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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 1505 reports were received this period.

. Arbovirus infections - Epidemic polyarthritis activity was reported from all eastern mainland States (91 reports received compared with 39 (two periods), 15 and 16 for the previous four periods). The cases, exhibiting an age distribution of 8-62 years (mean 33.6 years ± 11.5) and a male: female ratio of 1:1.67, emanated from Queensland (20; Brisbane-5, Goondiwindi-3, Cairns-2, Townsville-2, Toowoomba-2, Weipa-1, Moree-1, Tara-1, Cecil Plains-1, Gladstone-1, Kowanyama-1), Northern Territory (1; Darwin), New South Wales (30; Sydney-6, Albury-2, Jerilderie-1, Deniliquin-1, Dubbo-1, Corowa-1, unspecified-18 with 16 sera referred from the same pathology laboratory with locations at Tamworth, Leeton and the South Coast), and Victoria (20; Cohuna-6, Swan Hill-5, Mildura-4, Shepparton-3, Echuca-1, Horsham-1). The 19 reports from the Institute of Medical and Veterinary Science, Adelaide, were of sera referred from Menindee (New South Wales) and from locations along the River Murray.

Physicians at Griffith have recently been identifying two separate clinical entities of arbovirus disease - the classical epidemic polyarthritis with sudden onset of muscle and joint pains (principally ankles, knees, fingers and wrists), malaise and headache usually followed by a maculopapular rash in the majority of patients; and a less specific syndrome of intense headache and extreme lethargy, sometimes accompanied with nausea and myalgia, lasting one week, but without manifestations of rash or polyarthritis. Sera from three such cases have been forwarded to the University of New South Wales, Sydney, for serological diagnosis. Paired sera from one patient from Griffith, who presented with some signs of meningitis and extreme fatigue, exhibited a broad cross-reactivity against both flavivirus (Murray Valley encephalitis, Alfuy, Stratford, Sumarez Reef) and representative alphavirus antigens. These sera are being tested further for IgM and neutralisation antibody activity. Two other sera (similar cases from Griffith and Wee Waa) have also exhibited high, but stationary, cross-reacting titres against representative flavivirus antigens.

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INFLUENZA ENCEPHALITIS - VICTORIA

(Contributed by H. Newton-John, Fairfield Hospital, Melbourne).

On 9 October 1983, a previously-well 14 year old schoolboy with a history of eczema, was admitted to Fairfield Hospital with an altered conscious state. Five days previously, the boy complained of a sore neck, followed by a sore throat 24 hours later. Although the illness did not interrupt his activities, his parents noted that he was retiring to bed early. On the fourth day he fainted briefly during a cricket match, but he was able to take himself home and explain the incident. That evening he was unable to speak yet he understood his family's requests. The same day he was prescribed penicillin by his general practitioner. On the fifth day he spent most of the time in bed, but executed bursts of seemingly purposeless activity, e.g. having a shower and returning to bed without drying himself. Overall, he was vague and staring. He understood spoken words, but did not respond. He had no history of any drugs, glue sniffing or marihuana use.

On admission the child was afebrile, but with an altered state of consciousness - merely nodding his head in response to questions. He showed some co-operation with the examination, obeying commands initially but unable to sustain those instructions. Although he uttered no verbal response, he made some mouthing actions with puffed out cheeks. He masturbated constantly. His gait was unsteady, but no focal neurological abnormality was apparent. His throat was reddened without exudate. Both ears were normal, his chest clear and his cardiovascular system regular. A papular rash was evident on the anterior chest wall.

A lumbar puncture was performed with difficulty, providing only sufficient clear CSF for cell count (12 leucocytes/ml; 9% polymorphs, 91% lymphocytes) and viral culture (negative). As the cell count was suggestive of encephalitis, the patient was given supportive care, a CT scan and an EEG. The EEG trace was diffusely abnormal with paroxysms of slow wave activity (this pattern is not normally associated with encephalitis). Acyclovir (10 mg/kg/dose) was administered three times daily for five days. His condition remained static, but improved gradually after five days with a return of speech and lengthening periods of normal behaviour. A repeat lumbar puncture on 17 October gave 20 leucocytes (41% polymorphs; 59% lymphocytes), and an EEG on 21 October showed only minor abnormalities in the form of occasional paroxysms of theta activity. The boy was discharged on 24 October with a restoration of his normal conscious state and good intellectual function but reduced concentration span. He was able to recall his illness 'as if trying to wake out of a dream'. He stated he was aware of his abnormal behaviour at times, but was unable to exert any control.

No bacterial pathogens were cultured from blood or nasal and throat swabs, but herpes simplex type 1 and an influenza A (H_1N_1) strain resembling A/England/330/80 were isolated from the nose and throat. However a diagnostic rise in antibody titre was evident only against influenza; titres against hepatitis B (RIA), syphilis (RRP and TPHA) and Epstein-Barr virus (Paul Bunnell) were negative, and those against influenza B, Mycoplasma pneumoniae and herpes simplex (ELISA) were stationary.

Although cases of transverse myelitis and encephalitis following influenza infection have been reported[1,2], a

definite aetiological relationship has not been proved. Nevertheless, this case history and laboratory findings are suggestive of an influenza encephalitis.

References

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HAEMORRHAGIC FEVER WITH RENAL SYNDROME

Haemorrhagic fever with renal syndrome (HFRS) has been described previously under various names e.g. Korean haemorrhagic fever, epidemic haemorrhagic fever, nephropathia epidemica; benign nephropathy, epidemic nephritis, haemorrhagic nephroso-nephritis etc. in various parts of the world. The disease is now recognized to be an asymptomatic infection of wild rodents (rats, mice and voles), and is a major public health problem throughout most of the European and Asian land mass. The virus responsible, Hantaan, produces sporadic disease, but under special circumstances occurs in epidemic form. Although predominantly associated with rural areas, it is now being acknowledged as an urban problem in some countries, and a particular hazard to laboratory staff using rodents for biomedical research.

HFRS was noted in Asia in the early 1930's, but came dramatically to the attention of Western medicine when between 1951-54 more than 3 000 United Nations troups stationed in the demilitarized zone in Korea developed a rare disease not previously recognized[1]. The illness was characterised by fever, headache, severe abdominal and back pain, a flushed face and various haemorrhagic manifestations (see Table 1). Between 5-10% of those affected died of shock and renal failure. Since that time the disease has gradually moved southward, so that it is now widespread among both military and civilian populations in rural and urban areas. Cases of HFRS have also been recorded in urban and rural areas of 23 provences of China (30,000 cases in 1980 with 6.4% mortality rate), and in 1960 an outbreak occurred in a limited area of Osaka City, Japan, when it was attributed to the importation of infected rodents. The disease continued to occur for approximately ten years, and 119 cases were recorded. In Eastern Europe a major outbreak of HFRS occurred in Bosnia-Hacogoxina in 1967 when 114 people became ill and three died. In Norway, Sweden and Finland, a similar but milder form of HFRS called nephropathia epidemica (NE) has been known since 1934, but haemorrhagic manifestations with this infection are rare and the mortality is $< 1\%$[2].

The demonstration of a specific antigen in the lungs of the wild Asian rodent Apodemus agareus coreae in 1976[3], followed by the adaptation of the aetiological agent, Hantaan virus, a member of the Bunyaviridae[4,5], to grow in continuous cell lines (A-549, human lung alveolar epithelium[6]; Vero-E-6, cloned African green monkey kidney) provided the necessary impetus and sources of antigen to allow study and diagnosis of HFRS in laboratories outside Korea. Similarly the presence of an antigen - the Puumala agent - in the lungs of Finnish bank voles, Clethrionomys glareolus, has allowed the development of an immunofluorescent test for detecting antibody against NE[7]. Despite extensive attempts, human strains of this Scandanavian agent have not been isolated in voles or in cell culture. Simultaneous testing between these European and East Asian viruses has

TABLE 1 Clinical features of HFRS

(Based on "WHO Working Group on Haemorrhagic Fever with Renal Syndrome", Tokyo, Japan, 22-24 February 1982).

Most of the information on the clinical features of HFRS comes from studies of military cases during the Korean War. The disease varies greatly in severity, and although occasionally the illness is so mild as to make diagnosis difficult, all patients exhibit proteinuria and many show petechiae and some degree of haemoconcentration, hypotension and renal failure. Only about 20% of the patients exhibit such severe consequences as shock, serious haemorrhages and marked fluid and electrolyte imbalances. The clinical course and the important laboratory features of a case of moderate severity can be arbitarily divided into five phases; febrile, hypotensive, oliguric, diuretic and convalescent.

Febrile phase - The onset is usually abrupt and consists of chills, fever, lethargy and weakness. Severe frontal and retro-orbital headache and abdominal and lumbar pain are frequent features. There is a characteristic facial flush, injection of the conjunctive and widespread appearance of petechiae. Proteinuria appears on days 3-5, and is accompanied by a decrease in platelet count and a progressive leucocytosis. After about five days of illness, hypotension or shock may occur.

Hypotensive phase - Most of the symptoms and signs of the febrile phase remain, although the patients' headache frequently subsides. Apprehension, restlessness may appear, and later confusion, delerium and coma. About one third of the deaths occur at this stage. Massive proteinuria persists, the haemocrit rises and the urine specific gravity drops to 1.010. Ecchymoses, haemoptysis, haematuria, haematemesis and melena may occur at this stage as platelet counts continue to fall.

Oliguric phase - Over the next 3-4 days, oliguria becomes a prominent feature of the disease, blood nitrogen retention increases rapidly, and various electrolyte abnormalities may develop. Hypervolemia may occur, and cause death as a result of cerebral or pulmonary complications.

Diuretic phase - This phase may last for several weeks and is associated with a rapid improvement in renal function. Some patients fluctuate between shock and hypotension, with pulmonary oedema and severe electolyte imbalance may occur. About one third of deaths occur in this stage associated with shock and pulmonary complications.

Convalescent phase - During this phase, which may last from three weeks to three months, recovery is complete. While the mortality of untreated cases may be in excess of 15%, with modern medical treatment, including dialysis, it is usually $< 5\%$. Survivors, with the exception of those who have had haemorrhages into the CNS, usually make a complete recovery, and long-term sequelae are rare.

A similar but mild form of HFRS occurs in Scandanavia; the haemorrhagic features are scanty and the mortality is $< 0.5\%$. The disease has an acute onset with fever, headache, nausea and vomiting. Occasionally the predominant symptom may suggest hepatitis, carditis or meningoencephalitis. After 3-6 days, backache and abdominal pain become important features, and the patient develops proteinuria and oliguria. There is usually microscopic haematuria and moderate thrombocytopenia. The oliguria persists for a few days and is followed by polyuria as the patient's condition improves rapidly. The illness usually lasts for about three weeks and sequelae are uncommon.

indicated a one-way antigenic relationship[8,9], although convalescent sera from some European cases have exhibited heterologous activity suggesting that both viruses may exist in certain European regions, and that other serotypes or subtypes of HFRS viruses occur[10]. A third Hantaan - related virus (Prospect Hill agent) has been isolated from the lungs of North American native meadow mice, Microtus pennsylvanicus[11], but the role of this virus in human infection has yet to be defined.

Three epidemiological types of HFRS occur, each with a separate reservoir host.

Rural type - The majority of cases still occur in rural areas, where in Korea there are two seasonal peaks of activity (June and September - October). No secondary cases or evidence of person-to-person transmission have been documented, although many investigators working on the disease particularly those handling infected rodents, have developed HFRS. The reservoir host in rural Korea and much of China and the USSR are mice of the Apodemus species. Seasonal peaks of disease coincide with peak numbers and peak infection rates of Apodemus mice in the countryside. Farmers working in the fields during these periods come into contact with infective excreta either by the inhalation of aerosols or by direct inoculation through cuts and scratches in the skin. Infected animals may excrete the virus in saliva, urine and faeces for periods up to two years. In the Soviet Union, HFRS antigen has been detected in the lungs of ten different species of rodent trapped in different geographical regions, the highest infection rates being the urban rat Rattus norvegicus (25%) and the bank voles C. glareolus (12.8%). In Finland, the majority of cases occur in the rural areas around Lake Finland north of the 60th parallel, where the disease has a marked peak during November-January and the natural reservoir is C. glareolus. Recently two HFRS cases have been reported from France implicating exposure to wild rodents in a village barn 120 km north-east of Paris[12,13].

Urban type - In recent years cases of HFRS have been recorded in urban areas of Korea, China[14] and Japan where the reservoir of infection has appeared to be the household rat R. norvegicus. Antibodies to the Hantaan virus have also been found in urban rats in the USA[15], but no human cases of muroid virus nephropathy have been recognized to date.

Laboratory infections - Laboratory infections have occurred among medical and other personnel involved in typing and handling wild rodents, and people using laboratory-bred rodents for biomedical research[16,17,18]. Since 1975, 195 cases have been reported from 16 institutions in Japan, and outbreaks have occurred in laboratories in Moscow, Helsinki, Belgium and Seoul where certain strains of inbred rodent have been found to be chronically infected with Hantaan virus. These findings have had direct bearing on safety practices in animal laboratories throughout the world. The report of the Working Group on Haemorrhagic Fever with Renal Syndrome, Tokyo, Japan, 22-24 February 1982 recommended that specimens from potentially infected humans should be handled with great care, preferably in a facility which provides a high degree of operator protection. Studies with infected animals (trapping, bleeding, autopsies, inoculations) and the passage of strains in tissue culture require extreme precautions to protect laboratory personnel. In addition, laboratory rodents used for biomedical research should be housed in quarters which prevent contact with wild rodents, and breeding colonies tested regularly for

absence of infection with HFRS. Australian quarantine legislation already requires that all rats be monitored for Hantaan-like virus before importation.

The clinical spectrum of human responses to Hantaan viral infection is very variable. Antibodies to HFRS virus in sera from healthy people have been detected frequently in HFRS endemic regions and elsewhere[19,20], and some antibody carriers do not recall any symptoms of HFRS in their medical histories. Although no infections have so far been recorded in Australia, the lack of knowledge concerning the global extent of human disease and the potential to cause atypical or silent infections raises the possibility of the occurrence of missed clinical cases. Limited facilities to test convalescent sera for antibodies are available at Fairfield Hospital, Melbourne. Antibody against Hantaan virus has already been demonstrated in sera from two of ten R. norvegicus trapped by staff of Fairfield Hospital at various locations along the wharves of Melbourne dockyard in mid-1983 (L. Irving, personal communication). At present, rat sera are referred to K.M. Johnson, US Army Medical Research Institute of Infectious Diseases, Frederick, Maryland, for all laboratory testing. Animal trapping is being continued by officers of the Victorian Health Commission, but no additional positive specimens have been identified to date.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 5/1/84 - 18/1/84 BULLETIN NUMBER 84/2

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	2	2	2		3	4	3	1	17
0101 ADENOVIRUS TYPE 1	3					3		2	8
0102 ADENOVIRUS TYPE 2	1					2		4	7
0103 ADENOVIRUS TYPE 3	1			8					9
0104 ADENOVIRUS TYPE 4	1								1
0105 ADENOVIRUS TYPE 5		2				1			3
0106 ADENOVIRUS TYPE 6	2								2
0107 ADENOVIRUS TYPE 7	1								1
0108 ADENOVIRUS TYPE 8	1								1
0113 ADENOVIRUS TYPE 13								1	1
0118 ADENOVIRUS TYPE 18						1			1
0137 ADENOVIRUS TYPE 37								12	12
0199 ADENOVIRUS TYPING PENDING					2	1			3
0201 INFLUENZA A VIRUS	4								4
0203 INFLUENZA B VIRUS	1							1	2
0301 PARAINFLUENZA VIRUS TYPE 1	2				2	1			5
0303 PARAINFLUENZA VIRUS TYPE 3	4	3		3	11	13	6	1	41
0399 PARAINFLUENZA VIRUS TYPING PENDING						1			1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1				1	1	1		4
0500 RHINOVIRUS (ALL TYPES)	2			2	5	13	7		29
0600 MYCOPLASMA PNEUMONIAE	135			31	10	40	16	1	233
0700 ORNITHOSIS-PSITTACOSIS	4			2				1	7
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED								1	1
0816 COXSACKIEVIRUS A16							2		2
0901 COXSACKIEVIRUS B1						2			2
0902 COXSACKIEVIRUS B2	2					4		1	7
0903 COXSACKIEVIRUS B3	1					1			2
0905 COXSACKIEVIRUS B5	1								1
1000 ECHOVIRUS NOT TYPED	1								1
1006 ECHOVIRUS TYPE 6				2					2
1008 ECHOVIRUS TYPE 8								1	1
1009 ECHOVIRUS TYPE 9	1			1			1		3
1011 ECHOVIRUS TYPE 11	1			1					2
1017 ECHOVIRUS TYPE 17	3	1							4
1018 ECHOVIRUS TYPE 18	1								1
1101 POLIOVIRUS TYPE 1								1	1
1102 POLIOVIRUS TYPE 2	2								2
1103 POLIOVIRUS TYPE 3	1								1
1104 POLIOVIRUS-VACCINAL STRAIN			2						2
1200 MUMPS VIRUS	8		1	6					15
1300 HERPES VIRUS GROUP-NOT TYPED	27			6		2		2	37
1301 HERPES SIMPLEX VIRUS NOT-TYPED		5		5					10
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	12	1		1				10	24
1303 VARICELLA-ZOSTER VIRUS	6					1		1	8
1306 HERPES SIMPLEX TYPE 1	13	1	15	21		31	38	21	140
1307 HERPES SIMPLEX TYPE 2	117		7	36		12	55	55	282
1399 HERPES VIRUS TYPING PENDING			9	1	4	1	1		16
1401 COXIELLA BURNETI	9			3			13		25
1502 PICORNA VIRUS-NOT TYPED	14		3						17
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS	2			6	1	1		4	14
1522 RUBELLA VIRUS	31					3	15	1	50
1532 HEPATITIS B ANTIGEN	25		6	27		10	12	11	91
1535 HEPATITIS A ANTIBODY	3			5	1	3	7	10	29
1541 CHLAMYDIA A - C TRACHOMATIS	37						28	52	117
1556 CMV - CYTOMEGALOVIRUS	10			23	3		2	6	44
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	1	4	11	1	15	13			45
1599 ENTEROVIRUS TYPING PENDING			12		5	1			18
9901 ARBO. GROUP A.(UNSPECIFIED)				20					20
9992 ROSS RIVER VIRUS	15		1			19	34	1	70
9994 SMALL VIRUS (LIKE) PARTICLE				1					1
9995 DENGUE							1		1
9996 PARAMYXOVIRUS						1			1
9997 KUNJIN VIRUS							1		1
Total	509	19	69	212	63	186	243	204	1,505

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 5/1/84 to 18/1/84 .... 84/2

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.....							1				
0101 ADENOVIRUS TYPE 1.....		4					1				
0102 ADENOVIRUS TYPE 2.....		4				1					
0103 ADENOVIRUS TYPE 3.....		2		1							
0104 ADENOVIRUS TYPE 4.....		1									
0105 ADENOVIRUS TYPE 5.....		1					1				
0106 ADENOVIRUS TYPE 6.....		1					1				
0118 ADENOVIRUS TYPE 18.....							1				
0201 INFLUENZA A VIRUS.....	1	2							1		
0203 INFLUENZA B VIRUS.....		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		5									
0303 PARAINFLUENZA VIRUS TYPE 3....		34	2				1				1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		4									
0500 RHINOVIRUS (ALL TYPES).....		28									
0600 MYCOPLASMA PNEUMONIAE.....	39	178									
0700 ORNITHOSIS-PSITTACOSIS.....	2	4									
0816 COXSACKIEVIRUS A16.....											2
0901 COXSACKIEVIRUS B1.....							1				
0902 COXSACKIEVIRUS B2.....	1	3									
0903 COXSACKIEVIRUS B3.....		2									
0905 COXSACKIEVIRUS B5.....	1										
1000 ECHOVIRUS NOT TYPED.....			1								
1006 ECHOVIRUS TYPE 6.....				2							
1008 ECHOVIRUS TYPE 8.....						1					
1009 ECHOVIRUS TYPE 9.....				2							
1011 ECHOVIRUS TYPE 11.....		1									
1017 ECHOVIRUS TYPE 17.....				2			2				
1102 POLIOVIRUS TYPE 2.....	1										
1103 POLIOVIRUS TYPE 3.....	1										
1104 POLIOVIRUS-VACCINAL STRAIN....							2				
1200 MUMPS VIRUS.....		2	2	3							
1300 HERPES VIRUS GROUP-NOT TYPED..	1										
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1	4								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	5					1	1	2	1		
1303 VARICELLA-ZOSTER VIRUS.....	1										6
1306 HERPES SIMPLEX TYPE 1.....	8	2	1	1							75
1307 HERPES SIMPLEX TYPE 2.....	4										57
1399 HERPES VIRUS TYPING PENDING...	1										
1401 COXIELLA BURNETI.....	6	1									
1502 PICORNA VIRUS-NOT TYPED.....							1				1
1514 MOLLUSCUM CONTAGIOSUM.....	1										
1521 MEASLES VIRUS.....	1	2									11
1522 RUBELLA VIRUS.....	10			1							33
1532 HEPATITIS B ANTIGEN.....	39							42		1	
1535 HEPATITIS A ANTIBODY.....	3							24			
1556 CMV - CYTOMEGALOVIRUS.....	10	2			1					7	
1564 ROTAVIRUS.....							45				
9901 ARBO. GROUP A.(UNSPECIFIED)...	7										12
9992 ROSS RIVER VIRUS.....	24										19
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	167	285	10	12	1	3	59	68	2	8	218

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 5/1/84 to 18/1/84 ... 84/2

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.....	2								1	
0102 ADENOVIRUS TYPE 2.....							1		1	
0103 ADENOVIRUS TYPE 3.....								6		
0105 ADENOVIRUS TYPE 5.....									1	
0107 ADENOVIRUS TYPE 7.....	1									
0108 ADENOVIRUS TYPE 8.....	1									
0113 ADENOVIRUS TYPE 13.....	1									
0137 ADENOVIRUS TYPE 37.....	1	11								
0203 INFLUENZA B VIRUS.....							1			
0303 PARAINFLUENZA VIRUS TYPE 3....	1							2	2	
0500 RHINOVIRUS (ALL TYPES).....								1	1	
0600 MYCOPLASMA PNEUMONIAE.....			2				14	13	4	
0700 ORNITHOSIS-PSITTACOSIS.....								1		
0901 COXSACKIEVIRUS B1.....							1			
0902 COXSACKIEVIRUS B2.....							2	1		
1006 ECHOVIRUS TYPE 6.....								1		
1009 ECHOVIRUS TYPE 9.....									1	
1011 ECHOVIRUS TYPE 11.....							1	1		
1018 ECHOVIRUS TYPE 18.....								1		
1101 POLIOVIRUS TYPE 1.....										1
1102 POLIOVIRUS TYPE 2.....							1			
1200 MUMPS VIRUS.....			8					1	1	
1300 HERPES VIRUS GROUP-NOT TYPED..		1								
1301 HERPES SIMPLEX VIRUS NOT-TYPED		4								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			10					3	3	
1303 VARICELLA-ZOSTER VIRUS.....							1			
1306 HERPES SIMPLEX TYPE 1.....	6	46			1			1	1	
1307 HERPES SIMPLEX TYPE 2.....		227								
1401 COXIELLA BURNETI.....					1		2	14	1	
1521 MEASLES VIRUS.....			1					2	1	
1522 RUBELLA VIRUS.....			3		6	1		3	2	
1532 HEPATITIS B ANTIGEN.....					1				9	
1535 HEPATITIS A ANTIBODY.....								1	1	
1541 CHLAMYDIA A - C.TRACHOMATIS...		117								
1556 CMV - CYTOMEGALOVIRUS.....		4		1		4	4	4	9	
1563 CORONAVIRUS.....							1			
9901 ARBO. GROUP A.(UNSPECIFIED)...					12			1		
9992 ROSS RIVER VIRUS.....		2			40			1		
9995 DENGUE.....								1		
9997 KUNJIN VIRUS.....					1					
Total.....	13	412	24	1	62	5	29	59	39	1

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

(Weeks 41 - 44)
8 October - 4 November 1983

Bulletin 84/2...

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Amoebiasis	1		1	5	1		1		9	48
Ankylostomiasis				8					8	73
Anthrax									—	—
Arbovirus infection									—	4
Brucellosis	1								1	14
Campylobacter infections	43	N.N.	N.N.	71	N.N.	N.N.	1	N.N.	115	1256
Chancroid				N.N.	1	N.N.	N.N.		1	13
Cholera									—	4
Congenital rubella syndrome		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—
Diphtheria									—	—
Donovanosis		N.N.	11	N.N.	7	N.N.	2		20	98
Giardiasis	11	N.N.	N.N.	50	N.N.	N.N.	N.N.	N.N.	61	805
Genital herpes	80	N.N.	51	3	N.N.	N.N.	1	N.N.	135	1739
Gonococcal ophthalmia neonatorum		N.N.			N.N.	N.N.	1	N.N.	1	14
Gonorrhoea	153	101	131	49	136	5	73	8	656	9417
Hepatitis A (infectious)	12	23	12	16	5		6	4	78	878
Hepatitis B (serum)	30	27	12	16	2			2	89	808
Hepatitis - unspecified	24	1			7	N.N.	N.N.		32	292
Hydatid disease						1			1	8
Lassa Fever			N.N.			N.N.	N.N.	N.N.	—	—
Legionnaires disease	1		N.N.		N.N.	N.N.	N.N.	N.N.	1	21
Leprosy	3	4			1				8	57
Leptospirosis	3	34		4	3	1			45	174
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.			—	7
Malaria	4	5	8	5	1			1	24	492
Marburg Disease			N.N.			N.N.	N.N.	N.N.	—	—
Meningococcal infections	3		1	2		N.N.	2		8	92
Non-specific urethritis	313	N.N.	N.N.	88	N.N.	N.N.	N.N.	N.N.	401	4733
Ornithosis				1					1	18
Pertussis (whooping cough)	11	16	N.N.	3	N.N.	N.N.	1	N.N.	31	273
Plague									—	—
Poliomyelitis									—	—
Q. fever			5		N.N.		N.N.		5	143
Rabies		N.N.	N.N.			N.N.	N.N.	N.N.	—	—

DISEASE	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Salmonella infections	34	8	20	24	1	15	23		125	2645
Shigella infections	4	1	2	1	4		20		32	524
Smallpox									—	—
Syphilis	55	10	35	3	24		59		186	2230
Tetanus									—	7
Trachoma		N.N.	1		N.N.	N.N.			1	5
Tuberculosis (all forms)	45	27	12	12	13		3	1	113	1034
Typhoid fever	2								2	20
Typhus (all forms)	1								1	20
Vibrio parahaemolyticus infections		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—
Yellow Fever									—	—
Yersinia enterocolitica infections		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon voluntary reporting by medical practitioners etc.)

N.N. Not Notifiable

ADJUSTMENTS

Genital Herpes	+	63	NSW
Gonorrhoea	+	105	NSW
	−	9	QLD
Hepatitis A	−	1	SA
Hepatitis Unspecified	+	1	VIC
Legionnaires Disease	+	3	NSW
Leprosy	+	4	NSW
Leptospirosis	+	10	NSW
Ornithosis	+	1	NSW
Non-Specific Urethritis	+	338	NSW
Salmonella Infections	+	1	SA
Syphilis	+	4	NSW
Typhoid Fever	+	1	NSW