Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 853 reports were received this period, although figures from one laboratory were delayed in the mail. Patterns suggested by the reports comprise a downward trend to the seasonal increase of rotavirus gastroenteritis (36 reports received compared with 75, 66 and 100 for the previous three periods), and a continuation of the recent increase in rubella infections (42 reports compared with 42, 23 and 29). The latter reports included a congenital rubella syndrome from the State Health Laboratory, Brisbane, in a neonate with bilateral cataracts, deafness, heart defect and microcephaly. Rhinovirus and parainfluenza virus type 3 predominated the respiratory infections.

Individual reports included:

- Dengue - Four further cases of indigenous dengue fever were confirmed by the State Health Laboratory, Brisbane. These included a 32 year old male and 29 year old female from Thursday Island, a 50 year old male from Cairns, and a 55 year old male from Townsville (see also article in this issue).

  The Brisbane laboratory also confirmed a dengue type 4 infection in a 51 year old male from Wagga Wagga, New South Wales, who had recently returned from Fiji. To 17 October only 18 cases of dengue with one death have been recorded on the island group this year.

- Fairfield Hospital, Melbourne, reported two further isolations of adenovirus type 37 from patients with conjunctivitis. Their first isolation was reported in the virus tables of CDI 81/22. At present this new serotype is being coded as adenovirus type 19 until official classification has been accorded (see also CDI 81/16).

Other reports of interest include:

- The State Health Laboratory Services, Perth, diagnosed Legionnaires' Disease in a 61 year old male who presented in a collapsed state on 14 October 1981. Although serum specimens taken on 14 and 26 October gave IgG titres by ELISA of 1/20 and 1/1280 respectively against L. pneumophila serogroup 1, no IgM could be detected.

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DENGUE FEVER IN AUSTRALIA - 1981

In recent years there has been a global resurgence of dengue and dengue haemorrhagic fever (DHF) with major epidemics in the Western Pacific Region (1979-1980)[1] and the Carribean and Mexico (1980-81)[2]. As of 9 September 1981, 343,924 cases of dengue type 2 infection with 156 deaths were reported in Cuba alone[3]. Reports of dengue have subsequently decreased in the Western Pacific Region (Cook Islands, Fiji, Kirribati, New Caledonia and Tonga)[4], but 12 provinces of Indonesia recorded 498 confirmed or suspect cases of DHF with 17 deaths during March 1981 (compared with 306 for March 1980)[5], and there have been two reports of dengue fever in Australians living in, or returning from, Papua New Guinea.[6,7]. During 1980, 44 reports of confirmed or clinical dengue were received by CDI in patients returning from endemic areas, but this year the figure has fallen to four.

Indigenous dengue was last recorded in Australia in 1954-55, when approximately 40% of the population of Townsville in north Queensland were affected.[8]. The first indication of the reintroduction of dengue was recorded in July this year in a 46 year old female from Cairns[9]. To 17 November 1981, a total of 13 confirmed and four clinical reports of indigenous dengue have been received from the State Health Laboratory, Brisbane. Cases emanated from Cairns (7), Townsville (5) Innisfail (1), Chillagoe (1) and Thursday Island (3). The total number of clinical dengue cases and distribution in north Queensland is still being evaluated, but the outbreak on Thursday Island in the Torres Strait has so far involved approximately 150 cases (B. Kay, Queensland Institute for Medical Research, personal communication). Most patients presented with a diphasic fever (40°C), rash and joint pain. Preliminary investigation of 415 houses before the introduction of a vector control program indicated 120 positive findings of mosquito larvae. Cases have also been reported on the islands of York, Yam, Badu, Horn and Prince of Wales.

The classical form of dengue is an acute febrile disease with headaches and joint and muscular pains. However, the disease can progress to a haemorrhagic phenomenom (dengue haemorrhagic fever, DHF) with a tendency to develop a shock syndrome (dengue shock syndrome, DSS). Thrombocytopenia (100,000/mm^3 or less) with concurrent haemoconcentration (haemotacrit increased by 20% or more) is regularly observed. Table 1 on page 3 lists the clinical signs and symptoms.

Dengue is endemic in tropical regions where *Aedes* (*Stegomyia*) mosquitoes, principally *Ae. aegypti*, are present[10]. Epidemics occur only if the infectious agent, an immunologically susceptible human population and abundant arthropod vectors are present. *Ae. aegypti*, a container breeder, is the most efficient vector because of its domestic habitat. The female mosquito bites man during the day, and after feeding on a viraemic person is able to transmit dengue virus either immediately, or after an incubation period of 8-10 days following replication in its salivary glands. The Commonwealth Department of Health operates an *Ae aegypti* surveillance program involving the use of ovitraps and an active search for breeding places at the northern seaports and all Australian international airports.[11]

TABLE 1 Clinical features of dengue fever

1. Bull. WHO. (1980) 58:1
2. Guide for diagnosis, treatment and control of dengue haemorrhagic fever. Second edition. (1980). Technical Advisory Committee on Dengue Haemorrhagic Fever for the South East Asian and Western Pacific Regions, WHO.

CLASSICAL DENGUE(1)

Classical dengue has an incubation period of 5-8 days, followed by the onset of fever, chilly sensations, severe headache, retro-orbital pain, backache, pain in the muscles and joints, and later anorexia, constipation, weakness, prostration and epigastric discomfort. Onset is sudden, and may be preceded by grippe-like prodromal symptoms without fever. Fever persists for 5-7 days, sometimes with a diphasic presentation. There is a rapid pulse early in infection, followed by brachycardia. Also the face and neck may appear flushed, with a transient punctiform rash evident on the elbows and knees. A maculopapular or scarlatiniform rash appears on day 3-5, beginning on the chest and spreading centrifugally. Petechiae are sometimes evident. Lymphadenopathy and leukopenia are frequent findings, and jaundice has been described. Infants and young children may have an undifferentiated febrile disease with maculopapular rash; older children and adults may have either mild febrile syndromes or the classical incapacitating disease. The case fatility rate is exceedingly low. Bleeding complications such as petechial haemorrhage, epistaxis, gingival bleeding, gastro-intestinal bleeding and haematuria are sometimes seen in epidemics.

DENGUE HAEMORRHAGIC FEVER (DHF)(2)

Onset basically begins with a sudden temperature rise, accompanied by facial flush and other non-specific constitutional symptoms such as anorexia, vomiting, headache and muscle or joint pains. A sore throat and injected pharynx, epigastric discomfort, tenderness at the right costal margin and generalised abdominal pain are common. Temperature is high (occasionally 40-41°C with febrile convulsions), and persists for 2-7 days before falling to normal or subnormal by lysis. Skin haemorrhage is common:- a positive tourniquet test, easy bruisability and bleeding at venepuncture. Fine petechiae scattered on extremities, axillae, face and soft palate may be seen during the early phase, and a confluent petectial rash with characteristic coin-sized areas of normal skin is sometimes seen in convalescence. A macular, maculopapular or rubella-like rash may develop. Epistaxis and gum bleeding are infrequent. Gastrointestinal haemorrhage is rare, usually following a period of uncontrolled shock. The liver is usually palpable and tender early in the febrile phase, varying from just palpable to 2-4 cm below the costal margin. Size is not correlated with disease severity. Lysis of fever may be accompanied by profuse sweating and mild changes in pulse rate and blood pressure together with coolness of extremities and skin congestion. Patients usually recover spontaneously or after fluid and electrolyte therapy.

DENGUE SHOCK SYNDROME (DSS)(2)

In severe cases, the patient's condition suddenly deteriorates with a fall in temperature and signs of circulatory failure. The skin becomes cool, blotchy and congested; circumoral cyanosis is frequently observed and the pulse becomes rapid. Although some patients may appear lethargic, they become restless and go into a critical stage of shock.. Abdominal pain is a frequent complaint shortly after onset. Shock is characterised by a rapid weak pulse with narrowing of the pulse pressure (20 mm Hg or less, regardless of the pressure levels) or hypotension with cold clammy skin and restlessness. Without prompt appropriate treatment, patients may pass into profound shock, blood pressure and pulse becoming imperceptible. The duration of shock is short, resulting in death within 12-24 hours, or rapid recovery with appropriate antishock therapy. Uncorrected shock may give rise to a more complicated course with metabolic acidosis, severe gastro-intestinal bleeding and a poor prognosis. Convalesence in DHF with or without shock is short and uneventful, with a rapid recovery within 2-3 days. Return of appetite is a good prognostic sign. Bradycardia or sinus arrhythmia are occasionally seen; absolute bradycardia is an occasional finding.

Although the institution of reticulated water supplies with the concomitant disappearance of domestic rain-water tanks has reduced the incidence of Ae. aegypti in many areas, mosquito populations still exist to sustain local transmission. In April 1980, biting Ae. aegypti which had bred in the water-filled holders of pots containing decorative tropical plants were collected in Townsville airport passenger lounge.[12] Also the high potential for dengue transmission on Thursday Island was predicted after a mosquito investigation of the Torres Strait islands following an indigenous malaria outbreak on Moa Island[13] (P.I. Whelan, Northern Territory Department of Health, unpublished observations). Since Thursday Island is a prawn boat base, it was also recognized that the Aedes eggs could be transported to other regions via inter-island traffic. Table 2 summarizes the findings of that survey.

TABLE 2 Mosquito investigations on several Torres Strait Islands - January, 1980

Island	Larvae collections			Biting mosquitoes		
	A	B	C	A	B	C
Thursday	+	+	-	+	+	-
Sabai	-	+	-	-	-	-
Horn	ND	ND	ND	-	-	-
Yam	ND	ND	ND	-	-	-
Darnley	+	-	-	-	-	-
Moa	-	+	+	-	+	+

A. Ae. aegypti - vector of dengue fever.
B. C. annulirostris - vector of Australian encephalitis and epidemic polyarthritis
C. An. farauti - vector of malaria[16]
ND = not done; + = positive finding; - = negative finding;

In the Northern Territory, Ae. aegypti has only been found on one occasion since the commencement of surveys in 1974 (P.I. Whelan, personal communication), but the population movement to rural blocks and re-introduction of rain-water tanks could change this situation. In addition, Ae. katherinensis, which is endemic in the region, appears to be a good vector for dengue virus[14]. The presence of Ae. scutellaris scutellaris in north Queensland, a known dengue vector in Papua New Guinea[1], could also change the epidemiology of the disease. There is no evidence of Ae. aegypti in New South Wales from the results of a four year surveillance program (1976-80) conducted by the Commonwealth Institute of Health, Sydney, the Australian Army Preventive Medicine Unit and the Health Commission of New South Wales (R. Russell, Commonwealth Institute of Health, personal communication).

To be effective, Ae. aegypti control operations should begin when the first dengue cases are detected, or when there are sound reasons for anticipating an outbreak[15]. In areas where virus activity is already known to exist, it is not necessary to have laboratory confirmation.

A technical bulletin entitled "Guide for diagnosis, treatment and control of dengue haemorrhagic fever" (1980), has been published by, and is available from, the WHO Regional Offices for S.E. Asia and Western Pacific.

References

1. CDI (1980) 80/22:2
2. WER (1981) 56:237
3. CDWR (1981) 7:203
4. Monthly CD Notes (1981) 9/81 WHO, Regional Office for the Western Pacific
5. Berita Epidemiologi, Republik Indonesia. March 1981.
6. CDI (1981) 81/15:1
7. CDI (1981) 81/21:1
8. Prog. Med. Virol. (1974) 17:136
9. CDI (1981)81/14:1
10. Aust. J. Exp. Biol. Med. (1973) 51:131
11. CDI (1979) 79/20:3
12. MJA (1980) 2:676
13. CDI (1980) 80/2:5
14. Dengue Newsletter (1977) 5:South Pacific Commission
15. Epidemiological Bulletin, Pan American Health Organization (1981) 2/4:1
16. Roy. Soc. Trop. Med & Hyg. (1980) 74:830

HISTOPLASMOSIS IN NORTH QUEENSLAND

(Contributed by R.W. Guard and R.M. Symes, Commonwealth Health Laboratory, Cairns).

In the past four years, Histoplasma capsulatum infection has been diagnosed in four patients with no history of travel outside far north Queensland. These included two retired farmers, both living on the Endeavour River at Cooktown, a manager of a tyre factory in Cairns who gave a history of cleaning out bird droppings from a warehouse 18 months prior to his illness, and a farmer from Tolga on the Atherton Tableland. All these male patients were over 50 years of age and developed the progressive invasive form of the disease. Presentation included ulcers on the mouth, tongue, tonsils and/or larynx.

Diagnosis was made by histological examination of biopsy material, and was confirmed by culture in three cases. Serology performed at CDC, Atlanta, was completely negative for two cases; this emphasises its unreliability as a sole method of diagnosis[1,2,3].

One of the patients suffered four recurrences of the disease over four years and developed Addison's disease despite repeated courses of amphotericin B. Although the mucous membrane lesions cleared following therapy, new mucosal lesions reappeared after cessation of treatment. After this patient died of an unrelated cause, abcesses with H. capsulatum were found in his adrenals and kidneys. Presumably therapy was never able to eradicate the fungus at these sites, and relapses followed spread from these foci.

The present cases are the first documented from north Queensland, although one previous report was of a patient who had lived in Townsville until six months prior to his illness.[4] Previous reports of histoplasmosis in Australia have included ten cases of the invasive form and 16 cases of the benign inhalation form. The origin of these infections were considered to be Orange in New South Wales[5], Sydney[6,7], Brisbane[1,4], Rockhampton[8], Church Cave in southern New South Wales[9] and Mt. Lofty Range near Adelaide[10].

Ulceration of the upper gastrointestinal or respiratory tract appears to be the commonest form of presentation within Australia, and contrasts with the American experience where only about one-third of cases present with extra-pulmonary pathology. It has been suggested that there are morphological differences between varieties of H. capsulatum from S.E. Asia and America[11].

Editorial Comment

Histoplasma capsulatum is a dimorphic, saprophytic soil fungus, mycelial in its native state but a yeast at the body temperature of mammals. Human infection ordinarily results from inhalation exposure to airborne spores. These inhaled spores may reach the alveoli where they germinate releasing yeast forms, and replicate in the macrophages following ingestion. This proliferation may extend to regional lymph nodes until specific cellular immunity develops. The vast majority of human infections are asymptomatic, although the fungus may induce structural defects of the lung (chronic pulmonary histoplasmosis), and a less common opportunistic disseminated infection through immune defects yet to be established[12]. H. capsulatum is endemic in scattered areas of the world's temperate zones, and in at least 31 of the 48 contiguous states of the U.S.A. Point sources of infection usually exist in sheltered areas where avian and bat excreta have enriched the soil.

References

1. MJA (1968) 1:765
2. MJA (1970) 2:27
3. MJA (1971) 2:421
4. MJA (1953) 1:138
5. MJA (1948) 2:518
6. MJA (1953) 1:142
7. Aust. N.Z. J. Med. (1980) 10:563
8. MJA (1959) 2:640
9. MJA (1976) 2:243
10. Aust. Ann. Med. (1970) 19:151
11. Ann. Soc. belge Med. Trop. (1972) 52:435
12. Medicine (1981) 60:231

CORRIGENDUM - CDI 81/21

In the article "Epidemic polyarthritis and Ross River (RR) virus infection" the data presented on page 2 on antibody titres following infection are misleading. The paragraph should read:

"Specific HI antibody against RR virus is considered positive at titres of $\geq$ 1/20, but these levels may indicate infection years before. Titres are often high at presentation, even at two days after onset. Four-fold rises in titre from serial samples are diagnostic. Antibody titres usually decay slowly, and remain static for months when initial titres are moderate. An initial titre of 1/1280 or higher strongly suggests recent infection, as does the presence of IgM antibody, but the latter can persist for long periods and might represent an earlier symptomless infection unrelated to the presenting illness.".

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 2

REPORTING PERIOD - 29/10/81 - 11/11/81 BULLETIN NUMBER - 81/23

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
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	3			1		1		1	6
1303 VARICELLA-ZOSTER VIRUS	1		5			3		1	10
1306 HERPES SIMPLEX TYPE 1	13			18		20	14		65
1307 HERPES SIMPLEX TYPE 2	40			21		24	23		108
1399 HERPES VIRUS TYPING PENDING			6			3			9
1401 COXIELLA BURNETI	8			1		4	5		18
1502 PICORNA VIRUS-NOT TYPED								1	1
1521 MEASLES VIRUS	5	5	6	4		2			22
1522 RUBELLA VIRUS	3			8		3	26	2	42
1532 HEPATITIS B ANTIGEN	11		3	31		14	5	10	74
1535 HEPATITIS A ANTIBODY	5		3	6		3	3	6	26
1541 CHLAMYDIA A - C TRACHOMATIS	31		4			2		55	92
1556 CMV - CYTOMEGALOVIRUS	1	1	5	12		2		6	27
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS	5	8	3	12		5	2	2	37
1599 ENTEROVIRUS TYPING PENDING		1	7			1			9
ROSS RIVER VIRUS							10		10
SMALL VIRUS (LIKE) PARTICLE	3			1					4
DENGUE							5		5
PARAMYXO	1					2			3
Total	188	29	68	164		133	135	136	853

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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REPORTING PERIOD - 29/10/81 - 11/11/81 BULLETIN NUMBER - 81/23

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			1		3	7	1	15
0101 ADENOVIRUS TYPE 1	3		1					1	5
0102 ADENOVIRUS TYPE 2	3			3		2		3	11
0103 ADENOVIRUS TYPE 3	2			6		1			9
0105 ADENOVIRUS TYPE 5						1			1
0119 ADENOVIRUS TYPE 19	1			3		1			5
0199 ADENOVIRUS TYPING PENDING		1				3			4
0201 INFLUENZA A VIRUS	1		7						8
0203 INFLUENZA B VIRUS	1		1	1		2			5
0301 PARAINFLUENZA VIRUS TYPE 1				1			1		2
0303 PARAINFLUENZA VIRUS TYPE 3	4		1	3		7	5	1	21
0399 PARAINFLUENZA VIRUS TYPING PENDING						5			5
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	3	4				1	3	5	16
0500 RHINOVIRUS (ALL TYPES)	1	1		9			13		24
0600 MYCOPLASMA PNEUMONIAE	9		4	2		2	6	2	25
0700 ORNITHOSIS-PSITTACOSIS	3								3
0809 COXSACKIEVIRUS A9				3			1		4
0904 COXSACKIEVIRUS B4		5					1	1	7
0905 COXSACKIEVIRUS B5						1	1		2
1014 ECHOVIRUS TYPE 14				1					1
1017 ECHOVIRUS TYPE 17							2		2
1022 ECHOVIRUS TYPE 22			1						1
1025 ECHOVIRUS TYPE 25								2	2
1030 ECHOVIRUS TYPE 30				2					2
1100 POLIOVIRUS NOT TYPED						1			1
1101 POLIOVIRUS TYPE 1								2	2
1102 POLIOVIRUS TYPE 2						1		1	2
1103 POLIOVIRUS TYPE 3						1	1		2
1104 POLIOVIRUS-VACCINAL STRAIN	1		2						3
1200 MUMPS VIRUS	4			7		1	1	1	14
1300 HERPES VIRUS GROUP-NOT TYPED	19		9	4		11			43
1301 HERPES SIMPLEX VIRUS NOT-TYPED		3		2				32	37

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

3

PERIOD : 29/10/81 to 11/11/81 ....

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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.

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					3				
0102 ADENOVIRUS TYPE 2.....		5		1			3				
0103 ADENOVIRUS TYPE 3.....	1	1					1				2
0105 ADENOVIRUS TYPE 5.....							1				
0201 INFLUENZA A VIRUS.....	1	7									
0203 INFLUENZA B VIRUS.....	1	2									1
0301 PARAINFLUENZA VIRUS TYPE 1....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....	4	17				1					
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	14									1
0500 RHINOVIRUS (ALL TYPES).....	2	20									
0600 MYCOPLASMA PNEUMONIAE.....	3	16									1
0700 ORNITHOSIS-PSITTACOSIS.....		1									1
0809 COXSACKIEVIRUS A9.....	1			3							
0904 COXSACKIEVIRUS B4.....		5		1					1		
0905 COXSACKIEVIRUS B5.....							1				
1014 ECHOVIRUS TYPE 14.....				1							
1017 ECHOVIRUS TYPE 17.....	2										
1022 ECHOVIRUS TYPE 22.....							1				
1025 ECHOVIRUS TYPE 25.....						1					
1030 ECHOVIRUS TYPE 30.....				1							
1101 POLIOVIRUS TYPE 1.....	1	1									
1102 POLIOVIRUS TYPE 2.....	1	1									
1103 POLIOVIRUS TYPE 3.....	1						1				
1104 POLIOVIRUS-VACCINAL STRAIN....							3				
1200 MUMPS VIRUS.....	2	1		3							1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	1	1			1			1	1	24
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		1						2			

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 29/10/81 to 11/11/81 ....

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 other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
1303 VARICELLA-ZOSTER VIRUS.....	1		1	1						2	4
1306 HERPES SIMPLEX TYPE 1.....	2	9	1								33
1307 HERPES SIMPLEX TYPE 2.....	1										5
1401 COXIELLA BURNETI.....	6	1									
1521 MEASLES VIRUS.....		2									17
1522 RUBELLA VIRUS.....	4										34
1532 HEPATITIS B ANTIGEN.....	38							35			1
1535 HEPATITIS A ANTIBODY.....								26			
1556 CMV - CYTOMEGALOVIRUS.....	1	3	1	1			1	3		6	
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....	1						35	1			
ROSS RIVER VIRUS	6										
SMALL VIRUS (LIKE) PARTICLE	1						3				
DENGUE	1										3
PARAMYXO	1	2									
Total.....	85	113	4	12		3	54	67	2	9	128

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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PERIOD : 29/10/81 to 11/11/81 ...
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
0101 ADENOVIRUS TYPE 1.....								1		
0102 ADENOVIRUS TYPE 2.....			1		1		2	1		
0103 ADENOVIRUS TYPE 3.....	3		1					1		
0119 ADENOVIRUS TYPE 19.....	5									
0203 INFLUENZA B VIRUS.....								2		
0500 RHINOVIRUS (ALL TYPES).....								2		
0600 MYCOPLASMA PNEUMONIAE.....					1			3	1	
0700 ORNITHOSIS-PSITTACOSIS.....									1	
0905 COXSACKIEVIRUS B5.....										1
1025 ECHOVIRUS TYPE 25.....								1		
1030 ECHOVIRUS TYPE 30.....								1		
1200 MUMPS VIRUS.....			10							
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	11						1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			1	1			2			
1303 VARICELLA-ZOSTER VIRUS.....		1								
1306 HERPES SIMPLEX TYPE 1.....		22						3		
1307 HERPES SIMPLEX TYPE 2.....		102								
1401 COXIELLA BURNETI.....							5	6	1	
1502 PICORNA VIRUS-NOT TYPED.....						1				
1521 MEASLES VIRUS.....			1					2	1	
1522 RUBELLA VIRUS.....					15	1				
1541 CHLAMYDIA A - C TRACHOMATIS...	2	90								
1556 CMV - CYTOMEGALOVIRUS.....		4				5	4		1	
ROSS RIVER VIRUS					4					
DENGUE					1					
Total.....	11	230	14	1	22	7	13	24	5	1