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Communicable Diseases Intelligence

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<u>VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME</u>: A total of 1,542 reports were processed during this period.

Twenty-five cases of Q fever (18 males, 6 females, 1 gender not stated) were reported during this period. Ages ranged from 12 to 88 years. Patients included 3 abbatoir workers and 1 shearer. One patient, a 12-year-old female, reported contact with two Argentinians with Q fever.

Five reports of influenza B and two reports of influenza A (subtype not stated) were received during this period.

Of the 184 Ross River virus identifications reported during this period, 160 originated from the State Health Laboratory, Brisbane. Most of these samples (108/160) were collected in February 1989.

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LEGIONELLA IN SOUTH AUSTRALIA

(Contributed by Dr TW Steele, Institute of Medical and Veterinary Science, Adelaide, SA)

Over the last 10 years, the Institute of Medical and Veterinary Science (IMVS) has provided laboratory services for the diagnosis of legionellosis in South Australia. In this time only two outbreaks have been documented. The first involving *Legionella pneumophila* serogroup 1 occurred in 1986 and the second due to *L. longbeachae* serogroup 1. In both outbreaks some twenty persons were infected. A common source for the *L. pneumophila* outbreak was found but the cases with *L. longbeachae* infections occurred across the state though most were found in the southern Adelaide suburbs. The common epidemiological factor in those with *L. longbeachae* was gardening. Following investigations this organism was isolated in March 1989 from a potting mix made in South Australia. It is not known if soil was the source of infection for the majority of patients since a wide variety of potting mixes and other gardening aids were used by patients. In addition not all victims of *L. longbeachae* infection were gardeners.

The diagnosis of legionellosis has been confirmed or strongly suspected on the basis of laboratory tests in 119 patients in South Australia since 1978 (Table 1). During this time, IMVS has isolated many other *Legionella* species from environmental samples but only *L. pneumophila* and *L. longbeachae* have been isolated from patients with pneumonia.

Table 1: Diagnosis of legionellosis in South Australia, 1978-1989

	Definite		Presumptive
	Legionella Isolated	Seroconversion Alone	Single Test Antibody ≥1/256
L. pneumophila			
serogroup 1	20	45	17
serogroup 2	2	2	2
L. longbeachae	11	12	8

The infection caused by *L. pneumophila* and *L. longbeachae* was similar with patients presenting with fever, cough (often with little sputum) and (in more severe cases) breathlessness and confusion. *L. longbeachae* has been found more commonly in elderly patients who were non-smokers. Only two of the cases of *L. longbeachae* infection were in patients younger than 60 years. One was a heavy smoker and the other had been a cigarette smoker until 6 months before his illness.

Illness may be severe and fatal with both *L. longbeachae* and *L. pneumophila* and a high mortality rate (approximately 60%) occurs in patients admitted to hospital with renal and respiratory failure. Seriously ill patients who survived the early infective illness often died of complications of respiratory failure at a time when *Legionella* had disappeared from the lung.

Diagnosis

Legionellosis should be included in the differential diagnosis of any patient with evidence of pneumonia who fails to respond to penicillin treatment. If the patient is coughing up sputum this should be examined for legionellae by direct fluorescence microscopy and by culture. Examinations of acute and convalescent serum samples for antibodies to *Legionella* and to other respiratory pathogens such as mycoplasma and viruses is worthwhile and should be done in all who are ill enough to require hospital treatment. Most patients with Legionella infection seroconvert by the 11th day of illness but the antibody response may be delayed for several weeks. In our series, on only one occasion has a culture positive patient not shown seroconversion 6 weeks after the infection. All culture confirmed cases of *L. pneumophila* formed predominantly IgM antibody early in the illness whereas those with *L. longbeachae* seldom formed this class of antibody in response to their infection.

Diagnosis can be made:

- rapidly with fluorescent antibody smears within hours (this is only performed in a limited number of centres);
- with serological testing, by demonstrating a 4-fold or greater rise in antibody titre in paired serum specimens assayed by indirect immunofluorescence or other methods (if a rise in antibody titre is not present after 11 days of illness and legionella is strongly suspected this can be repeated at 3-4 weeks);
- demonstration of antigen in pleural fluid or urine using RIA or ELISA;
- specifically by culturing legionella from respiratory secretions or lung tissue using direct immunofluorescence; we have found colonies are usually visible within 7 days.

Special tests for epidemiological investigations

IMVS has developed considerable experience with *Legionella* in the last 6 years through clinical and environmental investigations. The lack of suitable serological reagents to identify the many species found in the environment led IMVS to develop latex agglutinating reagents, some of which are now commercially available (Legionella Serobact, Disposable Products). When conventional serological tests on isolates fail to lead to species identification, high performance liquid chromatography and gas liquid chromatography can be used to confirm that these isolates are *Legionella* species. DNA studies using dot blot and quantitative hybridisation are used to identify unusual isolates. More recently IMVS has developed DNA fingerprinting methods using restricted fragment polymorphism analysis to subtype *L. pneumophila* serogroup 1 strains isolated from patients and environmental samples suspected of being the source of the outbreak. These techniques were used to examine isolates from the recent Tasmania outbreak. With scientific colleagues at the Evolutionary Biological Unit of the South Australian Museum IMVS has compared these methods to allo-enzyme analysis to assess their value in subtyping *L. pneumophila* serogroup 1 strains as an aid to epidemiological investigations.

AN UNUSUAL SECONDARY CASE OF INVASIVE *HAEMOPHILUS INFLUENZAE* DISEASE

(Contributed by: Dr J Hanna, Medical Officer, Communicable Diseases Control Centre, Alice Springs; Dr D Brookes, Paediatric Registrar, Alice Springs Hospital; and Dr B Conlon, District Medical Officer, Barkly Region, Northern Territory)

The attack rate of invasive *Haemophilus influenzae* infections among the Aboriginal children of Central Australia is extremely high; prospective surveillance since mid-1985 indicates an annual point estimate of approximately 980 cases of invasive *H. influenzae* disease (all diagnostic categories) per 100 000 Aboriginal children under 5 years of age.

Secondary cases (ie. invasive *H. influenzae* infections more than 24 hours but less than 30 days after the onset of disease in the index patient) have been well described in household contacts (usually defined as those who spent four or more hours a day with index patient in the week before illness) of children with invasive *H. influenzae* infections, particularly meningitis. The secondary attack rate in the United States for all ages is about 0.3%; this represents approximately a 600 fold increase in risk, compared with the risk in the general population [1]. The risk is markedly age-dependent, highest for children younger than 2 years (4.4%) and negligible for contacts older than 5 years [1]. Seventy-five percent of secondary cases occur within one week of onset of symptoms in the index case [2].

In 1985 the National Health and Medical Research Council recommended rifampicin prophylaxis for all households in which a case of invasive *H. influenzae* type b disease has occurred, and in which another child under four years of age resides [3]. This recommendation is not routinely implemented in Central Australia because of: the difficulty with defining close household contacts in large extended families; the extreme mobility of Aboriginal people; and the difficulties many have with compliance with treatment regimens. Moreover in the first 3½ years of surveillance no secondary cases of invasive *H. influenzae* disease were evident. However recently two children (from the same community) with invasive *H. influenzae* disease were seen at the Alice Springs Hospital. Since there was a week between the onset of symptoms in the two children prophylactic doses of rifampicin were distributed to household contacts.

Case 1

A 7½-month-old Aboriginal child presented with an acutely swollen painful leg; she was initially thought to have tibial osteomyelitis and was treated with intravenous penicillin and flucloxacillin. These antibiotics were replaced by high dose ampicillin when ampicillin-sensitive *H. influenzae* type b was isolated upon blood culture. The swelling settled very slowly despite the ampicillin; X-rays on admission and 8 days later showed no evidence of periosteal changes (bone scan facilities are not available at Alice Springs Hospital). She was eventually discharged after 20 days in hospital; she was given a four day course of rifampicin (to eliminate any nasopharyngeal carriage of *H. influenzae*) prior to discharge.

Case 2

A 5-month-old Aboriginal boy presented with a swollen painful scrotum, seven days after the previous child had presented. He was commenced upon intravenous ampicillin with little initial improvement. At surgery, two days after admission, 2-3 ml of pus was released, the left testis was inflamed and the epididymis was swollen and inflamed.

A fully sensitive *H. influenzae* type b was isolated from the pus and from blood cultures; chloramphenicol was commenced before the sensitivities became available. His recovery was complicated by a mild chest infection; he was discharged from hospital after a course of rifampicin on the fourteenth day.

Epididymo-orchitis is rare in prepubertal boys. A review of 9 cases of epididymo-orchitis caused by *H. influenzae* type b has suggested that the infection is not secondary to a urinary tract abnormality or a urinary tract infection, but rather that the epididymo-orchitis is secondary to bacteraemia [4], as is *H. influenzae* type b meningitis [5].

The two children were cousins; both lived in a remote Central Australian community which has a population of about 80 people. An Aboriginal Health Worker informed us that although they lived in different dwellings the children were often in each others company. The families were visited and informed of the possibility of further cases occurring; seven close household contacts of the two children, including two other young children were identified. Prophylactic courses of rifampicin were dispensed to the identified contacts; to date no further cases of *H. influenzae* have occurred in the community.

Although the incidence of invasive *H. influenzae* infections in Aboriginal children in Central Australia is very high, rifampicin prophylaxis can only have a minimal impact on reducing the incidence since secondary cases only constitute an estimated 1-2% of the total number of cases [6].

Conjugate *H. influenzae* type b vaccines that are immunogenic and effective in infancy are being developed and trialled [7]; such vaccines may eventually be able to reduce the incidence of invasive *H. influenzae* disease in Aboriginal children.

REFERENCES

1. Prevention of secondary cases of Haemophilus influenzae type b disease. MMWR 1982;31:672-4,679-80.
2. Band JD, Fraser DW, Ajello G. Prevention of Haemophilus influenzae type b disease. JAMA 1984;251:2381-6.
3. Treatment of Haemophilus influenzae type b. National Health and Medical Research Council, 100th Session , November 1985, p 29.
4. Greenfield SP. Type B Haemophilus influenzae epididymo-orchitis in the prepubertal boy. J Urol 1986;136:1311-3.
5. Smith AL. Pathogenesis of Haemophilus influenzae meningitis. Pediatr Infect Dis J 1987;6:783-6.
6. Gilbert GL. Epidemiology and prevention of invasive Haemophilus influenzae type b infection. Aust Paediatr J 1987;23:323-7.
7. Weinberg GA, Granoff DM. Polysaccharide-protein conjugate vaccines for the prevention of Haemophilus influenzae type b disease. J Paediatr 1988;113:621-31.

NEONATAL CROSS-INFECTION FROM *LISTERIA MONOCYTOGENES*
(Based on CDR 89/16, 21 April 1989)

Introduction

Although current interest in *Listeria monocytogenes* infection focuses heavily on its transmission by food [1], other modes of transmission, such as neonatal hospital acquired infection caused by *L. monocytogenes* are well documented in Britain [2-7].

From cultures sent to the Division of Microbiological Reagents and Quality Control, Colindale, between 1971 and 1988, 18 pairs of cases of listeriosis occurred where neonatal cross-infection was considered likely, and these episodes showed a common pattern:

- Firstly, an infant was born with congenital listeriosis, with onset within one day of birth (early onset). Then, in the same hospital, and within 24 hours, an apparently healthy neonate was born who subsequently developed late onset listeriosis between the fifth and twelfth day post partum. *L. monocytogenes* was isolated from both infants in each episode.

- Of the 36 neonates, isolates were from:
  - blood and/or CSF in 29,
  - surface sites only in two, and
  - sites unknown in the remaining five.

- The organism was also isolated from eight of the mothers of early onset cases:
  - six from high vaginal swabs (HVS),
  - one from HVS and breast fluid, and
  - one from an unspecified site.

- *L. monocytogenes* was not cultured from any of the mothers of the late onset neonatal cases.

- In each episode the isolates from both infants and, where available, the mother of the early onset case, were indistinguishable using typing based on serology, lytic phages and DNA restriction fragment analysis.

- In 13 episodes the cases were either delivered or nursed in the same or adjacent rooms and consequently staffed and equipment were common to each paired case.

- In one episode, the mother of the early onset case was in an open ward and handled a neonate from a adjacent bed who acquired late onset listeriosis [3].

- No further epidemiological information was available in the four other episodes.

An illustrative case report follows:

Case report

A full-term baby born after a normal vaginal delivery was noted to be ill shortly after birth. *L. monocytogenes* was isolated from blood cultures and from nose and throat swabs. The baby's

condition improved on ampicillin and gentamicin and he was discharged home fully recovered after ten days. His mother had suffered a 'flu-like' illness a few days before going into labour and *L. monocytogenes* was isolated from an HVS taken 24 hours after delivery.

Six days after delivery of this baby, a second baby was admitted to the children's ward with clinical septicaemia and meningitis. *L. monocytogenes*, later shown to be of the same phage type as that from the first episode, was isolated from the CSF. He also made a full recovery on treatment with ampicillin and gentamicin. His mother had had an uneventful pregnancy and no listeria were isolated from an HVS taken eight days after delivery.

As neonatal listeriosis is a rare condition, investigations were made to assess whether there had been any cross-infection. The second baby had been born five hours after the first in an adjacent delivery room in the same suite. The mothers were attended by different midwives, different teams of doctors and were delivered during different duty periods. A search for equipment common to both deliveries showed that the same Resuscitaire and baby weighing scales were probably used in each case. As far as could be ascertained some days after the event, the same Resuscitaire may have been present in each room at the time of delivery. Nevertheless it was used only for one baby, and any contamination that may have occurred would have been small because only the attached suction apparatus was used and the baby remained on the bed. It was normal policy for the apparatus to be thoroughly cleaned after each delivery, whether it was used or not.

The scale pans used for weighing babies were washed and lined with fresh paper after each delivery so contamination through this route, though possible, was unlikely. The trolley on which the scales were carried also carried jars of labels, wrist bands, alcohol swabs and a rectal thermometer. Midwives commented that meconium sets on thermometers 'like concrete', making them very difficult to clean properly. It is possible that a thermometer may have been the vehicle of transmission, as has been reported with salmonella [8]. It is unlikely that the jars of labels or wrist bands could have become sufficiently contaminated by a midwife's finger for a subsequent dip into the same jar to receive an infecting dose.

Following the episode, procedures were changed so that as little equipment as possible is shared between delivery rooms. In addition, the use of plastic single use sleeves was introduced for the rectal thermometers.

Comment

During intra-uterine infection, amniotic fluid may be heavily contaminated with *L. monocytogenes* (10^8 CFU/ml)[9]. At birth, neonates and mothers of congenitally infected infants are also likely to be heavily contaminated [10], as will be staff hands, clothing and any equipment used. In addition, obstetric

complications and the birth of a sickly infant will necessitate the use of a variety of standard and emergency equipment which could act as vehicle for cross-contamination. Such items could include:

- infant rectal thermometers,
- baby scales,
- resuscitation apparatus,
- intubation forceps,
- laryngoscope blades,
- suckers and tubing.

Such equipment should, if practicable, be heat sterilised or single use. If this is not possible, equipment should be heat disinfected by boiling or thoroughly cleaned and immersed in a suitable chemical disinfectant such as 70% alcohol [11]. A single use barrier and alcohol wipes may be used on items such as the baby scale pan. Staff should wear gloves and aprons which should be changed and wash hands before attending another patient.

The occurrence of single (as described here) rather than multiple associated cases, suggests that a large inoculum shortly after birth may be needed to transmit infection. However cross-infection by *L. monocytogenes* from one neonate to four others has been reported, when a common rectal thermometer and staff hands were implicated in transmission [12].

In a series of 168 neonatal listeriosis cases that occurred in Britain between 1967 and 1985 [7], one quarter of the late onset cases were due to cross-infection, and for every ten early onset cases a late onset case resulted. Although listeriosis is a fairly rare disease, a recent increase in incidence has been noted [13], with one case per 37,000 births in 1978 [14] compared to one case per 9,700 births in 1987 [15]. This suggested that cases will be encountered more often, and appropriate infection control measures should be instigated to prevent neonatal hospital acquired infection.

REFERENCES

1. Listeriosis. Lancet 1989;1:84-4.
2. Campbell AN, Sill PR, Wardle JK. Listeria meningitis acquired by cross-infection in a delivery suite. Lancet 1981;2:752-3.
3. Isaacs D, Liberman MM. Babies cross-infected with Listeria monocytogenes. Lancet 1981;2:940.
4. Sinha SK, Jones D, Audurier A, Taylor AG. Perinatal listeriosis and hospital cross-infection. Arch Dis Child 1983;58:938-40.
5. Simmons MD, Cockcroft PM, Okubadejo OA. Neonatal listeriosis due to cross-infection in an obstetric theatre. J Infect 1986;13:235-9.
6. McLauchlin J, Audurier A, Taylor AG. Aspects of the epidemiology of human Listeria monocytogenes infections in Britain 1967-1984; the use of serotyping and phage typing. J Med Microbiol 1986;22:367-77.
7. McLauchlin J. Listeria monocytogenes, recent advances in the taxonomy and epidemiology of listeriosis in humans. J Appl Bacteriol 1987;63:1-11.
8. McAllister TA, Roud JA, Marshall A, et al. Outbreak of Salmonella eimsbuettel in newborn infants spread by rectal thermometers. Lancet 1986;1:1262-4.
9. Courcol RJ, Roussel-Delvallez M, Puech F, et al. Quantitative bacteriological analysis of amniotic fluid. Biol Neonate 1982;42:166-73.
10. Seeliger HPR, Finger H. Listeriosis. In: Remington JS, Klein JO, eds. Infectious diseases of the fetus and newborn infant, 2nd edition. Philadelphia, Sanders 1983:264-89.
11. Hoffman PN, Cooke EM, Larkin DP et al. Control of infection in general practice: a survey and recommendations. Br Med J (Clin Res) 1988;297:34-6.
12. Larsson S, Cederberg A, Ivarsson S, et al. Listeria monocytogenes causing hospital-acquired enterocolitis and meningitis in newborn infants. Br Med J (Clin Res) 1978; 2:473-4.

13. Hall SM, Crofts N, Gilbert RJ, et al. Epidemiology of listeriosis, England and Wales. Lancet 1988;2:502-3.
14. Perinatal listeriosis. Lancet 1980;1:911.
15. McLauchlin J, Saunders NA, Ridley AM, Taylor AG. Listeriosis and food-borne transmission. Lancet 1988;1:177-8.

ERRATA: CDI 89/10

Tuberculosis and human immunodeficiency virus infection: Recommendations of the U.S. Advisory Committee for the Elimination of Tuberculosis (ACET)

Page 5, 5th-7th line: Should read - tuberculin reactions of 5mm induration or more should be considered indicative of tuberculosis infection in an HIV-infected person.

Page 10, second paragraph, 4th-7th line: Should read - Isoniazid preventive therapy should also be recommended for all other IVDUs with a tuberculin reaction of 10mm induration or more regardless of age.

NOTICE TO SCIENTISTS WORKING IN THE FIELD OF LISTERIOSIS

The Bacterial Ecology Unit of the Pasteur Institute (Paris) is seeking to extend its worldwide network of contacts in the following aspects of listeriosis: clinical (human and veterinary), food, environment and epidemiology.

If you are working in this field, or know of anyone working on listeriosis, you are invited to get in touch with Dr Joyce Rocourt, Unité d'Ecologie Bactérienne, Institut Pasteur, 28, Rue du Dr Roux, F-75724 Paris Cedex 15, France.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 8/6/89 TO 21/6/89

1. CODE 019 - FAIRFIELD(VIC)
2. CODE 065 - STATE LAB(WA) PMH(WA)
3. CODE 110 - IMVS(SA)
4. CODE 111 - RCH(VIC)
5. CODE 112 - ICPMR(NSW) WVH(ACT)
6. CODE 113 - PHH POW(NSW)
7. CODE 114 - RAHC(NSW)
8. CODE 115 - STATE LAB(QLD)

		019	065	110	111	112	113	114	115	TOTAL
0100	ADENOVIRUS NOT TYPED	2	1	2	0	2	1	0	7	15
0101	ADENOVIRUS TYPE 1	0	0	1	0	0	0	0	0	1
0102	ADENOVIRUS TYPE 2	0	0	2	0	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	0	0	1	0	4	0	0	0	5
0105	ADENOVIRUS TYPE 5	0	0	1	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	1	1	0	0	2
0119	ADENOVIRUS TYPE 19	0	0	0	0	1	0	0	0	1
0126	ADENOVIRUS TYPE 26	1	0	0	0	0	0	0	0	1
0127	ADENOVIRUS TYPE 27	0	0	0	0	1	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	11	0	2	2	0	15
0201	INFLUENZA A VIRUS	0	0	2	0	0	0	0	0	2
0203	INFLUENZA B VIRUS	0	2	3	0	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	1	0	0	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	1	7	7	5	0	0	6	26
0303	PARAINFLUENZA VIRUS TYPE 3	1	0	0	3	0	0	0	5	9
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	0	0	3	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	6	23	91	35	30	14	24	92	315
0500	RHINOVIRUS (ALL TYPES)	1	1	9	6	5	1	0	2	25
0600	MYCOPLASMA PNEUMONIAE	2	3	7	3	5	1	0	0	21
0700	ORNITHOSIS-PSITTACOSIS	3	0	2	0	0	0	0	0	5
0809	COXSACKIEVIRUS A9	0	0	0	0	1	0	0	0	1
0903	COXSACKIEVIRUS B3	1	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	1	0	0	0	0	0	0	1
1009	ECHOVIRUS TYPE 9	1	0	0	0	0	0	0	0	1
1013	ECHOVIRUS TYPE 13	0	0	0	0	1	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	1	0	0	1
1019	ECHOVIRUS TYPE 19	0	1	0	0	1	0	0	0	2
1022	ECHOVIRUS TYPE 22	0	0	1	0	0	1	0	0	2
1030	ECHOVIRUS TYPE 30	2	5	0	0	3	0	0	0	10
1101	POLIOVIRUS TYPE 1	0	0	0	0	1	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	2	0	0	0	2
1200	MUMPS VIRUS	0	0	0	0	3	0	0	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	1	0	0	0	0	5	7
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	0	0	73	0	0	1	74
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	2	6	8	0	0	1	0	20	37
1303	VARICELLA-ZOSTER VIRUS	5	1	1	0	6	2	0	2	17
1306	HERPES SIMPLEX TYPE 1	31	36	17	0	3	10	0	22	119
1307	HERPES SIMPLEX TYPE 2	59	53	16	0	18	26	0	40	212
1399	HERPES VIRUS TYPING PENDING	1	0	0	8	0	0	0	0	9
1401	COXIELLA BURNETI	5	0	0	0	8	1	0	11	25
1502	PICORNIA VIRUS - NOT TYPED = E	0	2	0	0	0	5	0	13	20
1522	RUBELLA VIRUS	1	0	1	0	0	0	0	11	13
1532	HEPATITIS B ANTIGEN	0	15	11	0	31	6	1	19	83
1535	HEPATITIS A ANTIBODY	0	0	4	0	0	0	0	0	4
1541	CHLAMYDIA A - C. TRACHOMATIS	27	26	32	0	28	0	0	23	136
1555	PAPOVAVIRUS GROUP (PAPILLOMA -	1	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	28	4	4	7	0	6	2	18	69
1564	ROTAVIRUS	0	5	1	0	2	3	0	0	11
1599	ENTEROVIRUS TYPING PENDING	0	0	0	9	0	10	1	0	20
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	0	0	0	0	0	0	0	15	15
9990	AUSTRALIAN ENCEPHALITIS	0	0	0	0	0	0	0	1	1
9992	ROSS RIVER VIRUS	7	12	4	0	0	1	0	160	184
9995	DENGUE	0	0	0	0	0	0	0	2	2
9997	KUNJIN VIRUS	0	0	0	0	0	0	0	1	1
TOTAL		188	198	229	90	235	93	30	479	1542

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 8/6/89 TO 21/6/89

1. CODE 00, 99 .... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 .... - ENCEPHALITIS
4. CODE M3 .... - MENINGITIS
5. CODE 04 .... - PARALYSIS
6. CODE 05, 13 .... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	8	0	0	0	0	6	0	0	0	0	14
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
0102	ADENOVIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	2	0	0	0	0	3
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	1	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	0	0	0	0	0	0	0	1	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	12	0	0	0	0	2	0	0	0	1	15
0201	INFLUENZA A VIRUS	0	2	0	0	0	0	0	0	0	0	0	2
0203	INFLUENZA B VIRUS	0	4	0	0	0	1	0	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	1	23	0	0	0	0	0	0	0	0	0	24
0303	PARAINFLUENZA VIRUS TYPE 3	0	9	0	0	0	0	0	0	0	0	0	9
0399	PARAINFLUENZA VIRUS TYPING PEN	0	3	0	0	0	0	0	0	0	0	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	6	304	0	0	0	1	1	0	0	0	0	312
0500	RHINOVIRUS (ALL TYPES)	1	24	0	0	0	0	0	0	0	0	0	25
0600	MYCOPLASMA PNEUMONIAE	6	14	0	0	0	0	0	0	0	0	0	20
0700	ORNITHOSIS-PSITTACOSIS	0	5	0	0	0	0	0	0	0	0	0	5
0809	COXSACKIEVIRUS A9	0	0	0	1	0	0	0	0	0	0	0	1
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	0	0	0	0	0	1	0	0	0	0	1
1013	ECHOVIRUS TYPE 13	1	0	0	0	0	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	1	0	0	0	0	0	0	0	1
1019	ECHOVIRUS TYPE 19	1	1	0	0	0	0	0	0	0	0	0	2
1022	ECHOVIRUS TYPE 22	0	2	0	0	0	0	0	0	0	0	0	2
1030	ECHOVIRUS TYPE 30	1	1	1	4	0	0	2	0	0	0	0	9
1102	POLIOVIRUS TYPE 2	1	1	0	0	0	0	0	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	0	5	5
1301	HERPES SIMPLEX VIRUS - NOT TYP	22	1	0	0	0	0	0	0	0	0	13	36
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	6	5	0	1	0	0	0	1	0	0	1	14
1303	VARICELLA-ZOSTER VIRUS	2	1	0	0	1	0	0	0	0	0	11	15
1306	HERPES SIMPLEX TYPE 1	5	9	0	0	0	0	0	0	0	0	64	78
1307	HERPES SIMPLEX TYPE 2	10	0	0	0	0	0	0	0	0	0	51	61
1399	HERPES VIRUS TYPING PENDING	0	2	0	0	0	0	0	0	0	0	6	8
1401	COXIELLA BURNETI	2	1	0	0	0	0	1	0	0	0	2	6
1502	PICORNIA VIRUS - NOT TYPED = E	0	5	0	0	0	2	10	0	0	0	1	18
1522	RUBELLA VIRUS	2	0	0	0	0	0	0	0	0	0	9	11
1532	HEPATITIS B ANTIGEN	30	0	0	0	0	0	0	42	0	0	0	72
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	3	0	0	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	39	1	0	0	0	0	0	0	0	0	0	40
1556	CMV - CYTOMEGALOVIRUS	1	22	1	2	0	1	0	1	1	3	4	36
1564	ROTAVIRUS	1	0	0	0	0	0	10	0	0	0	0	11
1599	ENTEROVIRUS TYPING PENDING	0	3	0	4	0	0	9	0	0	0	1	17
9901	ARBOVIRUS GROUP A.(UNSPECIFIED	5	2	0	0	0	0	0	0	0	0	2	9
9990	AUSTRALIAN ENCEPHALITIS	1	0	0	0	0	0	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	96	2	1	0	0	0	0	0	0	0	7	106
9995	DENGUE	0	0	0	0	0	0	0	0	0	0	1	1
9997	KUNJIN VIRUS	0	0	0	1	0	0	0	0	0	0	0	1
TOTAL		242	473	3	14	1	5	45	47	2	3	179	1014

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 8/6/89 TO 21/6/89

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	0	0	0	0	0	0	1	0	1
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	0	0	0	0	0	2
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	0	1	0	0	1
0119	ADENOVIRUS TYPE 19	1	0	0	0	0	0	0	0	0	0	1
0127	ADENOVIRUS TYPE 27	0	0	0	0	0	0	0	0	1	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	0	0	0	1	1	0	2
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	1	0	0	2	0	3
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	0	0	1	0	1
1009	ECHOVIRUS TYPE 9	0	0	0	0	0	0	0	1	0	0	1
1030	ECHOVIRUS TYPE 30	0	0	0	0	0	0	0	0	1	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
1200	MUMPS VIRUS	0	0	1	0	0	0	0	0	1	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	2	0	0	0	0	0	0	0	0	2
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	36	0	0	0	0	0	1	1	0	38
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	10	4	2	0	0	4	3	0	23
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	0	2	0	2
1306	HERPES SIMPLEX TYPE 1	6	30	0	1	0	0	0	0	4	0	41
1307	HERPES SIMPLEX TYPE 2	0	137	0	0	0	0	0	0	14	0	151
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	1	0	1
1401	COXIELLA BURNETI	0	0	1	0	1	0	1	11	5	0	19
1502	PICORNIA VIRUS - NOT TYPED = E	0	1	0	0	0	0	0	0	0	1	2
1522	RUBELLA VIRUS	0	0	0	0	2	0	0	0	0	0	2
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	1	10	0	11
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	0	1	0	1
1541	CHLAMYDIA A - C. TRACHOMATIS	0	96	0	0	0	0	0	0	0	0	96
1555	PAPOVAVIRUS GROUP (PAPILLOMA -	0	0	0	0	0	0	0	0	1	0	1
1556	CMV - CYTOMEGALOVIRUS	0	1	0	4	2	2	0	6	18	0	33
1599	NTEROVIRUS TYPING PENDING	0	0	0	2	0	0	0	0	0	0	2
9901	BOVIRUS GROUP A.(UNSPECIFIED	0	0	0	0	6	0	0	0	0	0	6
9992	R SS RIVER VIRUS	0	0	0	0	69	0	0	2	7	0	78
9995	D NGUE	0	0	0	0	0	0	0	0	1	0	1
TOTAL		9	303	12	11	82	3	1	28	77	1	527