territory	Number of cases
ACT	0
NSW	14
NT	1
Qld	9
SA	2
Tas	2
Vic	3
WA	4

Table 2. Diagnosis for 33 cases of acute flaccid paralysis, Australia, March 1995 - February 1996

Diagnosis	Number of cases
Guillain-Barre syndrome	16
Transverse myelitis	5
Demyelination	3
Encephalomyelitis	2
Trauma	2
Hypotensive brainstem necrosis or demyelination	1
Lumbar radiculopathy	1
Post drug polyneuromyopathy	1
Infant botulism	1

under the age of 15 in 1991, the year in which the last case of polio occurred[7]. In the United Kingdom, the rate is approximately one case per 100,000 children under the age of 16 years[8]. The three cases of AFP identified in Victoria are fewer than the expected nine cases for that population and indicate there may be under-reporting in that State. In other States and Territories, the numbers of cases were as expected.

No cases of poliomyelitis were identified by this surveillance system. Although according to WHO criteria, 18 of the cases of AFP would be classified as polio compatible, most cases had multiple investigations and were treated by paediatric neurologists. In these circumstances it is unlikely that the diagnoses reported are incorrect. Paediatricians are encouraged however to continue stool testing to exclude not only poliomyelitis but other viral causes of paralysis. In one case during this study the testing identified an echovirus type 9 as the probable cause of the illness.

The causes of AFP identified in this study are consistent with the experience of similar surveillance in the United Kingdom, with 48% of cases being Guillain-Barre syndrome and 15% transverse myelitis[8]. While no definite vaccine-associated paralysis was identified, one case with a diagnosis of transverse myelitis did have Sabin-like poliovirus type 3 identified from his stool and documented seroconversion to the same virus. It is estimated that one case of vaccine-associated paralysis will occur with every 2.5 million doses of oral polio vaccine distributed[9]. Surveillance of AFP would be expected to occasionally identify these cases.

Australia will shortly be undergoing scrutiny to determine whether wild poliovirus exists in this country, as part of the process of certification of poliomyelitis-free status in the Western Pacific Region of the World Health Organization. A Regional Commission for the Certification of Poliomyelitis Eradication met for the first time in April 1996. Surveillance of cases of AFP will provide information to assist in certifying Australia and the Region polio-free.

While it is unlikely that Australia does have indigenous wild poliovirus, the potential for importation of the virus from other countries is still high. In 1992 in the Netherlands a wild poliovirus type 3 strain introduced from India was responsible for a large outbreak of poliomyelitis in a community opposed to immunisation[10]. In 1993 the same virus was identified in Canada through active surveillance in a linked community[11]. Continued high rates of routine vaccination against polio are still required in Australia to prevent similar importation. Surveillance for poliomyelitis is also important so that if imported poliomyelitis does occur, an immediate public health response can be commenced.

Acknowledgements

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References

1. World Health Assembly. *Global eradication of poliomyelitis by the year 2000*. Geneva: World Health Organization, 1988 (Resolution WHA11.28).
2. Centers for Disease Control and Prevention. Certification of poliomyelitis eradication - the Americas, 1994. *MMWR Morb Mort Wkly Rep* 1994;13:720-722.
3. Hall R. Notifiable Diseases Surveillance, 1917 to 1991. *Comm Dis Intell* 1993:17:226-236.
4. Benenson AS, editor. *Control of Communicable Diseases Manual*, Sixteenth Edition. Washington: American Public Health Association 1995.
5. Dean AG, Dean JA, Coulombier D *et al*. Epi Info, Version 6: a word processing, database, and statistics program for epidemiology on microcomputers. Atlanta: Centers for Disease Control, 1994.
6. Report of the Interim Meeting of the Technical Advisory Group on the EPI and Poliomyelitis Eradication in the Western Pacific Region, Beijing, October 29-November 2 1994. Report series number RS / 94 / GE / 40 (CHN).
7. Andrus JK, de Quadros CA, Olive J-M. The surveillance challenge: final stages of eradication of poliomyelitis in the Americas. *MMWR Morb Mort Wkly Rep CDC Surveill Summ* 1992:41:21-26.
8. Salisbury DM, Begg NT. Surveillance of poliomyelitis in the United Kingdom. *Public Health Rev* 1993-4;21:35-40.
9. Strebel PM, Sutter RW, Cochi SL, *et al*. Epidemiology of poliomyelitis in the United States one decade after the last reported case of indigenous wild virus-associated disease. *Clin Inf Dis* 1992;14:568-579.
10. Oostvogel PM, Van Wijngaarden JK, Van der Avoort HGAM *et al*. Poliomyelitis outbreak in an unvaccinated community in the Netherlands, 1992-93. *Lancet* 1994;344:665-70.
11. Health and Welfare Canada. Wild poliovirus isolated in Alberta, 1993. *Canada Comm Dis Rep* 1993;19:57-58.

Reporting cases of acute flaccid paralysis

Paediatricians should report cases of acute flaccid paralysis to the Australian Paediatric Surveillance Unit (APSU), and by telephone to Dr Ana Herceg on (06) 289 8638.

Stool specimen testing

Two stool specimens from every case of AFP should be collected, preferably 24 hours apart, within 14 days of the onset of paralysis. Following a World Health Organization Technical Advisory Group meeting in Canberra in April 1996, the laboratory protocol for testing stool specimens for AFP has changed. The WHO states that specimens should be tested in a WHO certified laboratory. In Australia, only the National Polio Reference Laboratory is certified.

All stool specimens should now be sent directly to the National Polio Reference Laboratory in Melbourne, not to the State or Territory virology laboratory as previously requested. Laboratories can obtain information on specimen transport from Mrs Margery Kennett on (03) 9280 2397. The National Polio Reference Laboratory will pay for specimen transport.

OVERSEAS BRIEFS

Source: World Health Organization

Viral meningitis, Romania

The National Reference Centre for Enteroviruses at the Institute Cantacuzino, Bucharest has confirmed enterovirus in specimens taken from two patients hospitalised with a clinical diagnosis of aseptic meningitis. Since 30 July, 342 cases of suspected viral meningitis and meningoencephalitis have been reported. Of these, 201 were still in hospital on 5 September while 124 had recovered and been released. Seventeen patients died. The epidemic has been characterised by a disproportionate involvement of elderly people, lack of clusters and a majority of cases living in Bucharest.

Public health control measures recommended by the Ministry of Health focus on personal and community hygiene. The Ministry of Health does not consider there is any risk for tourists in Romania or of importing food from Romania.

WHO interagency effort to control meningitis epidemics in Africa

The World Health Organization (WHO), in consultation with international partner agencies and governments, has launched a major initiative to control the recurring cycle of cerebrospinal meningitis epidemics in Africa and reduce their devastating consequences for people on the continent.

Upwards of 140,000 cases of cerebrospinal meningitis have occurred in Africa since the beginning of 1996, resulting in at least 15,000 deaths - the highest numbers ever reported to WHO for a single year in Africa. More than 95% of the cases and deaths were in the 'meningitis belt' of sub-Saharan Africa where the disease is endemic, a region stretching from Ethiopia in the east to Senegal on the west coast.

In the meningitis belt of Africa, epidemics of cerebrospinal meningitis recur in cycles of 8-12 years during the dry season, although intervals between epidemics have shortened and become more irregular in some countries since the beginning of the 1980s. Epidemic cycles in a particular country can last for two or three consecutive years. As is usually the case in West Africa, the current epidemic is caused by *Neisseria meningitidis* serogroup A. This is confirmed by analysis of specimens carried out by the WHO Collaborating Centre in Oslo, Norway.

Under the new initiative, WHO will work with international partners over the next three years to enable affected countries to identify epidemics at their outset, take timely preventative measures to reduce the spread of the disease, and provide rapid treatment of cases. To assist in this effort, WHO has prepared a new set of technical guidelines updating current knowledge on cerebrospinal meningitis.

The new WHO initiative will focus on strengthening national and regional health systems in surveillance, vaccination, vaccine supply, case management, epidemic response and epidemic preparedness.

Cholera, Philippines

The Govenrnment of the Philippines reported an outbreak of cholera on 10 September 1996. From 1 to 8 September, a total of 284 patients with suspected cholera were admitted to three hospitals in Manila. Affected areas were Paco and San Andres in Manila. There were five deaths due to severe dehydration. Most of the cases were children younger than 15 years of age. *Vibrio cholerae* serotype Ogawa was isolated from 33 patients. The number of admissions has decreased from 7 September.

COMMUNICABLE DISEASES SURVEILLANCE

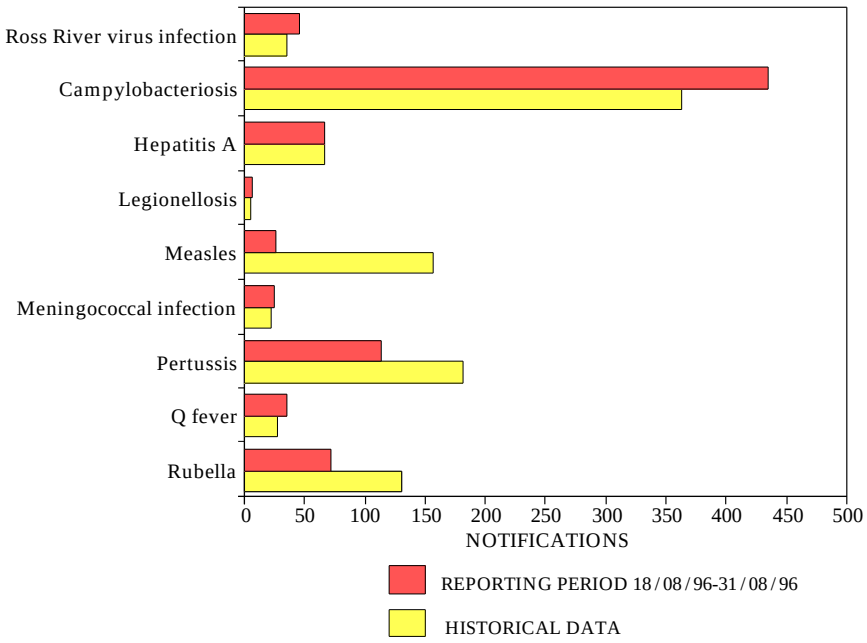
National Notifiable Diseases Surveillance System

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia-New Zealand. The system coordinates the national surveillance of 41 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 legislation. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1996;20:9-10.

Reporting period 18 to 31 August 1996

There were 1,930 notifications received for this two-week period (Tables 1, 2 and 3). The number of reports for selected diseases has been compared with average data for this period in the previous three years (Figure 1).

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



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

Table 1. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 18 to 31 August 1996

DISEASE[1]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2]			
									This period 1996	This period 1995	Year to date 1996	Year to date 1995
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae b infection	0	0	0	2	1	0	1	0	4	3	43	48
Measles	0	10	4	2	1	3	5	1	26	39	329	1006
Mumps	0	1	0	NN	0	0	3	2	6	4	82	97
Pertussis	1	14	0	35	18	0	39	7	114	173	2030	2757
Rubella	1	4	1	34	12	0	19	1	72	172	1668	1662
Tetanus	0	0	0	0	0	0	0	0	0	0	1	3

NN Not Notifiable.

1. No notifications of poliomyelitis have been reported since 1986.

2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 2. Notifications of other diseases[1] received by State and Territory health authorities in the period 18 to 31 August 1996

DISEASE	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2]			
									This period 1996	This period 1995	Year to date 1996	Year to date 1995
Arbovirus Infection (NEC)[3,4]	0	0	0	0	0	0	0	1	1	0	127	53
Barmah Forest virus infection	0	2	-	13	0	0	-	-	15	16	629	612
Ross River virus infection	0	10	5	28	1	-	0	2	46	27	7432	2329
Dengue	0	0	0	0	0	-	0	0	0	1	27	22
Campylobacteriosis[5]	7	-	4	99	126	21	115	63	435	470	7874	6798
Chlamydial infection (NEC)[6]	0	NN	28	153	1	6	90	58	336	218	5000	3271
Donovanosis	0	NN	0	0	NN	0	0	1	1	2	33	53
Gonococcal infection[7]	0	15	31	59	0	0	17	43	165	218	2588	1981
Hepatitis A	6	25	0	17	1	0	15	2	66	60	1623	1027
Hepatitis B incident	0	0	0	1	0	0	1	3	5	11	144	220
Hepatitis C incident	1	0	0	-	0	-	-	-	1	49	18	55
Hepatitis C unspecified	11	NN	12	141	NN	12	221	28	425	2	6653	6291
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	476	15	8
Legionellosis	0	4	0	0	0	0	0	2	6	7	127	125
Leptospirosis	0	1	0	2	0	0	5	0	8	6	164	84
Listeriosis	0	0	0	0	1	0	1	0	2	2	40	43
Malaria	1	11	0	29	2	1	8	0	52	23	600	452
Meningococcal infection	0	10	0	5	0	1	9	0	25	24	268	229
Ornithosis	0	NN	0	0	0	0	2	0	2	7	60	80
Q fever	0	21	0	3	1	0	11	0	36	29	383	315
Salmonellosis (NEC)	3	20	10	26	9	6	31	14	119	128	4136	4337
Shigellosis[5]	0	-	8	6	2	0	3	1	20	31	473	559
Syphilis	0	26	19	11	0	0	0	0	56	52	1008	1276
Tuberculosis	0	5	1	7	0	0	11	2	26	35	750	672
Typhoid[8]	0	0	0	1	1	0	0	0	2	1	58	45
Yersiniosis (NEC)[5]	0	-	0	6	0	0	1	0	7	10	172	233

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

NN Not Notifiable.

NEC Not Elsewhere Classified.

- Elsewhere Classified.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 18 to 31 August 1996

DISEASE[2]	Total this period	Reporting States or Territories	Year to date 1996
Brucellosis	2	Qld	25
Chancroid	0		1
Cholera	0		4
Hydatid infection	1	ACT	30
Leprosy	0		8

1 . Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1995.

2. No notifications have been received during 1996 for the following rare diseases: botulism; lymphogranuloma venereum; plague; rabies; yellow fever; or other viral haemorrhagic fevers.

Figure 2. Measles notifications 1991 to 1996, by month of onset

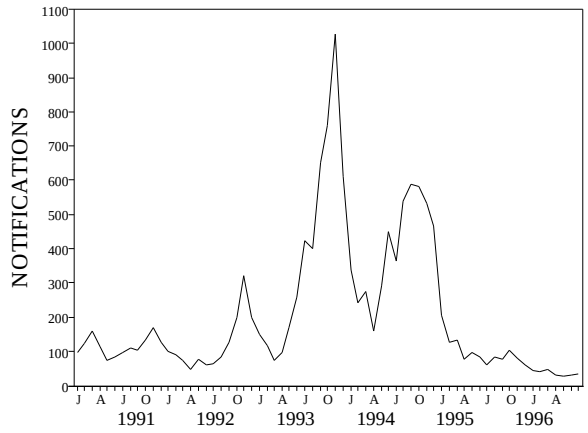
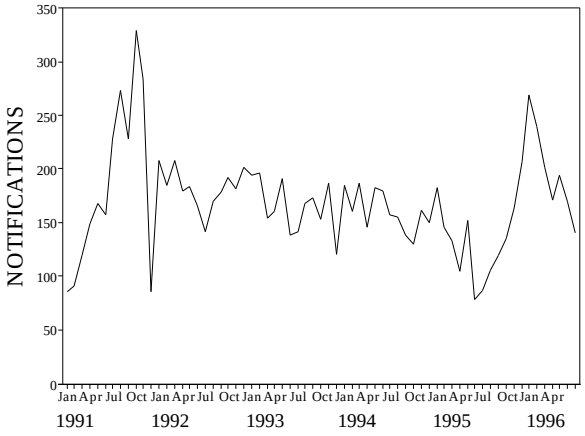


Figure 3. Hepatitis A notifications, 1996, by month of onset

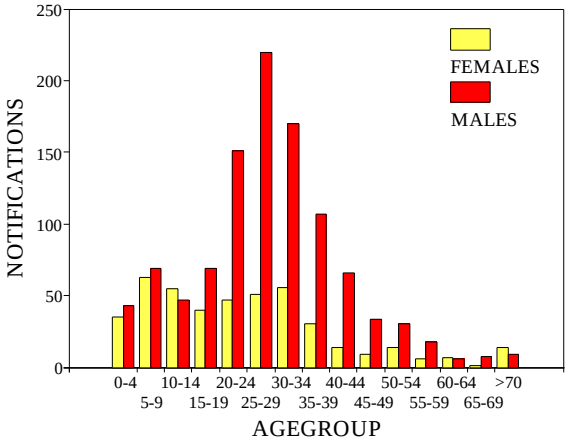


The number of notifications of **measles** has remained low in recent months (Figure 2). A total of 305 cases with onset dates in 1996 has been received of which 164 (54%) were for children under the age of 5 years.

Sixty-six notifications of **hepatitis A** were received this period. The number of reports has fallen in recent months after peaking in January (Figure 3). The highest number of notifications for the year to date has been for males in the 20 to 40 year age goup (Figure 4).

There were 25 notifications of **meningococcal infection** received this fortnight, including 4 cases from the same postcode region of New South Wales. A total of 259 reports with onset dates in 1996 has been received. Of these, 98 (38%) were for children under 5 years of age, and 40 (15%) for the 15 to 19 year old age group.

Figure 4. Hepatitis A notications, 1996, by age group and sex



HIV and AIDS Surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 332 4648 Facsimile: (02) 332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for March 1996, as reported to 30 June 1996, are included in this issue of *CDI* (Tables 4 and 5).

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 March 1996, by sex and State or Territory of diagnosis

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA			
										This period 1996	This period 1995	Year to date 1996	Year to date 1995
HIV diagnoses	Female	0	4	0	2	0	0	4	2	12	7	25	22
	Male	0	37	1	9	5	0	16	4	72	70	196	213
	Sex not reported	0	0	0	0	0	0	0	0	0	0	2	2
	Total[1]	0	41	1	11	5	0	20	6	84	78	223	241
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	4	0	11
	Male	0	23	0	5	0	0	6	1	35	59	108	185
	Total[1]	0	23	0	5	0	0	6	1	35	64	108	197
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	5	6	11
	Male	0	9	0	5	2	0	11	2	29	56	91	177
	Total[1]	0	9	0	5	2	0	11	2	29	61	97	188

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

Table 5. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 1996, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	AUSTRALIA
HIV diagnoses	Female	15	557	4	98	44	4	164	73	959
	Male	168	10002	82	1583	568	70	3369	759	16601
	Sex not reported	0	2048	0	0	0	0	42	0	2090
	Total[1]	183	12614	86	1686	612	74	3584	834	19673
AIDS diagnoses	Female	5	130	0	28	18	2	47	18	248
	Male	72	3789	26	647	272	32	1336	287	6461
	Total[1]	77	3929	26	677	290	34	1390	307	6730
AIDS deaths	Female	2	99	0	22	13	3	36	11	185
	Male	50	2673	20	449	188	21	1052	211	4664
	Total[1]	52	2778	20	473	201	23	1094	223	4864

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

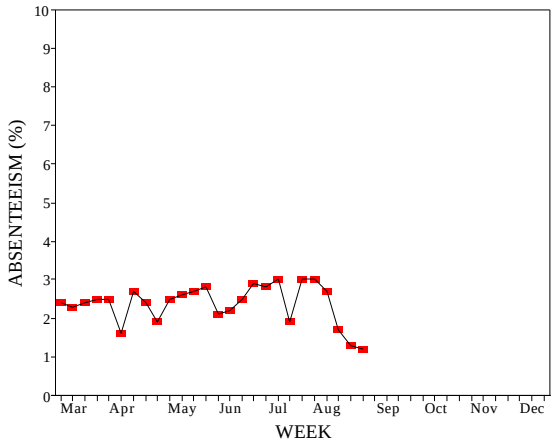
National Influenza Surveillance

Australian Sentinel Practice Research Network; Communicable Diseases Intelligence Virology and Serology Reporting Scheme Contributing Laboratories, New South Wales Department of Health; Victorian Department of Health; World Health Organisation Collaborating Centre for Influenza Reference and Research.

National Influenza Surveillance is conducted from May to September each year. Data are combined from a number of sources to provide an indication of influenza activity. Included are sentinel general practitioner surveillance, absenteeism data from a national employer, and laboratory data from LabVISE and the World Health Organization Collaborating Centre for Influenza Reference and Research. For further information, see CDI 1996;20:9-12.

The absenteeism rate recorded by Australia Post has continued to fall (Figure 5). Consultation rates for influenza-like illness in New South Wales and those recorded by ASPREN have fallen after peaking in late July. Consultations in Victoria peaked in late June. The Northern Territory has reported a dramatic increase in consultations since July after an earlier, much smaller peak in March (Figure 6). However, data for the past month may be subject to revision.

Figure 5. Australia Post absenteeism, 1996, by week



The number of laboratory reports of influenza A are also falling (Figure 7). In the last fortnight, 141 reports were received. Diagnosis was by virus isolation (75), antigen detection (25), single high titre (35) and fourfold rise in titre (6). Of these, 40% of patients were under five years of age and 13% over 65 years of age. For the year to date, 117 reports (10%) have been for persons over 65 years of age.

Fourteen reports of influenza A subtype H_3N_2 were received this fortnight. Of these, 12 were under five years of age.

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) comprises 99 sentinel general practitioners from throughout the country. A total of approximately 9,000 consultations are recorded each week for 12 conditions. Of these, CDI reports the consultation rate for influenza, rubella, measles, pertussis and gastroenteritis. For further information including case definitions see CDI 1996;20:98-99.

Data for week 32 ending 11 August to week 35 ending 1 September respectively are included in this issue of *CDI* (Table 6). The consultation rate for gastroenteritis has remained stable since mid-July. Consultation rates for chickenpox have also remained at a steady level over the last three months. Consultations for rubella, measles and pertussis continue to be reported at low rates.

Figure 6. Sentinel general practitioner influenza reports, 1996, by week

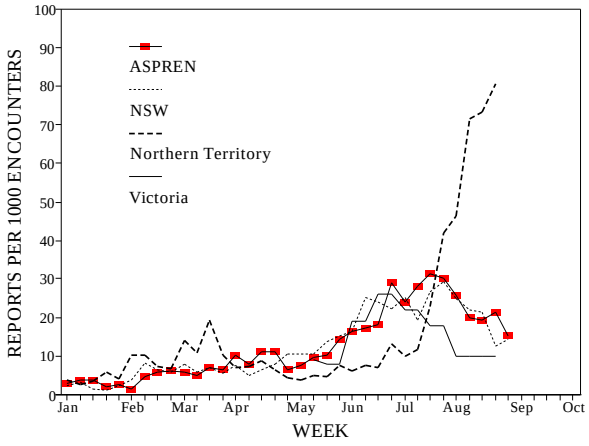


Figure 7. Influenza A laboratory reports, 1996, by method of diagnosis and week of specimen collection

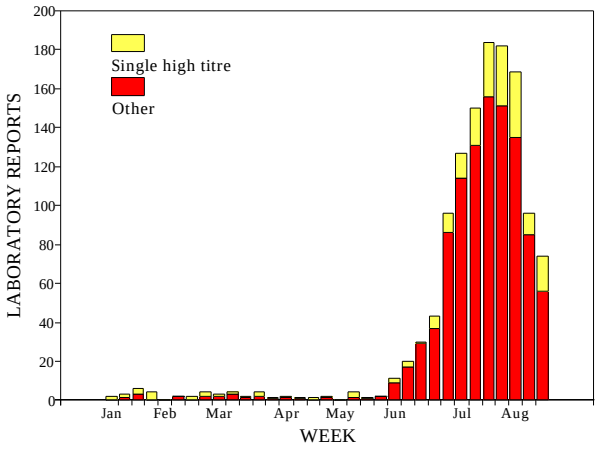


Table 6. Australian Sentinel Practice Research Network reports, weeks 32, 33, 34 and 35, 1996

Condition	Week 32, to 11 August 1996		Week 33, to 18 August 1996		Week 34, to 25 August 1996		Week 35, to 1 September 1996	
	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	159	19.9	160	19.4	166	21.5	100	15.2
Rubella	5	0.6	3	0.4	2	0.3	3	0.5
Measles	0	0	0	0	0	0	0	0
Chickenpox	7	0.9	10	1.2	7	0.9	5	0.8
Pertussis	4	0.5	3	0.4	1	0.1	0	0
Gastroenteritis	112	13.1	115	13.9	107	13.8	109	16.6

Gonococcal Surveillance

John Tapsall, Prince of Wales Hospital, High Street, Randwick NSW 2031, for Australian Gonococcal Surveillance Programme

Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics which are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level resistance to the tetracyclines. Tetracyclines are however not a recommended therapy for gonorrhoea. Comparability of data is achieved through the use of a standardised system of testing and a programme-specific quality assurance programme. Because of the geographic differences in susceptibility patterns, regional as well as aggregated data are presented.

Reporting period 1 July to 30 September 1995

In the third quarter of 1995, AGSP reference laboratories examined 467 isolates of *Neisseria gonorrhoeae*.

Penicillins

This group of antibiotics (penicillin, ampicillin, amoxycillin) remains useful in some parts of Australia. These antibiotics are least effective in the larger population centres of Sydney and Melbourne where more than 30% of isolates were penicillin resistant in this quarter.

Figure 8 shows the proportion of strains fully sensitive to penicillin, less sensitive, relatively resistant or penicillinase-producing (PPNG) in different regions and for all isolates throughout Australia. Strains which are PPNG or in the relatively resistant category usually fail to respond to the penicillins.

There were 30 PPNG detected throughout Australia in this quarter (6.4% of all isolates). Eleven of these were in Sydney (9% of isolates there), 10 in Melbourne (16.4%), 5 in Brisbane (3.3%), 3 in Perth and 1 in Darwin. Local (as opposed to overseas) acquisition of PPNG predominated in Melbourne and Brisbane.The 'imported' isolates were from south-east Asian countries.

There were more isolates (41) resistant to penicillin by chromosomal mechanisms - so called CMRNG - than there were PPNG. CMRNG were detected most often in Sydney (28 isolates, 23% of strains there) and Melbourne (9 strains, 15%) but were also present in Perth, Brisbane and the Northern Territory in small numbers. There were 40 PPNG and 61 CMRNG in the June quarter of 1995 and 28 PPNG and 54 CMRNG in the September quarter of 1994.

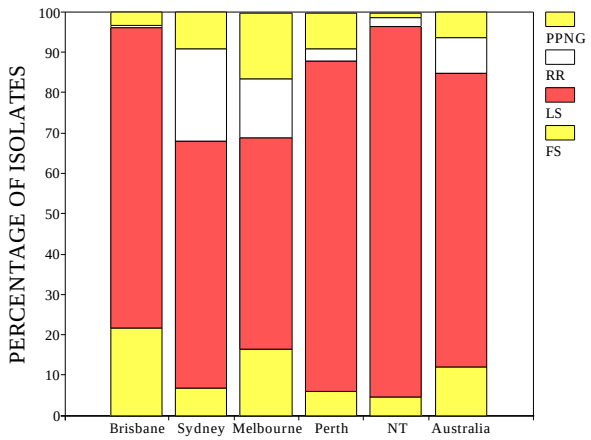
Ceftriaxone and spectinomycin

All 467 strains from all parts of Australia were sensitive to these injectable agents.

Although there has been some decrease in susceptibility of gonococci to ceftriaxone, no documented case of treatment failure has yet been reported. The significant activity of ceftriaxone against gonococci makes it the preferred cephalosporin for use in gonorrhoea.

Spectinomycin resistant strains were seen infrequently and sporadically in Australia in the 1980s. Only one spectinomycin-resistant isolate has been seen in recent years.

Figure 8. Penicillin susceptiblity of gonococcal isolates throughout Australia and by region, 1 July to 30 September 1995



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

Quinolone antibiotics

In this quarter, 17 isolates throughout Australia (3.6% of all strains) displayed altered quinolone sensitivity (QRNG, MICs ≥ 0.06 mg/L). These were detected in Melbourne (8 isolates - 13%), Sydney (5 isolates - 4%), Brisbane (3 isolates - 2%) and in a single strain in Perth. Strains with high level quinolone resistance (MICs ≥ 1 mg/L) were detected only in Sydney (5) and Melbourne (3).

In the previous quarter, 29 QRNG were detected, with 13 of these having high level resistance. In the past twelve months more QRNG with higher MICs have appeared in more centres.

Patients were infected with QRNG in China, Indonesia, Hong Kong and Japan and the Philippines before returning to Australia, but additionally a few locally acquired infections with QRNG were recorded. Quinolone-resistant gonococci are being isolated in increasingly high numbers in countries close to Australia so that consideration should be given to using alternative regimens for patients entering or returning to Australia from these areas.

High level tetracycline resistance (TRNG)

Twenty-five TRNG were detected in this quarter, 9 in Sydney (7.5% of strains there), 6 in Melbourne (9.8%), 4 in Perth (12%) and three each in Brisbane and the Northern Territory. This is a decrease on the 39 TRNG seen in the June quarter and approximates the 20 isolates of this type seen in the September quarter of 1994. Infections with TRNG were acquired overseas in Indonesia, Thailand, Singapore and, increasingly, through local contact.

LabDOSS

LabDOSS is a passive surveillance scheme that reports on significant bacterial and fungal isolates from normally sterile sites. Twenty laboratories currently forward reports of sterile site isolates to the Department of Health and Family Services. LabDOSS is published in alternate issues of CDI. Data from the LabDOSS scheme should be interpreted with caution. There is a potential for geographical, testing and referral pattern biases. In addition, risk factors and clinical information are not consistently provided by laboratories. For further information, see CDI 1996;20:9-10.

Data for this four weekly period have been provided by 6 laboratories. There were 237 reports of significant sepsis:

New South Wales: Royal North Shore Hospital 41.
Tasmania: Royal Hobart Hospital 31.
Queensland: Ipswich General Hospital 46; Sullivan and Nicholaides and Partners 66.
Western Australia: Sir Charles Gairdner Hospital 36.
Western Australia: Princess Margaret Hospital for Children 17.

Blood isolates

Organisms reported 5 or more times from blood are detailed in Table 7. Other blood isolates not included in Table 7 were:

Gram-positive: 2 *Bacillus* species, 2 *Corynebacterium* species, 3 *Enterococcus faecalis,* 2 *Enterococcus* species, 1 *Listeria monocytogenes,* 3 *Streptococcus* Group A, 3 *Streptococcus* Group B, 2 *Streptococcus* Group G, 2 *Streptococcus* 'milleri', 2 *Streptococcus sanguis* and 2 *Streptococcus* species.

Gram-negative: 1 *Acinetobacter* species, 1 *Aeromonas* species, *1 Brucella suis,* 1 *Capnocytophaga canimorsus, 1 Citrobacter freundii,* 2 *Enterobacter aerogenes,* 2 *Enterobacter cloacae,* 1 *Enterobacter* species, 1 *Enterobacter amnigenus,* 1 *Flavimonus oryzihabitans,* 4 *Haemophilus influenzae,* 1 *Haemophilus parainfluenzae,* 2 *Klebsiella oxytoca,* 1 *Morganella morganii,* 4 *Proteus mirabilis,* 2 *Serratia marcescens,* 1 *Serratia* species and 1 *Xanthomonas maltophilia.*

Anaerobes: 2 *Bacteroides fragilis,* 2 *Clostridium perfringens,* and 2 *Propionibacterium* species.

Fungi: 1 *Candida albicans* and 1 *Candida* species.

There were 176 (79% of total) blood isolates reported for patients over the age of 34 years (Figure 9).

Isolates from sites other than blood

CSF: Twelve reports of isolates from CSF or causing meningitis were received involving 1 *Cryptococcus neoformans,* 1 *Listeria monocytogenes,* 6 *Neisseria meningitidis,* 2 *Staphylococcus aureus,* 1 *Staphylococcus epidermidis* and 1 *Streptococcus pneumoniae.*

Joint fluid: Two reports from joint fluid were received involving 1 *Staphylococcus aureus* and 1 *Streptococcus* Group G.

Other: One report of an isolate from another sterile tissue was received involving *Klebsiella oxytoca.*

The final LabDOSS report

The surveillance scheme for organisms from normally sterile sites, LabDOSS, was evaluated recently as part of a review of national surveillance activities. The evaluation highlighted the limited extent to which LabDOSS has fulfilled an effective public health function.

The evaluation showed that data from the LabDOSS scheme was predominantly used by contributing laboratories to examine local trends within individual institutions but it was not used to develop policy or change practices. There was minimal use of LabDOSS data on a State or national basis. In general, the scheme failed to fulfil its defined objectives of: improving the understanding of the epidemiology of disease caused by invasive organisms; monitoring national trends of invasive disease; identifying emerging pathogens; guiding direction for further research; and developing and evaluating public policy based on the surveillance information. As a result of the evaluation it has been decided to discontinue LabDOSS. This is therefore the final report for LabDOSS in *CDI*.

We would like to thank contributors for their input to the LabDOSS scheme.

Figure 9. LabDOSS reports of blood isolates, by age group

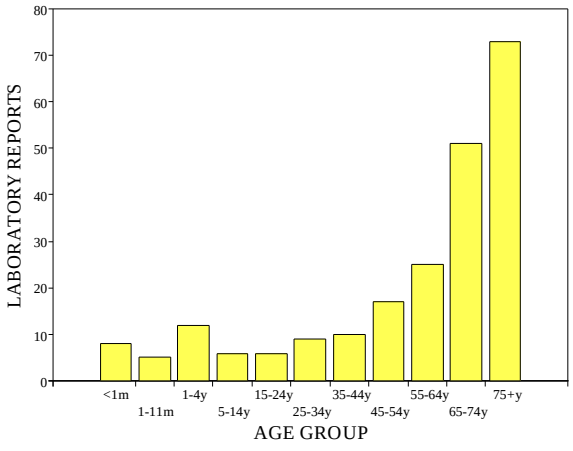


Table 7. LabDOSS reports of blood isolates, by organism and clinical information

Organism	Clinical information						Risk factors					Total[1]
	Bone/Joint	Lower respiratory	Endocarditis	Gastrointestinal	Urinary tract	Skin	Surgery	Immunosuppressed	IV line	Hospital acquired	Neonatal	
Escherichia coli					6		3	6	2	3		38
Klebsiella pneumoniae					1		2	3	1	2		12
Pseudomonas aeruginosa					1	1	3	3		2		10
Staphylococcus aureus				5	2		6	11	7	14		38[2]
Staphylococcus coagulase negative			1		1		1	8	4	1		44[3]
Streptococcus pneumoniae								5		1		20

1. Only organisms with 5 or more reports are included in this table.
2. MRSA 8.
3. Includes *Staphylococcus epidermidis*.

LabVISE

The Virology and Serology Reporting Scheme, LabVISE, is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence each fortnight. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1996;20:9-12.

There were 1,056 reports received in the *CDI* Virology and Serology Reporting Scheme this period (Tables 8 and 9).

Reports of **parvovirus** have increased over the last few months with 25 reports in July being the highest recorded since 1992 (Figure 10). In the last fortnight, 12 reports were received with one diagnosed by single high titre and the remainder by IgM detection.

Although data for August may be incomplete, reports of **respiratory syncytial virus** appear to have peaked (Figure 11). The total reports for July are marginally higher than the number reported in 1995. In the last fortnight, 294 reports were received. Diagnosis was by antigen detection (188), virus isolation (100), single high titre (4) and four-fold rise in titre (2). Most reports (272) continue to be for children under five years of age.

Figure 10. Parvovirus laboratory reports, 1992 to 1996, by month of specimen collection

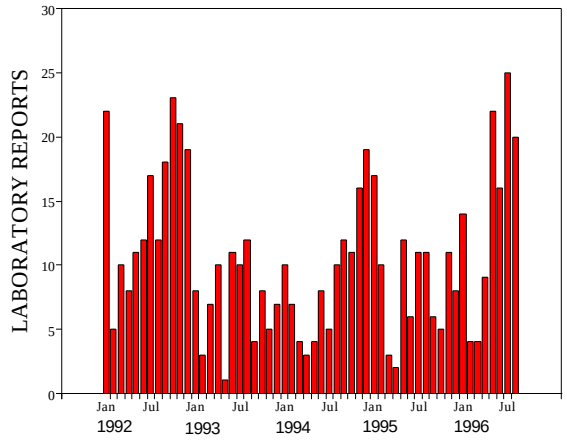


Figure 11. Respiratory syncytial virus laboratory reports, 1994 to 1996, by month of specimen collection

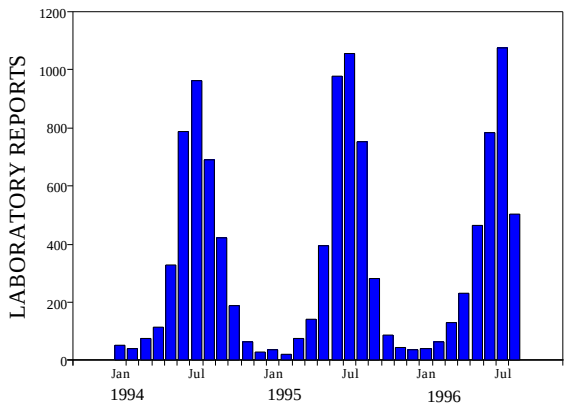


Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period 22 August to 4 September 1996, historical data[2], and total reports for the year

	State or Territory[1]								Total this fortnight	Historical data[2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA											
Mumps virus							1		1	3.0	30
Rubella virus				14			2		16	20.0	390
HEPATITIS VIRUSES											
Hepatitis A virus				10			2		12	15.7	325
Hepatitis D virus				1					1	1.0	11
ARBOVIRUSES											
Ross River virus			1	11					12	6.7	3,062
Barmah Forest virus				6					6	2.7	176
Stratford virus				1					1	.0	1
ADENOVIRUSES											
Adenovirus type 2							2		2	1.3	21
Adenovirus type 8					1				1	1.3	4
Adenovirus type 9							1		1	.0	1
Adenovirus not typed / pending		1		19	7	1	14	11	53	43.5	1,010
HERPES VIRUSES											
Cytomegalovirus		3		20	2	1	12	6	44	58.7	1,197
Varicella-zoster virus		2		15	4		14		35	35.0	896
Epstein-Barr virus		4		15			7	1	27	54.5	1,427
OTHER DNA VIRUSES											
Parvovirus	1			4			7		12	4.5	133
PICORNA VIRUS FAMILY											
Coxsackievirus B5							1		1	.3	3
Echovirus type 7							3		3	.0	10
Poliovirus type 2 (uncharacterised)						1			1	.5	14
Poliovirus type 2 (vaccine strain)						1			1	.0	2
Rhinovirus (all types)		2		9	1		16		28	39.3	531
Enterovirus not typed / pending		1		25			6		32	33.0	643
ORTHO / PARAMYXOVIRUSES											
Influenza A virus		16		65	33	1	22	4	141	83.8	1,251
Influenza A virus H3N2				14					14	6.3	64
Influenza B virus				2			1		3	27.3	40
Parainfluenza virus type 1				4	1		2		7	4.5	281
Parainfluenza virus type 2					2				2	1.7	59
Parainfluenza virus type 3				8	2		6	6	22	37.7	405
Parainfluenza virus typing pending						1		1	2	2.5	13
Respiratory syncytial virus		24		36	40	24	117	53	294	259.3	3,469
Paramyxovirus (unspecified)							1		1	.0	16
OTHER RNA VIRUSES											
Rotavirus		28			5	4	66	13	116	187.2	1,050
Calicivirus							1		1	.0	6

Table 8. Virology and serology laboratory reports by State or Territory[1] for the reporting period 22 August to 4 September 1996, historical data[2], and total reports for the year, continued

	State or Territory[1]								Total this fortnight	Historical data[2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
OTHER											
Chlamydia trachomatis not typed		4	6	25	19	4	3	2	68	90.7	2,744
Chlamydia psittaci							3		3	3.7	71
Chlamydia species				2					2	1.2	66
Mycoplasma pneumoniae		19		7			11		37	21.7	508
Coxiella burnetii (Q fever)		2					1		3	4.5	138
Rickettsia australis			1				1		2	1.0	17
Rickettsia tsutsugamushi				1					1	.7	9
Bordetella pertussis							16		16	19.3	335
Bordetella species				11					11	7.5	215
Leptospira canicola				1					1	.0	2
Leptospira pomona				1	2				3	.0	4
Leptospira hardjo		1		2					3	.0	17
Leptospira australis				2					2	.0	7
Leptospira species				7					7	.2	51
Schistosoma species		1					3		4	5.0	208
TOTAL	1	108	8	338	119	38	342	97	1,056	1,086.7	20,933

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

Table 9. Virology and serology laboratory reports by contributing laboratories for the reporting period 22 August to 4 September 1996

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	35
	Royal Prince Alfred Hospital, Camperdown	2
	South West Area Pathology Service, Liverpool	57
Queensland	Queensland Medical Laboratory, West End	159
	State Health Laboratory, Brisbane	203
South Australia	Institute of Medical and Veterinary Science, Adelaide	116
Tasmania	Royal Hobart Hospital, Hobart	43
Victoria	Microbiological Diagnostic Unit, University of Melbourne	3
	Monash Medical Centre, Melbourne	35
	Royal Children's Hospital, Melbourne	204
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	103
Western Australia	Princess Margaret Hospital, Perth	96
TOTAL		1056