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Communicable Diseases Intelligence

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VIRUS REPORTING SCHEME: A total of 1 467 reports were processed for this period.

Eight cases of Q fever were reported (4 from Queensland, 3 from South Australia and 1 from New South Wales). Occupational exposure data were available for:-

. one South Australian case, a 23 year old male abattoir worker
. four Queensland cases; 3 meatworkers, one female aged 35 and two males aged 30 and 46 respectively, and one 43 year old male grazier.

None of these eight patients were involved in the Q fever vaccine field trial conducted in South Australia.

Herpes Simplex virus was isolated from the upper digestive tract (mouth, upper and mid-oesophagus) of a 2 year old female with acute lymphoblastic leukaemia (ALL). The virus was recovered ten days after a ten day course of acyclovir treatment for severe herpes stomatitis.

Rotavirus activity is still increasing with 159 cases of rotavirus associated diarrhoea reported for this period compared to 120 and 102 cases reported for the previous two periods. The demographic patterns of the cases remained unaltered. Rotavirus was also isolated from the faeces of a 2 year old male with meningitis. This virus has been reported to induce meningitis.

The expected increase in Respiratory Syncytial Virus (RSV) activity predicted in CDI 86/13 has now been evidenced by 200 cases reported for this period compared to 98, 81 and 45 cases reported for the previous three periods. It was noted that the virus was also isolated from the nasal aspirate of a 5 year old male with meningitis. RSV primarily causes upper respiratory tract infection, less often involves the lower respiratory tract and has not been reported to infect the central nervous system.

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Figures given may be subject to revision.

VIRAL MENINGITIS

(Based on Infectious Diseases Bulletin, Royal Children's Hospital, Melbourne, 22 June 1986)

During the March-May quarter of 1986 there was a significant epidemic of 'aseptic' meningitis among children presenting at the Royal Children's Hospital, Melbourne. There were 7, 7 and 26 cases respectively in each of the 3 months of the quarter. During that quarter the total of 40 previously healthy children whose CSF showed changes consistent with viral meningitis, included:

- 2 males aged 11 and 14 years with mumps meningitis (positive CSF culture in one and detection of specific IgM in serum in both)
- one 3 year old male with proven herpes simplex encephalitis
- one 8 year old male with presumed encephalitis with a short history of severe convulsions and rapid recovery following treatment with acyclovir
- one 12 year old male with an unusual meningitis presenting as severe headache and photophobia associated with a parietal lesion on CT Scan, high CSF protein and some unusual neurological signs.

The diagnosis in the last 2 patients remained unknown but was unlikely to have been enteroviral meningitis.

Apart from the 5 patients described above, 34 of the remaining 35 children with viral meningitis required admission to hospital. The demographics of the patients were:

SEX: Male, 20; Female, 15.
AGE: 0-3 months, 10; 3-24 months, 10; 2-11 years, 15.

Eight of these patients had received antibiotic therapy before admission, five of whom were initially treated with penicillin and chloramphenicol in hospital. Eleven of the remaining 27 patients who had had no previous therapy were treated with antibiotics, including one who also had acyclovir because of suspected herpes simplex encephalitis.

Sixteen of the above 35 patients were initially treated for suspected bacterial meningitis with only one patient receiving more than 3 days treatment. The CSF white cell counts are summarised in Table 1.

Table 1. CSF White cell counts in patients with enteroviral meningitis

CSF white cells ($\times 10^6$/L)	No. of patients	% polymorphs: >90	60-90	<60	Age of patients <2yrs	>2yrs
200-300	6	6	-	-		
100-199	12	7	3	2	13	5
<100	17	9	5	3	7	10
TOTAL	35	22	8	5	20	15

The majority of patients had a predominance of polymorphs. The highest total white cell count was 292 x 10^6/L. However there was no difference in white cell counts in patients who were treated with antibiotics compared with those who were not. The level of CSF protein was distributed as follow:

> 1 g/L, 1;
0.75 - 1 g/L, 3;
0.5 - 0.75 g/L, 9;
< 0.5 g/L, 3.

Younger patients generally had higher CSF white cell counts. Thirteen of the 20 children aged less than 2 years had counts greater than 100 x 10^6/L compared with 5 of the 15 children aged over 2 years. All CSF specimens, tested by latex agglutination for antigens of Haemophilus influenzae type b, Streptococcus pneumoniae and Neisseria meningitidis, were negative.

The duration of illness preceding hospital admission was up to 2 days in 21 patients (of whom 3 had antibiotic treatment) and more than 2 days in the other 14 patients (of whom 5 had been treated with antibiotics). Twenty two of these patients had an admission temperature greater than 38°C. Although several remained febrile for several days all patients improved rapidly and were discharged within 6 days with the exception of:

- two children aged 6 days and 20 months in whom herpes simplex infection was suspected; and
- one 7 week old child who also had an E.coli urinary tract infection and was thought to have early bacterial meningitis.

Enterovirus 71 was isolated from one or more specimens (faeces, urine, throat swabs and vesicular lesions) of 12 patients and echovirus 21 was isolated from the specimens of one patient. CSF specimens were obtained from 30 patients and cultured with no detectable enterovirus growth.

The isolation of enterovirus 71 in this instance has been linked to an outbreak of hand, foot and mouth disease in the community. This disease has been associated particularly with coxsackievirus A16, although A4, A5, A9 and A10 have been implicated. This apparent outbreak of hand, foot and mouth disease is unusual in its association with an outbreak of viral meningitis due to enterovirus 71 since the latter is not one of the enteroviruses most commonly involved in causing meningitis.

The differential diagnosis of aseptic meningitis based on typical CSF findings cannot rely on a predominance of polymorphonuclear leucocytes early in the disease progress (Table 2). The CSF findings which may differentiate viral from bacterial meningitis appeared to be a decrease in CSF protein level and a borderline or normal CSF glucose levels. Previous treatment with oral antibiotics does not appear to significantly alter most of the CSF parameters, with the exception of a slight decrease in the number of positive cultures.

Table 2. Patients with meningitis - Total CSF white cells $500 \times 10^6/L$

	Total	Polymorphs >60%	Protein >1g/l	Glucose <2.5mmol/l	Gram stain +ve	Antigen +ve
*Bacterial meningitis 160 cases	21	18	13	5/18	12/20	15/19
Viral meningitis Current epidemic	35	30	1	0	0	0

*Patients with CSF WCC $< 500 \times 10^6/L$

H.influenzae type b = 13/116
Strep.pneumoniae = 7/28
N.meningitidis = 1/15
Group G streptococcus =0/1

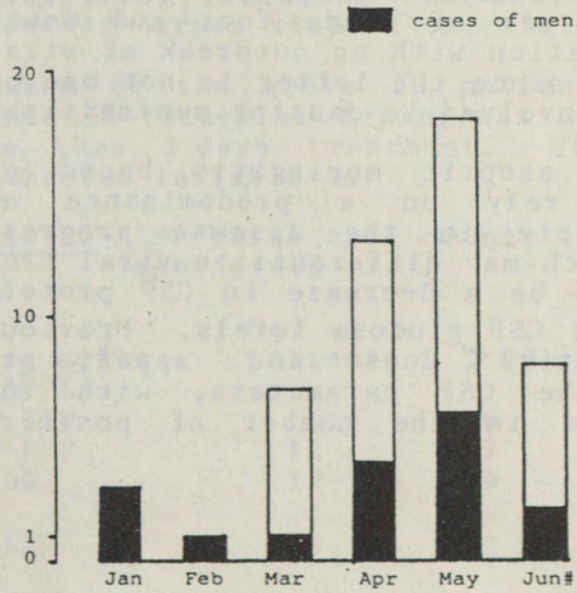
TOTAL = 21/160 (13%)

CDI EDITORIAL COMMENT

CDI 86/15 reported an apparent increase in enterovirus type 71 activity in children aged 1 year and below. Viral activity for the past two quarters is shown in Figure 1.

Enterovirus 71, the most recently discovered human enterovirus serotype, is the first non polio enterovirus with the potential to cause epidemic paralytic disease. However the Australian experience is that the clinical manifestations of this disease resemble those of coxsackievirus A16 infections. During an earlier Melbourne outbreak which was confined to the summer months of 1972-73, the majority of patients had either typical hand, foot, and mouth disease or aseptic meningitis, usually following a prodrome of 1-3 days of fever; serious CNS disease was not observed (1).

FIGURE 1. ENTEROVIRUS TYPE 71 (Prototype strain BrCr) ACTIVITY *



* Figures extracted from the CDI virus reporting scheme
# as notified to CDI in reporting period ending 21/7/86

Although enterovirus 71 was isolated with high frequency from a number of clinical specimens including faeces, oesopharyngeal secretions, vesicular skin lesions, it appeared to be never recovered from the cerebrospinal fluid, a finding also observed with poliovirus. Primary isolation appeared to have been most successful in African green monkey kidney cell culture and in suckling mice. Even under optimal conditions cytopathic effect may take 5-8 days to become visible, progressing slowly and incompletely[2]. Because standard enterovirus antiserum pools do not contain antiserum to enterovirus 71, isolates of this serotype may be reported as 'nontypable enteroviruses'.

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POLIOMYELITIS IN FINLAND

In Finland, regular immunisation against poliomyelitis began in 1957. Mass vaccination during the early 1960s resulted in the disappearance of the disease in 1964[1]. The vaccine used throughout the vaccination program has been the Salk-type inactivated poliovirus vaccine (IPV) containing formaldehyde-treated poliovirus types 1, 2 and 3. The immunisation schedule consists of six injections given at 5, 6 and 20-24 months and 6-7, 11-13 and 16-17 years.

In spite of the success of vaccination against the disease, surveys of the antibody titres to poliovirus in the Finnish population revealed that the inactivated vaccine was of low immunogenicity, particularly with regard to poliovirus type 3[1]. As a consequence, in September, 1984, the decision was made to use a new, more antigenic, trivalent IPV preparation[2] from 1986 onwards. However, between August, 1984, and January, 1985, an outbreak of poliomyelitis occurred in which nine paralytic cases and one non-paralytic case were reported[3, 4].

The age distribution of cases was 6-48 years. Two of the paralytic cases had no history of vaccination against the disease. One patient died from ventricular fibrillation during the induction of general anaesthesia. All of the nine paralytic cases were independent of each other and no secondary cases were identified among their contacts.

The single reported non-paralytic case occurred in a 6 year old boy who was admitted to hospital with serious meningitis. He made a complete and uneventful recovery and was discharged after a few days.

During the outbreak, poliovirus type 3 was isolated from the patients, from sewage water and from approximately 15% of healthy persons tested over a wide geographic area. Based on such findings it is estimated that of the order of 100,000 persons were infected by the virus during the outbreak[3].

As soon as the extent of spread of the virus was recognised the poliovirus vaccination program was intensified. From November, 1984, to early February, 1985, 1.5 million doses of IPV were given to children under 18 years of age. With the appearance of paralytic poliomyelitis in adults, the decision was taken to vaccinate adults as well. Oral poliovirus vaccine (OPV) was

offerred to the entire population between February 9 and March 15, 1985. Children under the age of 6 months were given the usual three doses of the regular IPV and immunocompromised individuals received a single dose of the new high-potency IPV. After this campaign there were no further reports of poliomyelitis and vaccine-like viruses were isolated from faecal specimens and from sewage up till the end of May, 1985.

In the year before the outbreak, 60% of samples tested for neutralising antibody to poliovirus type 3 had a titre 1/4. This had risen to 97% after the outbreak and the associated vaccination campaign.[3]

The recent re-emergence of poliomyelitis in Finland is not simply the result of the general low level of immunity to poliovirus type 3, since the same type of vaccine and vaccination protocol had been used in that country for the 20 years in which no cases of the disease were reported. Instead, it appears that the outbreak was due to the emergence of a strain of poliovirus type 3 that was sufficiently antigenically different to break through the low level of herd immunity[3]; the epidemic strain of poliovirus type 3 being found to differ from the type 3 vaccine strains in both immunological and molecular properties.

Exactly where and when the new poliovirus strain(s) originated is unknown. Similar antigenic variants have not yet been identified among poliovirus type 3 isolates from other regions of the world. Although genetic variation in poliovirus has been documented previously[5, 6], the wider epidemiological significance of the antigenic drift in poliovirus type 3 remains to be established.

CDI Editorial Comment

IPV has been used routinely in Finland for vaccination against poliomyelitis for nearly 30 years. The Finns have not made the switch to OPV because of fears of vaccine-induced paralytic disease. Such complications are an extremely rare occurrence. From 1969 to 1980, approximately 290 million doses of OPV were distributed in the United States. During this period only 92 cases of vaccine-associated paralysis were reported[7].

The vaccine for poliomyelitis that is used in Australia is the Sabin-type oral poliovirus vaccine (OPV) containing living attenuated strains of all three antigenic types. Doses are given at 2, 4 and 6 months of age with a booster dose recommended at school entry or entry to nursing school. Apart from the obvious ease of administration of OPV over IPV, it also has the advantage that the attenuated viruses follow the natural route of infection and induce antibody formation in

both the blood and the gut epithelium. This leads to a reduction in the number of symptomless excretors of wild-type poliovirus. IPV is also available for primary immunisation in persons for whom a live vaccine is contraindicated.

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MEASLES IN PRESCHOOL POPULATIONS

As a result of the initial success of the Childhood Immunisation Initiative undertaken in the United States in 1977, a measles eradication campaign was established in 1978 with the goal of eliminating indigenous measles by 1982[1,2,3]. The aims of this program were to: (i) achieve and maintain high immunisation levels, in part by the enactment and enforcement of school immunisation laws, (ii) undertake effective surveillance to detect measles cases and (iii) respond vigorously to outbreaks of the disease. Although these measures have resulted in a marked decline in the incidence of measles, eradication has not been achieved.

In 1985, a provisional total of 2,813 cases of measles were reported in the United States, representing an incidence rate of 1.2 cases per 100,000 population[4]. The highest incidence rate occurred among preschool-aged children (4.7/100,000) followed by individuals aged 15-19 (4.5/100,000). Cases in the latter group represent a large number of outbreaks on college campuses. Of the 826 preschool-aged children with measles, 211(25.5%) were under 12 months of age. The significance of the preschool population, both in terms of numbers of cases and their role in sustaining transmission is illustrated by a recent report on the 1983 epidemic of measles in Chicago[5].

In the period from January 4 through to August 12, 1983. physicians in the emergency room (ER) and outpatients clinics of Wyler Children's Hospital (WCH), Chicago, monitored patients for suspected measles infection after identifying two cases in the first week in January.

During the 32 week period of active surveillance, 107 patients were recognised as having illnesses resembling measles. Adequate diagnostic specimens were collected from 85(79%) patients and 54% of these were confirmed as having the illness. Complete information about the clinical features present at the time of initial evaluation was available for 49 of the patients with confirmed measles infection. Of these, 41(84%) met a clinical case definition of measles.

Although the 54 patients ranged in age from 4 months to 23 years, 41(76%) were less than 5 years of age and 23(43%) were under 16 months of age.

Information on measles immunisation status was available for 50 of the patients with confirmed measles. Of 22 patients who were less than 16 months old, none had been immunised against measles, although 15(68%) had received all of the other age-appropriate immunisations. Only four (22%) of the 18 patients aged 16 months to 4 years had a history of measles immunisation. Seven of ten school-aged children had a record of measles immunisation at 15 months or older.

Of the 54 confirmed cases of measles, information about cases of measles in household contacts was available for 45 (83%). The age-specific attack rate among household contacts less than 5 years of age was 38% (8/21), compared with 3% (4/116) for contacts aged 5 to 25 years.

Five putative sources of infection were evaluated serologically and all were confirmed to have had recent measles infections. Three of these sources were children less than 15 months of age. Of the patients with proved measles, only eight were in day-care centres or schools at the time they contracted the infection.

A retrospective study of sera collected from 204 children presenting at WCH in the year before the epidemic revealed that none of 23 patients less than 15 months of age had detectable antibody. Three (38%) of eight patients aged 15 months and 108(62%) of 173 patients aged 16 months to 4 years had antibody present.

The age distribution of cases in the 1983 Chicago epidemic resembles that of previous epidemics in the city.[6,7] Preschool-aged children have also been the major populations affected in recent measles epidemics in other cities in the United States.[8,9]

Preschool-aged children appeared to sustain the epidemic since the control measures directed at school-aged children, ie exclusion from school of children lacking proof of immunity and the subsequent termination of the school year, failed to change significantly the incidence of cases in the ER patients. The involvement of preschool-aged children in transmission was also documented in the 1967-1968 Chicago epidemic.[6]

The role played by preschool-aged children in the 1983 epidemic and the low prevalence of measles antibodies in children of this age, indicate that the current immunisation practices do not reach an epidemiologically important population. Since similar rates of seroconversion are obtained in children immunised against measles at 12 months or at 15 months[10,11], one approach to the problem would be to lower the age of immunisation in inner-city areas from 15 months to 12 months. For the 1983 epidemic it is estimated that such a measure may have directly prevented 15% of the cases detected.[5]

CDI Editorial Note

The experience with measles in the United States is particularly relevant to current initiatives to eliminate the disease in Australia. The role played by the preschool

population in the 1983 Chicago epidemic, particularly the group less than 16 months of age, emphasises the importance of ensuring measles vaccination of children at the recommended age of 12-15 months.

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AUSTRALIAN ENCEPHALITIS - A CASE REPORT
(based on clinical notes from Dr M. Bucens, A. Broom and T. Wright - State Health Laboratory Services - Perth)

On 29 April 1986, a 6 year old part-Aboriginal male from Fitzroy Crossing was admitted to the Fitzroy Crossing Hospital in a delirious state with a body temperature of 40°C following a 36 hour history of fever, delirium and a brief twitching episode. Although lumbar CSF appeared microscopically clear, intravenous antibiotics (Amoxicillin and Chloramphenicol) was initiated and the patient was transferred to Derby Hospital the following morning.

On arrival at Derby Hospital, the patient appeared more lucid, alert and orientated, however in the course of the afternoon, he developed a 41°C fever, became delirious and had rigors. These symptoms persisted during the next 24 hours, leading to a generalised hyperesthesia with the patient assuming a flexed posture with an extended neck. The White Cell Count in a second lumbar CSF remained nil but showed the following polymorphs proportion in the blood: 81% neutrophils, 40% lymphocytes and 5% monocytes. Other haematological values were within the normal range. Intravenous administration of fluids and antibiotics were continued with the addition of Gentamicin. A presumptive diagnosis of encephalopathy, either viral or due to Reyes syndrome was made.

The prolonged fever subsided on 2 May 1986 but the patient remained semiconscious without response to verbal stimuli. Blood and CSF specimens collected on that day indicated the

presence in the blood of antibodies to Herpes Simplex virus, measles (by complement fixation but no specific IgM detected) and Murray Valley Encephalitis (MVE) virus (by complement fixation with specific IgM detected). Antibody was not detected in the CSF. These serological findings were confirmed by subsequent blood specimens collected on 12 May 1986 and a diagnosis of encephalitis due to MVE virus was established.

Intravenous antibiotics were discontinued on 4 May 1986 and replaced by oral Amoxicillin and Chloramphenicol. The patient made a slow and gradual recovery and was discharged on 13 May 1986 without a full assessment by the Occupational Therapist. The patient was however believed to have no difficulty with co-ordination and motor planning.

A subsequent epidemiologic review of the case indicated that the patient might have acquired his infection at or near Noonkanbah Station (100 km west of Fitzroy Crossing) when the child had gone to stay with his aunt from 14 to 25 April 1986 while his father travelled to the Eastern states. During that period he had camped at Sandy Billabong (8 km from Noonkanbah) and had complained of being severely bitten by mosquitoes.

The MVE virus activity in the mosquitoes population of that region was also confirmed by sentinel chicken studies:

- Sentinel chickens from Fitzroy Crossing and Derby bled on 4-7 April 1986 were negative by ELISA for MVE virus antibody.
- 2/12 sentinel chickens from Derby bled in early May were positive for antibodies to MVE virus.
- 3/22 sentinel chickens from Fitzroy Crossing and 1/10 bantams and roosters from Noonkanbah Station collected on 20 May 1986 were positive for antibodies to MVE virus.

MYCOBACTERIAL SURVEILLANCE SCHEME

A Special Interest Group (Mycobacteria) which includes microbiologists from every large Australian mycobacteriology Laboratory has been formed within the Australian Society for Microbiology (ASM) to expand and improve the collection of data pertaining to mycobacterial diseases in Australia.

The Group will pay particular attention to diseases due to atypical mycobacteria and details of bacteriologically confirmed cases will be held in a computerised database, titled 'AUSTRALIAN ATYPICAL MYCOBACTERIA SURVEILLANCE SCHEME' at the Queen Elizabeth II Medical Centre, Perth.

The Group requests the assistance of medical practitioners and clinicians in providing laboratory scientists with relevant patients' details including residential history, associated diseases, therapy and outcome. Clinicians are also reminded that isolates should be forwarded to the State Reference Laboratory.

The data on atypical mycobacterial diseases should prove valuable in complementing the annual tuberculosis notifications compiled by the Commonwealth Department of Health. Further enquiries concerning either the database or the surveillance scheme should be addressed to:

Mr David Dawson
Convenor - SIG Mycobacteria
State Health Laboratory
PO Box 495
BRISBANE 4001

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD 22/7/86 - 4/8/86 BULLETIN NUMBER 86/16
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED			3		13	2	3		21
0101 ADENOVIRUS TYPE 1								1	1
0102 ADENOVIRUS TYPE 2				2		2		1	5
0103 ADENOVIRUS TYPE 3				1		2			3
0105 ADENOVIRUS TYPE 5				1					1
0108 ADENOVIRUS TYPE 8	1		1	1					3
0111 ADENOVIRUS TYPE 11	2								2
0113 ADENOVIRUS TYPE 13	1								1
0119 ADENOVIRUS TYPE 19	3								3
0199 ADENOVIRUS TYPING PENDING					4				4
0203 INFLUENZA B VIRUS	1	1	2						4
0301 PARAINFLUENZA VIRUS TYPE 1	1				2	1	3	2	9
0302 PARAINFLUENZA VIRUS TYPE 2	3	1		4	12	16	1		37
0303 PARAINFLUENZA VIRUS TYPE 3				1	1	1		1	4
0399 PARAINFLUENZA VIRUS TYPING PENDING						1	3		4
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	48	27	9	22	43	8	42	1	200
0500 RHINOVIRUS (ALL TYPES)	1			2	16	10	5	1	35
0600 MYCOPLASMA PNEUMONIAE	1	1	2	1			3	2	10
0700 ORNITHOSIS-PSITTACOSIS	1							1	2
0901 COXSACKIEVIRUS B1		3							3
1007 ECHOVIRUS TYPE 7	1								1
1011 ECHOVIRUS TYPE 11	3		1					9	13
1014 ECHOVIRUS TYPE 14	1								1
1016 ECHOVIRUS TYPE 16						1			1
1022 ECHOVIRUS TYPE 22	2								2
1100 POLIOVIRUS NOT TYPED			3		4				7
1101 POLIOVIRUS TYPE 1								1	1
1102 POLIOVIRUS TYPE 2				2		1			3
1103 POLIOVIRUS TYPE 3		1				1			2
1200 MUMPS VIRUS								5	5
1300 HERPES VIRUS GROUP-NOT TYPED	13		2					3	18
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1						2	3
1303 VARICELLA-ZOSTER VIRUS	1		2			2		3	8
1306 HERPES SIMPLEX TYPE 1	19			42		10	29	32	132
1307 HERPES SIMPLEX TYPE 2	77			81		26	61	76	321
1399 HERPES VIRUS TYPING PENDING					7	2		1	10
1401 COXIELLA BURNETI	1					3	4		8
1502 PICORNA VIRUS-NOT TYPED			2				12	2	16
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS		1							1
1522 RUBELLA VIRUS			1			1	2	3	7
1532 HEPATITIS B ANTIGEN	40		11	17	4	27	11	11	121
1535 HEPATITIS A ANTIBODY		1		3		19	1	16	40
1541 CHLAMYDIA A - C TRACHOMATIS	24		4			45	16	44	133
1556 CMV - CYTOMEGALOVIRUS	4		1	16	5	6	7	16	55
1564 ROTAVIRUS	35	15	20	7	20	61		1	159
1571 ENTEROVIRUS TYPE 71 (BRCR)	1			5					6
1599 ENTEROVIRUS TYPING PENDING		2	8		7				17
9992 ROSS RIVER VIRUS							6		6
9993 ASTROVIRUS	3								3
9994 SMALL VIRUS (LIKE) PARTICLE		5							5
Total	292	60	72	208	138	249	209	239	1,467

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 22/7/86 - 4/8/86
Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir-atory	Enceph-alitis	Mening-itis	Para-lysis	CNS other unspec	GI	Hepa-tic	CVS	Urin-ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....		1									
0102 ADENOVIRUS TYPE 2.....		4				1					
0103 ADENOVIRUS TYPE 3.....		2									
0105 ADENOVIRUS TYPE 5.....		1									
0111 ADENOVIRUS TYPE 11.....	2										
0113 ADENOVIRUS TYPE 13.....	1										
0119 ADENOVIRUS TYPE 19.....	2										
0203 INFLUENZA B VIRUS.....		2							1		
0301 PARAINFLUENZA VIRUS TYPE 1....		9									
0302 PARAINFLUENZA VIRUS TYPE 2....		34	1								
0303 PARAINFLUENZA VIRUS TYPE 3....		4									
0399 PARAINFLUENZA VIRUS TYPING PENDING.....		2									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	194		1			1			1	
0500 RHINOVIRUS (ALL TYPES).....	2	30		2							
0600 MYCOPLASMA PNEUMONIAE.....	3	8									
0700 ORNITHOSIS-PSITTACOSIS.....	1	1							1		
0901 COXSACKIEVIRUS B1.....		1		1							1
1007 ECHOVIRUS TYPE 7.....		1									
1011 ECHOVIRUS TYPE 11.....		1		4		2	4			1	
1014 ECHOVIRUS TYPE 14.....							1				
1016 ECHOVIRUS TYPE 16.....				1							
1022 ECHOVIRUS TYPE 22.....	1	1									
1100 POLIOVIRUS NOT TYPED.....		1	1								
1101 POLIOVIRUS TYPE 1.....		1									
1102 POLIOVIRUS TYPE 2.....	1	1									
1103 POLIOVIRUS TYPE 3.....		1									
1200 MUMPS VIRUS.....		3				1					1
1301 HERPES SIMPLEX VIRUS NOT-TYPED			1	1							1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	2							1			
1303 VARICELLA-ZOSTER VIRUS.....	2	1		1		1					2
1306 HERPES SIMPLEX TYPE 1.....	6	8		1						1	70
1307 HERPES SIMPLEX TYPE 2.....	10					1					63
1401 COXIELLA BURNETI.....	1	1									
1502 PICORNA VIRUS-NOT TYPED.....		4		1			5				2
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....		1									1
1522 RUBELLA VIRUS.....											5
1532 HEPATITIS B ANTIGEN.....	37							62			
1535 HEPATITIS A ANTIBODY.....	5							31		1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	9	1					1				1
1556 CMV - CYTOMEGALOVIRUS.....	3	20				1	1			6	1
1564 ROTAVIRUS.....	2	1		1			152				
1571 ENTEROVIRUS TYPE 71 (BRCR)....	1			1							4
1599 ENTEROVIRUS TYPING PENDING....											1
9993 ASTROVIRUS.....							3				
9994 SMALL VIRUS (LIKE) PARTICLE...				1			4				
Total.....	93	340	3	16		7	172	94	2	10	154

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 22/7/86 - 4/8/86
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0102 ADENOVIRUS TYPE 2.....		1								
0103 ADENOVIRUS TYPE 3.....								1		
0108 ADENOVIRUS TYPE 8.....	3									
0119 ADENOVIRUS TYPE 19.....	1									
0203 INFLUENZA B VIRUS.....									1	
0302 PARAINFLUENZA VIRUS TYPE 2....							1	2	1	
0399 PARAINFLUENZA VIRUS TYPING PENDING.....								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1	1	
0500 RHINOVIRUS (ALL TYPES).....										1
0600 MYCOPLASMA PNEUMONIAE.....								2		
0901 COXSACKIEVIRUS B1.....							1			
1011 ECHOVIRUS TYPE 11.....								1	1	
1102 POLIOVIRUS TYPE 2.....										1
1103 POLIOVIRUS TYPE 3.....										1
1200 MUMPS VIRUS.....					1					
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			2	2			1	1	1	
1303 VARICELLA-ZOSTER VIRUS.....		1						1		
1306 HERPES SIMPLEX TYPE 1.....		42				1			5	
1307 HERPES SIMPLEX TYPE 2.....		249							1	
1399 HERPES VIRUS TYPING PENDING...		1								
1401 COXIELLA BURNETI.....					2		3	4		
1502 PICORNA VIRUS-NOT TYPED.....										1
1522 RUBELLA VIRUS.....					1			1	1	
1532 HEPATITIS B ANTIGEN.....									22	
1535 HEPATITIS A ANTIBODY.....									4	
1541 CHLAMYDIA A - C.TRACHOMATIS...	4	115							2	
1556 CMV - CYTOMEGALOVIRUS.....		2	2	2		4	1		15	1
1564 ROTAVIRUS.....		1								
9992 ROSS RIVER VIRUS.....					6			5		
Total.....	8	412	4	4	10	5	7	20	55	5