AUSTRALIA

Communicable Diseases Intelligence

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CHOLERA IN QUEENSLAND

El Tor cholera (sero-type Inaba) has been confirmed in a 2½ year old boy from the town of Beaudesert in Queensland. He was admitted to Ipswich Hospital acutely ill with diarrhoea and dehydration on February 1, having arrived from Beaudesert the day before. He has now fully recovered. Faecal screening of close contacts revealed that his mother also carried the organism, although she was completely asymptomatic. Following antibiotic therapy no further isolations have been obtained from either of them.

It is presumed that the infection originated from contact with water from the Albert-Logan river system near Beaudesert, although town water supplies taken from this system are all chlorinated. Vibrio cholerae organisms have been isolated from this river system intermittently since they were first discovered in it in 1977, following the occurrence of an infection by the same organism in a woman from Beenleigh (downstream of Beaudesert) in February of that year. The original source of infection of the river system has never been identified.

Very occasional isolations of V. cholerae have also been made from the Brisbane river - a completely different river system.

Editorial comment (contributed by Dr R.G.A. Sutton, Department of Health, Canberra)

The recent case (and possible carrier) of cholera in Queensland, and the continued findings of V. cholerae in the Albert-Logan River system over a period of three years, again brings into the spotlight the question of the epidemiology of cholera in non-endemic areas. Although water has long been considered the major (but not the only) vehicle of cholera transmission, it has usually been accepted that the water is contaminated by faeces or vomitus from human cases or carriers, and that the organism survives in the water for varying periods of time depending on a number of factors including salinity, organic content, temperature, and exposure to sunlight, but in any event rarely longer than one month - and that during this time the water may infect other persons. This

(cont'd on page 6)

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Contributions are solicited, and do not preclude later publication elsewhere.

PRIMARY AMOEBIC MENINGOENCEPHALITIS - Western Australian outbreak.
(Based on a report by Dr Henderson, State Health Laboratories, Nedlands,W.A

Two fatal cases of amoebic meningitis from which Naegleria species have been identified are reported from Princess Margaret Hospital for Children, in Perth, Western Australia. The children were a 7 year old boy from the township of Merredin and a 10 year old girl from Beverley. Diagnosis was based on microbiological and histopathological evidence.

Infection by Naegleria fowleri, the causative organism of primary amoebic meningoencephalitis, is usually associated with contaminated water, particularly in warm environments.

The water supply to these two inland towns is derived from the Goldfields and Agricultural Water Supply System which originates from Mundaring Weir and extends to Kalgoorlie supplying Merredin on its course; branch lines supply other townships including Beverley.

The main is chlorinated as it leaves Mundaring Weir. At various points along its distance there are open storage tanks; these also serve as balance tanks. Re-chlorination is performed at any point between the Weir outlet and the towns' supplies wherever there is an earth-floored reservoir.

Isolates of Naegleria species were made from the mains supply to the Beverley swimming pool and from the backwash of the filter. However the pool water was otherwise notably clean and satisfied W.H.O. standards for potable water. Further isolates have been made from mains water at five other areas. However at the time of writing, amoebae have not been isolated from water at Merredin. It is currently believed that there is adequate opportunity for earth-borne amoebae to gain access to town water in this system even when the mains supply is adequately chlorinated at its source.

PRIMARY AMOEBIC MENINGOENCEPHALITIS - Australian cases.
(Provided by B. Robinson and R. Walters, State Water Laboratories, Adelaide

Current figures relating to Australian cases are:

South Australia	13 cases	
Port Augusta	8	1961-1; 1963-1; 1965-2; 1966-1; 1970-1; 1972-2
Port Pyrie	3	1955-1; 1971-2 (1 is suspected not proven)
Kadina/Paskeville	2	1965-1; 1969-1
Queensland		
Mount Morgan	1	1971-1
Western Australia	2 cases	1980-2 (See above article.)

PRIMARY AMOEBIC MENINGOENCEPHALITIS - A review. (Contributed, at the request of the Editor, by Y.H. Thong, Department of Paediatrics,

Adelaide Children's Hospital and R.F. Carter, Department of Histopathology, Adelaide Children's Hospital, South Australia. It is a synopsis of an article to be published in the Medical Journal of Australia within the next three months.)

Primary amoebic meningoencephalitis is a fulminant and rapidly fatal infection, principally affecting children and young adults.[1,2] Since the first description of the disease from South Australia 15 years ago, more than 100 cases have been reported worldwide.[3] The causative organism, Naegleria fowleri, is a free-living amoeba found in most soil and fresh-water habitats,[4-7] such as ponds, lakes, rivers, sewers, swimming pools, domestic water supplies and natural and industrial thermal waters. The distinct preference of this amoeba for high temperatures explains in part the higher incidence of the disease in summer. Summer is also the time when greater exposure to contaminated water occurs during play, swimming and other activities. On reaching the nasal cavity, the amoeba then makes its way into the nasopharynx and penetrates the cribriform plate to reach the brain.[8,9]

Pathological changes

At autopsy, extensive destruction of tissue is seen along the path of amoebic invasion. The nasopharyngeal mucosa is ulcerated. The olfactory nerves are inflamed and necrotic. The brain is oedematous. Haemorrhagic necrosis is usually confined to the olfactory bulbs and base of the brain. The exudate sanguinopurulent in character, contains numerous mononuclear cells and amoebae. A more widespread amoebic involvement is evident in brain sections, where the organism can be found in the meninges, perivascular spaces and lytic areas within the brain substance. In the perivascular spaces and brain substance, inflammation is minimal. These changes are pathognomonic of primary amoebic meningoencephalitis.

A subacute granulomatous form of primary amoebic meningoencephalitis is also recognised.[1,2] This form is usually seen in older patients who have an underlying debilitating illness. The causative organism is thought to be Hartmanella (=Acanthamoeba) amoebae, belonging to a separate genus of free-living amoeba. This species appears to be susceptible to sulphonamides, while N. fowleri is not.

Clinical features

The incubation period can be as short as 1 day or as long as 2 weeks.[1,8] Early symptoms include headache, mild fever, rhinitis and pharyngitis. Severe headache, high fever, nausea, vomiting and neck rigidity follow within a day or two. By the third or fourth day, most patients are comatose and convulsing. Untreated, death usually occurs by the fifth or sixth day. Such a rapidly fatal course makes it imperative that the disease be diagnosed early for chemotherapy to be effective. As it cannot be distinguished clinically from other causes of meningoencephalitis, a high index of suspicion must be maintained by physicians and diagnostic laboratories. Diagnosis can be made by observing for amoeboid movement, a specimen of cerebrospinal fluid (CSF) which has not been subjected to

centrifugation or refrigeration. Alternatively, the CSF can be stained with Wright's or Gram's stain. It can be isolated by culture on salt-free agar plated with Escherichia coli on which it feeds.[1,10]

Treatment

Chemotherapy is at present unsatisfactory. Only 2 well-documented case have survived to date. In both cases, diagnosis was made early. The first patient, an adult, received amphotericin B alone, administered intravenously and intraventricularly via an Ommaya reservoir.[1,11] The second patient, a 7 year old girl, received a combination of amphotericin B, miconazole and rifampicin intravenously and intrathecally. Amphotericin B, clotrimazole, miconazole, rifampicin and tetracycline have been shown to have activity against N. fowleri either in-vitro, in-vivo or both.[13-18] The most promising combination appears to be amphotericin B and tetracycline.[16,17]

Prevention

Swimming or other recreational activities which subject the nasal cavities to contact with water is generally inadvisable in regions where contamination with N. fowleri is likely to occur. Strictly controlled treatment of swimming pools and domestic water supplies with customary concentrations of chlorine can further reduce the risk.[1] It has been shown[19] that a residual free chlorine level of 0.5 mg/L is sufficient to kill both trophozoites and cysts in 30 min at pH 7.0.

Conclusions

Naegleria fowleri is one of the most lethal organisms known to medical science. Much more needs to be learned about this organism, so that effective measures can be devised for prevention and treatment of this fearsome disease. This organism also holds a fascination for biological scientists because of its unique position at the interface between the free-living and parasitic modes of existence, once thought to be quite distinct. As such, these amoebae can offer valuable insights into biological adaptations to parasitism.

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FOLLOW-UP ON ROSS RIVER VIRUS EPIDEMIC IN FIJI

(Contributed by J. Aaskov. Queensland Institute of Medical Research (QIMR) Brisbane.)

An epidemic of Ross River virus infections in Fiji in 1979 was mentioned in CDI bulletin Nos. 79/12, 79/15 and 79/18.

Serological studies have shown that 13% of a sample of several hundred sera collected prior to the epidemic contained HI antibody to Ross River virus at a titre of $\geq 1/20$. However, 92% of sera collected post-epidemic from an entire village contained anti-Ross River HI antibody at a titre of $\geq 1/20$. Neutralization tests on post-epidemic sera showed antibody activity against Ross River virus but little or none against other group A arboviruses. The age and sex distribution of clinical symptoms in the above village was similar to that seen in Australia i.e. 23% of the village had clinical symptoms and of this group 2% were children (<20 years of age) while 17% were adult females and 4% adult males.

Three virus isolates were made from polyarthritis patients in Fiji by Dr Jonah Mataika. One of these was sent to Q.I.M.R. by Dr Frank Austin (University of Otago, N.Z.). Neutralization and kinetic HI tests have been unable to distinguish it from the prototype strain of Ross River (T48).

Rheumatogical and immunological studies on several Australian tourists returning to Australia have shown a pattern of reactivity identical to that seen in Australian epidemic polyarthritis patients.

In addition Dr Nair (Lautoka Hospital) has collected maternal and cord blood from 368 pregnancies. These were assayed at the QIMR for antibody to Ross River virus. Fifty-six percent of mothers had HI antibody to Ross River virus at a titre of $>1/20$. Three percent of the cord bloods (11/368) contained IgM antibody to Ross River virus suggestive of *in utero* infection with this virus. These IgM + children were of average weight at birth and showed no apparent abnormalities.

β-LACTAMASE PRODUCING N. GONORRHOEAE

Two reports of isolations of this organism during the month of December 1979 have been received from the State Health Laboratory, Brisbane; a 28 year old male who contracted his infection in Singapore, and a 27 year old male who had recently returned from El Salvador in South America.

The first report of an isolation in 1980 has been received from the Commonwealth Pathology Laboratory in Alice Springs - it was from a 31 year old male who was thought to have contracted his infection in Thailand.

CONGENITAL MALARIA (Based on MMWR 29(1):3)

A 28 day old Kampuchean boy was admitted to hospital with a 2-day history of fever and vomiting. On admission he was noted to have hepatosplenomegaly, thrombocytopaenia and monocytosis; blood smears indicated Plasmodium vivax infection. Both parents, Kampuchean refugees, had been screened for malaria on arrival in the United States 2 months previously; results were negative. The mother had no history of malaria except for unexplained chills before delivery. The infant was well at birth but had prolonged jaundice.

The child was successfully treated with chloroquine phosphate; primaquine is unnecessary in congenital malaria infection because of absence of an exo-erythrocytic stage.

Cholera in Queensland (cont'd from page 1)

classical theory assumes that the continued presence of V. cholerae in water is dependent on re-contamination by clinical cases or carriers.

In the case of the Queensland rivers, there is no evidence of continued re-contamination despite extensive efforts by Queensland health authorities to locate human sources. In addition, the extent of the contamination in the river, which flows through a relatively uninhabited area, is not consistent with a human source of contamination.

This poses the question that V. cholerae may adapt to the river environment and may maintain itself at a low level over long periods of time. During favourable conditions (at this stage, we do not know exactly what these are) the organism may multiply and may give rise to infection.

Such a theory should not be too difficult to accept. It is well-known that V. parahaemolyticus behaves in this way in a marine environment. During winter, when the water temperature goes below 13-14°C, the organism is found in only very small numbers, but as the water temperature (and the amount of plankton) increases, the number of V. parahaemolyticus found in the water also increases. It is also known that other vibrios can be found routinely in fresh water.

It may be significant that both the Queensland cases occurred in February - one in 1977 and the other in 1980, and that although the organism has been found sporadically in the river system during this period, the numbers present in summer are greater than those found during winter. In Brisbane, the summers of 1976/1977 and 1979/1980 were climatically similar.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24 - 1 - 80 - 6 - 2 - 80 BULLETIN NUMBER 80. 3

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

1

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			6	1			8		15
0101 ADENOVIRUS TYPE 1					2	1		1	4
0102 ADENOVIRUS TYPE 2		1	2	1	3	4		1	12
0103 ADENOVIRUS TYPE 3	1					1		2	4
0104 ADENOVIRUS TYPE 4			1			2			3
0105 ADENOVIRUS TYPE 5					1	1		1	3
0107 ADENOVIRUS TYPE 7	4			2	1			1	8
0108 ADENOVIRUS TYPE 8						3			3
0119 ADENOVIRUS TYPE 19					1				1
0199 ADENOVIRUS TYPING PENDING			1		8	6			15
0201 INFLUENZA A VIRUS	1		2						3
0202 INFLUENZA A VIRUS SUBTYPE H3N2						1			1
0203 INFLUENZA B VIRUS							1		1
0301 PARAINFLUENZA VIRUS TYPE 1						1			1
0302 PARAINFLUENZA VIRUS TYPE 2					9				9
0303 PARAINFLUENZA VIRUS TYPE 3		1		1	6		1	4	13
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)						2	1		3
0500 RHINOVIRUS (ALL TYPES)				2	5				7
0600 MYCOPLASMA PNEUMONIAE	8		1	1	1	2	4		17
0700 ORNITHOSIS-PSITTACOSIS						1			1
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED								1	1
0809 COXSACKIEVIRUS A9				1	1		2		4
0821 COXSACKIEVIRUS A21						1			1
0902 COXSACKIEVIRUS B2	1			1			2		4
0903 COXSACKIEVIRUS B3	1			1	1				3
0904 COXSACKIEVIRUS B4	1			1		1	4		7
0906 COXSACKIEVIRUS B6				1					1
1009 ECHOVIRUS TYPE 9						1			1
1011 ECHOVIRUS TYPE 11	23	4	3	3	5	3	10		51
1032 ECHOVIRUS TYPE 32							1		1
1033 ECHOVIRUS TYPE 33	1								1

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24-1-80 . 6-2-80 BULLETIN NUMBER 80. 3
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

2

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	MCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
1100 POLIOVIRUS NOT TYPED					1				1
1102 POLIOVIRUS TYPE 2						1			1
1200 MUMPS VIRUS	7	3	5	2		1	5	3	26
1300 HERPES VIRUS GROUP-NOT TYPED	2			1					3
1301 HERPES SIMPLEX VIRUS-NOT TYPED	10	1	1	1	1	1	29	32	76
1302 EPSTEIN-BARR VIRUS (EB VIRUS)						3			3
1303 VARICELLA-ZOSTER VIRUS			1			1			2
1306 HERPES SIMPLEX TYPE 1	4		1	19		9			33
1307 HERPES SIMPLEX TYPE 2	33			23		6			62
1399 HERPES VIRUS TYPING PENDING			3	1		1			5
1401 COXIELLA BURNETI	15					5	14		34
1521 MEASLES VIRUS			2	1		2	3		8
1522 RUBELLA VIRUS	2			2		5	7	5	21
1530 HEPATITIS A VIRUS								5	5
1532 HEPATITIS B ANTIGEN	1		7	27		5	3	4	47
1535 HEPATITIS A ANTIBODY						4			4
1541 CHLAMYDIA A - TRAC TYPE	19		2					38	59
1556 CMV - CYTOMEGALOVIRUS	2	1	4	12	2	3	3	9	36
1564 ROTAVIRUS			3	11		2		1	17
1599 ENTEROVIRUS TYPING PENDING		3	9		5	4			21
ROSS RIVER VIRUS				1		2	2	1	6
ASTROVIRUS	1								1
DENGUE							2		2
Total	137	14	54	117	53	86	102	109	672

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24/1/80 to 6/2/80 .... (80/3)
Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; 83 -Encephalitis; 83 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

3

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	5		2			2				1
0101 ADENOVIRUS TYPE 1.....		2					2				
0102 ADENOVIRUS TYPE 2.....	1	3		1			5				1
0103 ADENOVIRUS TYPE 3.....		2									
0104 ADENOVIRUS TYPE 4.....							1				
0105 ADENOVIRUS TYPE 5.....		2	1								
0107 ADENOVIRUS TYPE 7.....		3									
0119 ADENOVIRUS TYPE 19.....	1										
0201 INFLUENZA A VIRUS.....		2							1		
0202 INFLUENZA A VIRUS SUBTYPE H3N2		1									
0203 INFLUENZA B VIRUS.....		1									1
0301 PARAINFLUENZA VIRUS TYPE 1....		1									
0302 PARAINFLUENZA VIRUS TYPE 2....		9									
0303 PARAINFLUENZA VIRUS TYPE 3....		9	1			1	1			1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		3									
0500 RHINOVIRUS (ALL TYPES).....		3									
0600 MYCOPLASMA PNEUMONIAE.....	3	9							1		
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....											1
0809 COXSACKIEVIRUS A9.....	1			2							
0821 COXSACKIEVIRUS A21.....		1									
0902 COXSACKIEVIRUS B2.....	2			1							
0903 COXSACKIEVIRUS B3.....	1			1			1				
0904 COXSACKIEVIRUS B4.....				2			1				
0906 COXSACKIEVIRUS B6.....				1							
1009 ECHOVIRUS TYPE 9.....											1
1011 ECHOVIRUS TYPE 11.....	12	3	4	16			5				

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24/1/80 to 6/2/80 ...(80/3)
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 ...

4

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0100 ADENOVIRUS NOT TYPED	2			1					1	
0101 ADENOVIRUS TYPE 1								1		
0102 ADENOVIRUS TYPE 2	1									
0103 ADENOVIRUS TYPE 3	2							1		
0104 ADENOVIRUS TYPE 4	1							1		
0107 ADENOVIRUS TYPE 7	2							3		
0108 ADENOVIRUS TYPE 8	3									
0302 PARAINFLUENZA VIRUS TYPE 2								1		
0303 PARAINFLUENZA VIRUS TYPE 3								4		
0600 MYCOPLASMA PNEUMONIAE			1					6		
0700 ORNITHOSIS-PSITTACOSIS								1		
0809 COXSACKIEVIRUS A9							1			
0902 COXSACKIEVIRUS B2								1		
0904 COXSACKIEVIRUS B4							2	3		
1011 ECHOVIRUS TYPE 11	1			1			5	6	1	
1200 MUMPS VIRUS			6	2				4		
1301 HERPES SIMPLEX VIRUS-NOT TYPED		21						3		
1302 EPSTEIN-BARR VIRUS (EB VIRUS)							1	1		
1306 HERPES SIMPLEX TYPE 1	2	4						3		
1307 HERPES SIMPLEX TYPE 2		60								
1401 COXIELLA BURNETI			2		1		13	13		
1521 MEASLES VIRUS								2	1	
1522 RUBELLA VIRUS			4		2				1	
1541 CHLAMYDIA A - TRIC TYPE	1	20								
1556 CMV - CYTOMEGALOVIRUS		1	1			4	1	1	4	
ROSS RIVER VIRUS					4			1		
DENGUE (TYPE 3)					1			1		
Total	15	106	14	4	8	4	23	57	8	

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24/1/80 to 6/2/80 .... (80/3)
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.-CONTINUED 5

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 membr
1032 ECHOVIRUS TYPE 32.....	1										
1033 ECHOVIRUS TYPE 33.....				1							
1102 POLIOVIRUS TYPE 2.....							1				
1200 MUMPS VIRUS.....	1	2	1	9		1	1				
1300 HERPES VIRUS GROUP-NOT TYPED..											3
1301 HERPES SIMPLEX VIRUS-NOT TYPED	9	4		1	1	2			1		37
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	1										
1303 VARICELLA-ZOSTER VIRUS.....											2
1306 HERPES SIMPLEX TYPE 1.....		1	1				1				22
1307 HERPES SIMPLEX TYPE 2.....											4
1401 COXIELLA BURNETI.....	5	3									2
1521 MEASLES VIRUS.....											6
1522 RUBELLA VIRUS.....	1	3									18
1530 HEPATITIS A VIRUS.....	1							4			
1532 HEPATITIS B ANTIGEN.....	15							32			
1535 HEPATITIS A ANTIBODY.....								4			
1541 CHLAMYDIA A - TRIC TYPE.....	38										
1556 CMV - CYTOMEGALOVIRUS.....	14	2	1			1		2		4	1
1564 ROTAVIRUS.....							17				
ROSS RIVER VIRUS	1										2
ASTROVIRUS							1				
DENGUE (TYPE 3)	1										
Total.....	110	74	9	37	1	5	39	42	3	5	102

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

12th 4 Weekly Period for 1979

BULLETIN 80/3

3.11.79 - 30.11.79 inclusive

DISEASE	Total	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	CUMULATIVE TOTAL TO DATE FOR YEAR
Amoebiasis	1				1					16
Ankylostomiasis	1					1				* 147
Anthrax										1
Arbovirus infection	5		4	1						64
Brucellosis	10	2			8					56
Campylobacter infections										—
Chancroid	2			2						19
Cholera										1
Congenital rubella syndrome										—
Diphtheria										—
Donovanosis	9			9						64
Giardiasis										—
Genital herpes										—
Gonococcal ophthalmia neonatorum										—
Gonorrhoea	832	228	180	158	67	142	8	24	25	10,828
Hepatitis A (infectious)	199	114	32	11	18	6	3	8	7	* 1,800
Hepatitis B (serum)	58	27	9	12	7			2	1	727
Hepatitis - unspecified	9					8			1	* 77
Hydatid disease	1	1								24
Lassa Fever										—
Legionnaires disease										—
Leprosy	3		2			1				46
Leptospirosis	11		9	1	1					58
Lymphogranuloma venereum										1
Malaria	28	4	3	15		4			2	332
Marburg Disease										—
Meningococcal infections	4		1	3						74
Non-specific urethritis										—
Ornithosis	1				1					11
Pertussis (whooping cough)	9		9							172
Plague										—
Poliomyelitis										—
Q. fever	51	9	1	37	4					548
Rabies										—

DISEASE	Total	N.S.W.	Vic	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	CUMULATIVE TOTAL TO DATE FOR YEAR
Salmonella infections	116	12	15	25	17	17		28	2	1672
Shigella infections	81			8	8	25		40		608
Smallpox										—
Syphilis	204	53	12	53	23	19		44		2914
Tetanus	3	1	1	1						14
Trachoma										1
Tuberculosis (all forms)	145	50	27	28	14	24		1	1	* 1513
Typhoid fever	1	1								22
Typhus (all forms)										2
Vibrio parahaemolyticus infections										—
Yellow Fever										—
Yersinia enterocolitica infections										—

Data collected under the Notificable 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.

* Corrections made to the cumulative total since last report.

Ankylostomiasis + 10 cases for N.T.

Hepatitis A + 2 cases for N.T.

Hepatitis Unspecified - 2 cases for N.T.

Tuberculosis + 1 case for N.T.
- 6 cases for Vic.