Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 912 reports were received this period. General patterns as suggested by these reports include:

- Notification of respiratory syncytial virus infections has continued to decrease - 50 reports received compared to 85, 165 and 184 for the previous three periods - and this decline suggests the end of the annual winter infection cycle.
- A rise in the number of rubella infections - 35 reports received compared to 23, 20 and 14 for the previous three periods. Eight reports originated from the Woden Valley Hospital, Canberra, and all of the patients showed evidence of recent infection.
- An increase in the number of reports of adenovirus type 19 isolated from genital sources were received from the State Health Laboratory, Perth - 20 reports received compared with 5, 5 and 9 for the previous three periods.

Other reports of interest include:

- Further investigation into the three unresolved cases of group B arbovirus infection reported by the State Health Laboratory, Brisbane, in CDI 80/15 has resulted in:

  Japanese B encephalitis has been diagnosed in the 30 year old female with meningoencephalitis who was stationed with the army in Malaysia.

  The diagnosis of the 43 year old housewife from the Burnett area of Queensland, who was regarded as the possible index case in the re-establishment of indigenous dengue fever, remains unresolved. Further serum samples have shown no activity against arbovirus group B antigen, and an error in the labelling of her first serum samples is suspected.

  Diagnosis of the 44 year old male hospital orderly who had no history of overseas travel also remains unresolved, and investigations are continuing

- Salmonella singapore has been isolated by the Microbiological

(continued on page 8)

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EPIDEMIOLOGY OF HUMAN LEPTOSPIROSIS IN AUSTRALIA

(Contributed by B. Adler and S. Faine, Department of Microbiology, Monash University, Melbourne. Some of the data was supplied to them by N. Stallman, State Health Laboratory, Brisbane.)

Leptospirosis refers to infection with spirochaetes of the genus Leptospira. Weil in 1886 described a particular type of jaundice accompanied by splenomegaly, renal dysfunction, conjunctivitis and skin rashes, which was subsequently named Weil's disease. Thirty years elapsed before Japanese workers isolated the causal organism and named it Spirochaeta icterohaemorrhagiae. The generic name Leptospira was adopted in 1917. The identification of many other serologically distinguishable strains followed and at present over 150 serovars are recognized. Their distribution is worldwide and leptospires have been isolated from many species of terrestrial and marine animals. At present, all pathogenic leptospires are classified in a single species (Leptospira interrogans) comprising many serovars. A second complex (Leptospira biflexa) contains the serovars of the saprophytic, free-living leptospires.

It is difficult to obtain accurate and up to date information about human leptospirosis in Australia from current medical, veterinary or microbiology textbooks. In most instances these refer to the situation which exists in Great Britain or the United States and which may be inappropriate for Australia. Even when these do mention leptospirosis in Australia, they refer to the situation as it was understood 20 or more years ago when human infection was found mainly in sugar cane workers in Northern Queensland. The epidemiological picture in Australia has changed and, except in Northern Queensland, it is now similar to that found in other farming communities in temperate climates such as New Zealand and Northern China.

Because of its many and varied symptoms, leptospirosis often passes undiagnosed. The classical and severe presentation as described in text-books is mostly inappropriate, e.g. in a recent New Zealand outbreak only one out of 58 cases presented with jaundice. Furthermore, the human antibodies produced in response to infection cross-react widely between different serovars so that it is difficult to ascertain from sero-epidemiological data which are the prevalent serovars causing human infection. So unless infecting serovars are isolated (this is not routinely done) epidemiological knowledge is limited. Therefore much of the description below is from the observations and experiences of a few interested individuals rather than from a large formal collection of data.

In most cases the initial source of infection is the carrier animal which sheds organisms in its urine. Infection may occur by either direct contact such as dairy farmers with cattle or by contact with contaminated waters or soils. Pathogenic leptospires can survive for at least several weeks in surface waters if conditions are favourable. Man may also become infected from animals with current acute infections, e.g. farmers or veterinarians handling aborted foetuses.

The epidemiological picture of leptospirosis in Australia may conveniently

be divided into two types.

1. Tropical - This pattern, restricted to Northern Queensland, is similar to that observed in other tropical countries such as Fiji or Malaysia. There is no particular occupational distribution and many different serovars may be involved arising from several different species of host animal. The table on page 4 shows the different serovars of pathogenic leptospires which have been isolated in Australia. In general, infection in tropical areas follows contact, either direct or indirect, with infected urine usually of wild carrier animals such as field rodents and marsupials, but also sometimes from domestic animals. Classical severe Weil's disease caused by serovar copenhageni (formerly icterohaemorrhagiae AB) is very rare in Australia and canicola infection is virtually unknown. The unimportance of the domestic dog as a source of canicola infection anywhere in Australia contrasts with the situation in the United States.

2. Temperate - In temperate climate communities the disease is almost always occupation associated and nearly always contracted from domestic animals. Thus the number of serovars involved is limited to those associated with particular hosts (see table), mainly hardjo but some pomona in cattle, and tarassovi and pomona in pigs. There is evidence, in Victoria at least, for widespread infection of sheep with serovar hardjo. Although pomona has been isolated from horses, the importance of this host as a source of human infection is not yet known. In contrast to the tropical situation temperate leptospirosis shows a marked seasonal distribution with the majority of cases occurring in spring and summer.

   The epidemiology of leptospirosis in temperate Australia has been characterised by a dramatic change from pomona to hardjo as the predominant infecting serovar during the last 12 years. Figures from Southern Queensland show that the percentage of hardjo infections rose from 5% in 1967/68 to 48% in 1975/76 and now remains at about 50%. During the same period the proportion of pomona infections fell from 74% to 40%. In Southern Australia the change from pomona to hardjo appears to have been even more dramatic, although extensive figures are not available, and in dairy farming areas of New Zealand hardjo now accounts for 80-90% of human infections. No satisfactory explanation is apparent for this shift.

   The role of the wildlife population in temperate leptospirosis is likewise a matter for conjecture. Although leptospires may be isolated from wild animals it is probable that domestic animals themselves are in the main reservoir. New Zealand studies showed that the serovar pattern in the wildlife population was different from that in domestic animals and in that situation at least it was concluded that wild animals were of little importance in transmitting the disease to animals or man.

TABLE Leptospires isolated in Australia

Serogroup	Serovar	Man	Animal Hosts Domestic	Wild
Icterohaemorrhagiae	icterohaemorrhagiae	-	dog	
	copenhageni	+	dog	rat
	mankarso	+		
Celledoni	celledoni	+		bandicoot
Canicola	canicola	+		bandicoot
	broomi	+		rat
	bindjei	+		rat
Pyrongenes	zanoni	+		bandicoot rat mouse
	robinsoni	+		rat
Autumnalis	bulgarica	+		
Australis	australis	+	cattle	rat bandicoot mouse
	bratislava	+		
Pomona	pomona	+	cattle sheep pigs horses	rat
Grippotyphosa	grippotyphosa	+		bandicoot rat
Hebdomadis	kremastos	+		bandicoot
	szwajizak	+		bandicoot
	medanensis	+		bandicoot
	perameles	-		bandicoot
	hardjo	+	cattle sheep	possum
	balcanica	-		possum
Tarassovi	tarassovi	+	pigs	
	bakeri	-		rat

CYTOMEGALOVIRUS INFECTION

A total of 745 cytomegalovirus (CMV) identifications were reported to CDI in 1979 - an increase of 245 over 1978. Of the reports in 1979, 283 were from contributing laboratories in Victoria, 232 from New South Wales

and the Australian Capital Territory, 85 from Western Australia, 81 from South Australia and 64 from Queensland. However, these figures do not necessarily reflect the national distribution of CMV infection, and not all the reports were of new cases.

The tables below show age and clinical symptoms for the identifications reported.

Table 1 CMV Identification by Age Groups

Age Group	No. of reports	Age Group	No. of reports
0-5 months	153	15-24 years	109
6-11 months	84	25-59 years	159
1-4 years	109	60+ years	24
5-14 years	33	Age not stated	74

Table 2 Clinical Symptoms associated with CMV Infection

Clinical Symptoms	No. of reports	Clinical Symptoms	No. of reports
No illness or no data	214	Skin/Mucous Membrane	7
Respiratory	151	Eye	2
Encephalitis/Meningitis	13	Genital	11
Paralysis	9	Endocrine/Salivary Glands	15
Central Nervous System	18	Reticuloendothelial System	14
Gastrointestinal	15	Muscle/Joint	6
Hepatic	28	Congenital	58
Cardiovascular System	5	Fever/Malaise	114
Urinary	35	Other	101
		Sudden Infant Death Syndrome	7

Comment

(Based on information contributed by K. Hayes, Department of Virology, Fairfield Hospital, Melbourne.)

CMV is now acknowledged to be one of the most common viruses of man - although it rarely causes clinical disease. It can remain latent for long periods in various tissues and organs. Infected cells may have a characteristic swollen appearance with intranuclear inclusions. Transmission occurs by close physical contact. Patients excrete virus in urine, saliva and genital secretions, and it may also be isolated from breast milk, blood, bronchial mucosa and cerebrospinal fluid.

Prenatal infection - Studies at Fairfield Hospital on the incidence and significance of prenatal CMV infection have shown that the risk of a woman becoming infected for the first time during pregnancy is about 1 in 300, and the subsequent risk of foetal infection about 50%. Tests

on 6000 newborn showed prenatal infection rates of 1 in 180 births for public patients, and 1 in 330 births for private patients. These figures indicate that not all prenatal CMV infection can be accounted for by transplacental spread from primary maternal infections.

Although most infants with congenital CMV infection appear normal at birth, some exhibit features such as hepatosplenomegaly, jaundice, thrombocytopenia, pneumonitis, intrauterine growth retardation and evidence of brain damage. Nearly all the latter ultimately exhibit permanent handicaps such as intellectual deficiency, cerebral palsy, microcephaly, epilepsy, deafness or visual defects. When there is no evidence of clinical disease at birth, most neonates remain normal, but a minority may subsequently show evidence of brain damage in the form of delayed development, deafness or minor neurological deficits.

Amniocentesis offers an opportunity for the diagnosis of prenatal CMV, but because most maternal infections are subclinical, the fact that a foetus may be at risk is usually not suspected. Definitive diagnosis of prenatal infection can only be made if appropriate laboratory tests are performed during the first two weeks after birth.

Children infected before birth continue to excrete virus in urine and saliva for long periods, although it is considered that after 12 months they need not be regarded as contagious, because of the low amounts of virus excreted by this age.

Early postnatal infection - Early postnatal CMV infection is quite common as the virus is frequently present in the birth canal, breast milk and occasionally in maternal saliva or urine. There is no evidence that early postnatal CMV causes brain damage - indeed, abnormal clinical signs are rare. Occasionally, however, an illness like glandular fever, hepatitis or pneumonitis may develop.

CMV can be transmitted by blood, so transfused infants are at risk of iatrogenic infection. In Melbourne, the incidence of CMV infection in infants given exchange transfusions was 29%, compared with 19% for infants given simple blood transfusions, and 10% for infants not given blood.

Similar studies in the U.S.A. and U.K. have also shown that CMV contributes significantly to morbidity and mortality in infants given blood transfusions, particularly in infants of women with no CMV antibodies. The incidence of transfusion-associated CMV infection can be reduced significantly by using blood from CMV-seronegative donors, or possibly by using blood that has been stored for more than 72 hours.

Further definition of the clinical effects of prenatal and early postnatal CMV infection is being attempted at Fairfield Hospital. Correlation of these effects with virus excretion and the persistence of virus specific IgM antibodies may then indicate the appropriateness of the CMV-specific IgM antibody test as a routine screening for all newborn infants.

Childhood infection - As yet there is no accurate Australian data on prevalence of active CMV infection during childhood, although serological data indicates that 50% of the lower socio-economic groups have been infected by 15-19 years. Young children are probably an important source of CMV infection for persons in close contact with them, e.g., members of their household.

Older age groups - Both primary and reactivated CMV infections have been recognised. Reactivation frequently occurs in patients with deficiencies in cell-mediated immunity, particularly in patients receiving prolonged immunosuppressive therapy for renal and other transplants, leukaemia and other malignancies. Infection may present as disseminated disease or in a localised form involving the lymphatic, hepatic or pulmonary systems. Reactivation also occurs in some pregnant women.

Control of CMV - From a public health point of view, the main problems attributed to CMV are in congenital infection, which is an important cause of intellectual and neurological deficiency, and in premature infants and immunosuppressed patients dependent on blood transfusions.

Experimental vaccines have been developed, the most recent of which is a sub-unit (non-infectious) vaccine. However, prophylaxis is complicated by the requirement for cell mediated immunity as well as a possible serotype specific antibody response. In addition, CMV epidemiology influences vaccination policy since postnatal infections are usually subclinical, and the prevalence of naturally acquired immunity increases with age. Selection of CMV-seronegative blood and kidney donors for patients at risk is an alternative approach.

Although certain antiviral drugs have been used in the management of serious CMV infections, problems of inefficacy and toxicity have become apparent. Other present and potential therapies include lymphocyte transfer factor, interferon, and hyperimmune plasma.

ACUTE HAEMORRHAGIC CONJUNCTIVITIS IN INDO-CHINESE REFUGEES

(Based on MMWR (1980) 29:445)

In mid-July, an outbreak of conjunctivitis was seen among 60 of 520 South East Asian refugees arriving at Oakland, California. Following establishment of a surveillance system at all U.S. quarantine stations, 528 arriving refugees of 9376 surveyed were found to have clinical conjunctivitis. Most of the cases were characterised by conjunctival injection, swelling of eyelids, and scanty white discharge in one or both eyes, with no systemic symptoms. However, 21 of the 528 cases had haemorrhagic manifestations. Picornavirus were isolated from four specimens, one of which was typed as enterovirus type 70. This virus is the agent predominantly responsible for acute haemorrhagic conjunctivitis (AHC).

Screening procedures were set up in Thailand. Of the 2356 refugees surveyed, 8.5% had conjunctivitis of which 58% were haemorrhagic. Separation and detention until the infection was clinically resolved of those families with members having signs of AHC, reduced the numbers seen

at U.S. quarantine stations.

Epidemic conjunctivitis due to enterovirus type 70 infection (as well as adenovirus type 11 and type 8) also occurred among South East Asian refugees on Guam in 1975. Conjunctivitis sometimes occurred among American medical personnel in Guam in close contact with the patients, but no transmission was documented in the United States. This fact, and the crowded and less adequate hygienic conditions usually reported with outbreaks of AHC, make the likelihood of secondary spread in a host country minimal.

β-LACTAMASE PRODUCING N. GONORRHOEAE

Six further isolations have been reported for the months September and October. This brings the 1980 total to 111.

Sex	Age	State	Origin of Contact
M	39	Victoria	Local
M	25	Victoria	Bangkok
M	28	Victoria	Thailand
F	26	Victoria	Fiji
M	31	Victoria	Singapore
M	28	Victoria	Bangkok

CDI DISTRIBUTION

The distribution of the CDI bulletin is running at just under 960 copies per issue at present. An approximate breakdown is as follows:

Universities/Major Teaching Hospitals	- 21.5%
Other Hospitals/Official Laboratories	- 21%
Private Practitioners/Individuals	- 18.5%
State Departments of Health/Health Authorities	- 12%
Overseas	- 10%
Private Laboratories	- 5%
Non-Health Departments, Institutes, Businesses, Colleges, etc.	- 4.5%
Internal Distribution	- 3%
Professional Bodies (Red Cross, etc.)	- 3%
Armed Services	- 1%
Miscellaneous (Press,etc.)	- 0.5%

Contributed articles for this bulletin are welcomed.

(continued from page 1)

Diagnostic Unit, Melbourne, from seven staff members of a Melbourne Metropolitan Hospital. The species, first isolated from two patients, has been found in three domestic ward staff, three kitchen staff and a night nursing aide.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 2/10/80 to 15/10/80 ...

80/21

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
0100 ADENOVIRUS NOT TYPED.....	1									1
0102 ADENOVIRUS TYPE 2.....										1
0103 ADENOVIRUS TYPE 3.....									1	
0105 ADENOVIRUS TYPE 5.....	1						1	1		
0107 ADENOVIRUS TYPE 7.....	2									
0110 ADENOVIRUS TYPE 10.....		1								
0119 ADENOVIRUS TYPE 19.....	5	20								
0201 INFLUENZA A VIRUS.....	1		3				3	9	2	
0202 INFLUENZA A VIRUS SUBTYPE H3N2								1		
0203 INFLUENZA B VIRUS.....							1	3		1
0301 PARAINFLUENZA VIRUS TYPE 1....								1		
0302 PARAINFLUENZA VIRUS TYPE 2....			1					1		
0303 PARAINFLUENZA VIRUS TYPE 3....			1				1	1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1		1
0500 RHINOVIRUS (ALL TYPES).....							1			1
0600 MYCOPLASMA PNEUMONIAE.....			1				1	3		
0700 ORNITHOSIS-PSITTACOSIS.....							1			
0809 COXSACKIEVIRUS A9.....										2
1007 ECHOVIRUS TYPE 7.....								1		
1030 ECHOVIRUS TYPE 30.....										1
1101 POLIOVIRUS TYPE 1.....										1
1104 POLIOVIRUS-VACCINAL STRAIN....									1	1
1200 MUMPS VIRUS.....	1		7					2		
1300 HERPES VIRUS GROUP-NOT TYPED..		1							1	
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	19						2		
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..			4					1		
1303 VARICELLA-ZOSTER VIRUS.....	1							1	1	

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 6

PERIOD : 2/10/80 to 15/10/80 ... 80/21
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 ... **-CONTINUED**

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
1306 HERPES SIMPLEX TYPE 1.....	2	8						4	1	
1307 HERPES SIMPLEX TYPE 2.....		102								
1401 COXIELLA BURNETI.....			1		2		6	10	1	
1522 RUBELLA VIRUS.....			1		3			2		
1541 CHLAMYDIA A - TRIC TYPE.....	2	84								
1556 CMV - CYTOMEGALOVIRUS.....		4			1	2	1	2	4	1
ROSS RIVER VIRUS					2					
Total.....	17	239	19		8	2	16	46	12	11

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 3

PERIOD : 2/10/80 to 15/10/80 .... 80/21

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	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0100 ADENOVIRUS NOT TYPED.....		3		1			8		1		
0101 ADENOVIRUS TYPE 1.....		1	1			2	3				
0102 ADENOVIRUS TYPE 2.....		5					2				
0103 ADENOVIRUS TYPE 3.....							2				
0105 ADENOVIRUS TYPE 5.....		3				1	3				
0107 ADENOVIRUS TYPE 7.....		2									
0115 ADENOVIRUS TYPE 15.....							1				
0201 INFLUENZA A VIRUS.....		20							2		1
0202 INFLUENZA A VIRUS SUBTYPE H3N2		10									
0203 INFLUENZA B VIRUS.....		23							2		3
0301 PARAINFLUENZA VIRUS TYPE 1....		2									
0302 PARAINFLUENZA VIRUS TYPE 2....		3									
0303 PARAINFLUENZA VIRUS TYPE 3....		23				1	1				1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	45				1			1		
0500 RHINOVIRUS (ALL TYPES).....		18									
0600 MYCOPLASMA PNEUMONIAE.....	4	9							1		1
0809 COXSACKIEVIRUS A9.....				1							
0816 COXSACKIEVIRUS A16.....											2
0901 COXSACKIEVIRUS B1.....			1								
1006 ECHOVIRUS TYPE 6.....	1										
1009 ECHOVIRUS TYPE 9.....						1					
1011 ECHOVIRUS TYPE 11.....				1							
1022 ECHOVIRUS TYPE 22.....		1				1					
1025 ECHOVIRUS TYPE 25.....				1							
1030 ECHOVIRUS TYPE 30.....		1	1	3							
1101 POLIOVIRUS TYPE 1.....		2									
1102 POLIOVIRUS TYPE 2.....	1						2				

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 4

PERIOD : 2/10/80 to 15/10/80 .... 80/21

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS otner unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
1103 POLIOVIRUS TYPE 3.....							2				
1104 POLIOVIRUS-VACCINAL STRAIN....		1				1	1				
1200 MUMPS VIRUS.....	2			2			1				1
1300 HERPES VIRUS GROUP-NOT TYPED..	1					2					5
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1		1								29
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	1										
1303 VARICELLA-ZOSTER VIRUS.....					1						1
1306 HERPES SIMPLEX TYPE 1.....	1	6	1							1	20
1307 HERPES SIMPLEX TYPE 2.....											6
1401 COXIELLA BURNETI.....	2	2		1							
1521 MEASLES VIRUS.....	1	1	1								5
1522 RUBELLA VIRUS.....	2										32
1530 HEPATITIS A VIRUS.....	3							6			
1531 HEPATITIS B VIRUS.....	14							25			
1532 HEPATITIS B ANTIGEN.....								2			
1535 HEPATITIS A ANTIBODY.....								6			
1541 CHLAMYDIA A - TRIC TYPE.....											1
1556 CMV - CYTOMEGALOVIRUS.....	2	6	1					1		1	3
1564 ROTAVIRUS.....		2		1			79				
ROSS RIVER VIRUS		1									
ASTROVIRUS							3				
SMALL VIRUS (LIKE) PARTICLE							6				
Total.....	38	190	7	11	1	10	114	40	7	2	111

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 1

REPORTING PERIOD - 2-10-80 — 15-10-80 BULLETIN NUMBER . 80/21

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			1	1	1	1	2	7	13
0101 ADENOVIRUS TYPE 1			1	1	2	1		1	6
0102 ADENOVIRUS TYPE 2	4		1		2	1			8
0103 ADENOVIRUS TYPE 3	1		2						3
0105 ADENOVIRUS TYPE 5	2			1	1	4			8
0107 ADENOVIRUS TYPE 7			2					2	4
0110 ADENOVIRUS TYPE 10								1	1
0115 ADENOVIRUS TYPE 15						1			1
0119 ADENOVIRUS TYPE 19	1			2				20	23
0199 ADENOVIRUS TYPING PENDING					7	6			13
0201 INFLUENZA A VIRUS	1	1	4	8		7	4	12	37
0202 INFLUENZA A VIRUS SUBTYPE H3N2				5	3	1	1		10
0203 INFLUENZA B VIRUS	1		3	5	7	6	5	4	31
0301 PARAINFLUENZA VIRUS TYPE 1								3	3
0302 PARAINFLUENZA VIRUS TYPE 2					2		1	1	4
0303 PARAINFLUENZA VIRUS TYPE 3	1	2		1	8	4	4	8	28
0399 PARAINFLUENZA VIRUS TYPING PENDING								5	5
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	3	2	1	4	18	13		9	50
0500 RHINOVIRUS (ALL TYPES)		2		2	7	1	7	1	20
0600 MYCOPLASMA PNEUMONIAE		1	8			4	4	2	19
0700 ORNITHOSIS-PSITTACOSIS						1			1
0809 COXSACKIEVIRUS A9					2	1			3
0816 COXSACKIEVIRUS A16	1			1					2
0901 COXSACKIEVIRUS B1						1			1
1006 ECHOVIRUS TYPE 6							1		1
1007 ECHOVIRUS TYPE 7								1	1
1009 ECHOVIRUS TYPE 9						1			1
1011 ECHOVIRUS TYPE 11						1			1
1022 ECHOVIRUS TYPE 22								1	1
1025 ECHOVIRUS TYPE 25				1					1
1030 ECHOVIRUS TYPE 30	1			2	3				6
1099 ECHOVIRUS TYPING PENDING			1						1

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 2.

REPORTING PERIOD - 2-10-80 — 15-10-80 BULLETIN NUMBER - 80/21

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

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
1101 POLIOVIRUS TYPE 1				1				2	3
1102 POLIOVIRUS TYPE 2			2					1	3
1103 POLIOVIRUS TYPE 3						2			2
1104 POLIOVIRUS-VACCINAL STRAIN					5				5
1200 MUMPS VIRUS	1	1		3	2	2	3	1	13
1300 HERPES VIRUS GROUP-NOT TYPED	3		3			3	1		10
1301 HERPES SIMPLEX VIRUS NOT-TYPED	8			4				35	47
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	4			1				1	6
1303 VARICELLA-ZOSTER VIRUS			5						5
1306 HERPES SIMPLEX TYPE 1	4		2	18		9	9		42
1307 HERPES SIMPLEX TYPE 2	44		10	21		15	17		107
1399 HERPES VIRUS TYPING PENDING			5		1	5			11
1401 COXIELLA BURNETI	5					6	10		21
1521 MEASLES VIRUS		3	2	1	1			1	8
1522 RUBELLA VIRUS	18		4	3			7	3	35
1530 HEPATITIS A VIRUS						4		5	9
1531 HEPATITIS B VIRUS				20		8	5	6	39
1532 HEPATITIS B ANTIGEN			2						2
1535 HEPATITIS A ANTIBODY				6					6
1541 CHLAMYDIA A - TRIC TYPE	28		1					57	86
1556 CMV - CYTOMEGALOVIRUS	1		9	3	4	3	3	4	27
1564 ROTAVIRUS	24		10	11	1	7		28	81
1599 ENTEROVIRUS TYPING PENDING		1			20	3			24
ROSS RIVER VIRUS							2	1	3
ASTROVIRUS	3								3
SMALL VIRUS (LIKE) PARTICLE	5			1		1			7
Total	164		79	127	97	123	86	223	912