AUSTRALIA

Communicable Diseases Intelligence

Bulletin number 81/24

Issue date: 4 December 1981

Contents:

VIRUS REPORTING SCHEME - A total of 818 reports were received this reporting period. Patterns suggested by the reports included a continuation of the increase in measles (29 reports received compared with 31, 13 and 25 for the previous three periods) and rubella (45 compared with 42, 36 and 23) infections. Among the recent measles reports from Fairfield Hospital, Melbourne, a six year old boy developed otitis media and acute mastoiditis requiring immediate surgery, two children developed spontaneous mediastinal emphysemia which resolved spontaneously, and one child required tracheotomy for croup. The rubella reports from the same hospital included a neonate with pneumonia and microcephaly. The mother had rubella at 26 weeks gestation.

. Dengue fever was confirmed by the State Health Laboratory, Brisbane, in three patients from Cairns; a 22 year old male, a 29 year old female and a 38 year old female.

. Australian encephalitis was diagnosed by the State Health Laboratory Services, Perth, in a 36 year old man and his 25 year old wife from Newman. Both patients had mild fever, a generalised faint maculopapular rash, mild photophobia and myalgia. Although specific IgM against MVE virus was detected, the sera are being further tested against other arbovirus group B antigens since the couple had visited Southern India one month prior to onset. The husband is the "chief mosquito catcher" in Newman.

. The State Health Laboratory, Brisbane, reported vaccinia lesions over the upper lip and nose in a one year old boy. The child had not been vaccinated, but had visited a medical practitioner's surgery where vaccinia was regularly used for the treatment of warts. Smallpox vaccination has not been shown to have any therapeutic effect on herpes simplex, herpes zoster or warts.

. A generalised neonatal HSV-2 infection in a 1 month old boy was reported by the Royal Alexandra Hospital for Children, Sydney. Onset was four days after birth, and presented as a respiratory infection progressing to marked hepatomegaly, splenomegaly and coagulopathy. There was no CNS involvement. Acyclovir was administered without success. The mother was negative for herpes, but the father had a history of genital herpes during his wife's pregnancy. Of the 35 HSV-2 reports from Fairfield Hospital this period, four were in women that were 36-40 weeks pregnant.

The Bulletin is compiled and distributed by the Environmental Health Branch, Department of Health, P.O. Box 100, Woden, A.C.T. 2606, Australia, and is available on request.

HERPES SIMPLEX VIRUS INFECTIONS

Herpes virus untyped and herpes simplex virus (HSV) infections comprised 26.4% and 28% of the total reports received by CDI in 1980 and the first nine months of 1981 (January-September) (see Table 1). Tables 2, 3 and 4 are analyses of these reports with respect to age, sex and clinical symptoms. The following article is a review of the current knowledge of HSV infections, giving some perspective to the CDI data.

HSV infections are among the commonest diseases affecting man. Infections are usually troublesome, often establishing persistent conditions, and occasionally becoming life-threatening. The two strains of virus, herpes simplex virus type 1 (HSV-1) and herpes simplex virus type 2 (HSV-2), were formerly categorized as attacking above and below the waist. There is no difference in the severity of infection caused by each type. Primary infections usually present as acute gingivostomatitis, pharyngitis, conjunctivitis, keratitis, herpes genitalis and herpetic whitlow. Infections may progress to an acute generalised form or an acute necrotising encephalitis. Severe generalised infections in neonates have a very high mortality.

Genital herpes

Genital herpes is now one of the major sexually transmitted diseases. In the USA, it has been reported to be diagnosed more frequently than gonorrhoea in women attending private gynaecology clinics.[1] In addition to the morbidity associated with recurring episodes, females may transmit the infection to their newborn, and may incur an increased risk of cervical cancer.[2]

The increasing incidence of genital herpes has indicated a change in the traditional HSV-1/HSV-2 pattern. Recent surveys have reported incidences of HSV-1 genital herpes as high as 32%[3] and 37%[4] in females. From the CDI data (Table 3), HSV-1 accounted for 11.8% and 15.2% of genital herpes in females in 1980 and January-September 1981 respectively, with corresponding percentages of 4.2% and 4.9% for males. Although the incidence of genital herpes has increased in the world, the proportion of the population which has been infected with HSV-2 remains low, since transmission is principally by sexual contact, and antibody is seldom acquired before 15 years of age. By contrast, non-genital HSV-1 infections seem to have declined, especially in the upper socioeconomic groups.[5,6]

Recurrent infection occurs with both HSV-1 and HSV-2. The virus establishes a latent state in the neural cells of the sensory and autonomic ganglions of nerves innervating the site of recurrent mucocutaneous lesions. The molecular status of the HSV genome is unknown, but only a minor component of the Epstein-Barr virus genome is integrated into host cell DNA, with the remaining sequences existing as a closed circular molecule.[7] It is not known what precipitates recurrences of genital herpes. Emotional stress was the triggering factor most cited in one study, although a relationship between the onset of a recurrence and the onset of menses 5-12 days later was also tentatively proposed.[8] In this study, from 138 pelvic examinations performed, of which 74 presented with external lesions, no cervical or vaginal lesions were found.

TABLE 1 Notifications of herpes infection to CDI reporting scheme

Virus	1980	1981 (Jan-Sept)
Herpes and HSV not typed	1939	1083
HSV-1	884	858
HSV-2	2182	2019
Total CDI reports	18908	14166

TABLE 2 Age distribution of HSV infections

Age	1980		1981 (Jan-Sept)	
	HSV-1	HSV-2	HSV-1	HSV-2
0-5 months	7	3	12	4
6-11 months	13	2	21	-
1-4 years	95	1	102	2
5-14 "	97	3	106	7
15-24 "	230	620	202	565
25-59 "	274	880	266	904
60 + "	50	12	53	18
unknown	118	661	96	520

TABLE 3 Sex distribution of HSV infections

Sex	1980		1981 (Jan-Sept)	
	HSV-1	HSV-2	HSV-1	HSV-2
Males	371	1113	355	1017
Males with genital disease	48	1028	48	937
Females	482	945	489	954
Females with genital disease	116	868	153	851

TABLE 4 Clinical symptoms reported for HSV infections.

Symptoms	1980		1981 (Jan-Sept)	
	HSV-1	HSV-2	HSV-1	HSV-2
Genital	176	2072	210	1842
Skin/mucous membrane	481	102	460	166
Respiratory	67	3	50	2
Eye	59	-	76	1
Fever/malaise	58	2	31	1
Urinary	18	1	6	1
Encephalitis	17	1	22	1
Other CNS	3	1	3	-
Gastro-intestinal	6	-	1	1
Hepatic	3	-	2	-
Cardiovascular	4	-	-	-
Endocrine/salivary	4	-	1	-
Reticuloendothelial	1	-	3	-
Congenital	-	-	-	2
Other	8	3	5	2
No information	13	3	15	13

However, cervical shedding of HSV was observed in 33% of patients during recurrence and in 4% of patients between recurrences. This rarity of internal lesions, and the low titres of virus observed in the absence of lesions, suggests that the risk of HSV transmission through sexual intercourse during asymptomatic periods is small.

Another study has shown that recurrences of genital herpes are more frequent in men than women, and genital HSV-1 infection was less likely to recur than genital HSV-2 infection.(9) This trait was independent of gender and titre of HSV neutralizing antibody in acute or convalescent serum. Among patients with primary genital HSV-2 infection, those with seroconversion and high titres of neutralizing antibody to HSV-2 were more likely to experience recurrences than those with no HSV-2 neutralizing antibody. This contrasts with HSV-1 infections in which prior HSV-1 exposure may afford protection against the acquisition of symptomatic genital HSV-1 disease.(10,11,12) Such findings suggest that HSV-1 is less likely to produce a latent infection of the sacral dorsal root ganglion after genital infection than HSV-2, or that the two virus types behave differently once latency has been established. The presence of neutralising antibody to HSV-2 after primary genital infection may represent a serological marker for the existence of latent ganglionic infection with a subsequent increased likelihood of recurrent infection.

Although yet to be established, HSV-2 infection has been proposed as a carcinogenic stimulus for squamous-cell carcinoma in situ (CIS) of the vulva, which has been associated with cervical CIS and invasive cancer.(13) In a recent study, HSV-2 non-structural protein antigens were found in CIS of the vulva lesions in nine of ten patients.(14) The presence of non-structural antigens implied that the patients did not have an acute HSV-2 infection, but the presence of serum IgG antibodies indicated a recent exposure.

Nosocomial and neonatal infections

Patients at risk of severe illness from HSV infection include the immunocompromised, particularly renal transplant recipients (15), patients with eczema or extensive burns and newborn babies. Neonatally acquired infection has a mortality of about 50%, and many of the survivors have permanent sequelae.(16) Babies most at risk are those whose mothers have primary genital or facial infections, particularly if the membranes rupture prematurely and delivery is delayed.(17,18) Infection may also occur after delivery from hospital or from household contacts with active lesions, (19,20,21) or transmitted indirectly from infant to infant.(22) Eight neonatal deaths and three cases of neonatal morbidity were recorded by the Australian Bureau of Statistics and 73 major obstetric units in 2,469,889 live births between 1967-77.(23) In the UK, 66 cases were reported between 1973-80.(24) However these figures may be underestimates, since the diagnosis may be missed in mild infections or in severe ones where mucocutaneous lesions are absent. In a one year study at the Mercy Maternity Hospital, Melbourne, (25) 119 of 347 staff members in direct contact with patients reported 165 non-genital HSV infections involving an aggregate loss of 778 working days. HSV was also isolated from the saliva of 9.6% of asymptomatic staff members. However, the study also showed that 80% of mothers possessed HSV antibodies,

thus affording some degree of passive protection. In 1978, the National Health and Medical Research Council recommended that "sufferers from active herpes simplex lesions, irrespective of type, be excluded from duties involving the handling of, or close contact with, infants, maternity patients or patients with eczematous conditions".

The risk of HSV infection is however a two way transmission. Herpetic whitlow can result from contact with HSV containing secretions removed by catheter suction, particularly if gloves are not worn.[26] In 1976 a nosocomial herpetic outbreak occurred in the intensive care unit of a large US university-affiliated children's hospital.[27] Three nurses suffered primary infections of herpetic whitlow (one with encephalitic symptoms), the husband of one nurse had acute gingivostomatitis, and a fourth nurse had acute pharyngitis. Consequently, the identification of active HSV infection in patients and staff members, together with the wearing of gloves on both hands by personnel when applying suction to oral and respiratory secretions, or giving mouth care to any patient, are the cornerstones of transmission prevention.

Antiviral drugs

Effective antiviral drugs are only just being developed, but similar to other pathogens, viruses have diverse strategies for drug resistance.[28] The two 5- substituted 2'-deoxyuridines (idoxuridine and trifluorothymidine) have been shown to be useful in the treatment of ocular herpes, but their toxicity and poor selectivity against healthy cells are too high for systemic use.[28] A third compound, adenine arabinoside (vidarabine), is suitable for systemic use, but it is rapidly inactivated by a cellular enzyme. The most promising antiviral agents so far tested are acyclovir (acycloguanosine)[29] and bromovinyldeoxyuridine.[30,31] These are highly selective nucleoside analogues which after conversion to their respective nucleotides are selectively phosphorylated by a virus specific thymidine kinase[32]. These monophosphates are further converted to the triphosphate, and these molecules then interfer with the action of the virus induced DNA polymerase, thus inhibiting virus replication.[33] Both drugs have proved effective against acute HSV infections in animals, and trials in man are underway.[34] In the USA, the National Institutes of Health are to fund trials comparing intravenous acyclovir with vidarabine for neonatal infection and herpes encephalitis. In one published trial involving bone-marrow transplant recipients, no lesions appeared in the ten patients who received acyclovir compared with seven of ten patients who received placebo.[35] None of the patients had evidence of drug toxicity. However, when treatment was terminated, five of the acyclovir patients had mild culture positive HSV infection, and two additional patients shed virus without having lesions. Although a potent inhibitor of HSV replication providing effective prophylaxis against reactivated infection, acyclovir does not appear to eradicate latent virus. Similar conclusions have been inferred from experimental infections in mice.[36,37] Since the drug interacts with the viral proteins synthesised late in the productive replication cycle, it suggests that most of the HSV genome is not expressed in latency.

Adverse side effects and drug resistance are always problems associated with chemotherapy, particularly long term treatment. Exhaustive studies are necessary to evaluate the

true therapeutic/toxic ratios. To date, delerium has been recorded in two patients treated with acyclovir, [35] and in a recent study of the combination of vidarabine and interferon against chronic hepatitis B infection, increased side effects as well as increased efficacy were associated with treatment.[38] Resistant strains to idoxuridine [39,40] and acyclovir (in vitro) [41] have also been reported.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

1

REPORTING PERIOD - 12/11/81 - 25/11/81 BULLETIN NUMBER - 81/24

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	6					6	16		28
0101 ADENOVIRUS TYPE 1	1			1		2		1	5
0102 ADENOVIRUS TYPE 2			1	2		3			6
0105 ADENOVIRUS TYPE 5	1			1		4			6
0115 ADENOVIRUS TYPE 15	1								1
0199 ADENOVIRUS TYPING PENDING			7	1	3	5			16
0201 INFLUENZA A VIRUS	1						2	4	7
0203 INFLUENZA B VIRUS	3								3
0301 PARAINFLUENZA VIRUS TYPE 1				1	2				3
0302 PARAINFLUENZA VIRUS TYPE 2						1			1
0303 PARAINFLUENZA VIRUS TYPE 3	2	1		6	3	7	7	4	30
0399 PARAINFLUENZA VIRUS TYPING PENDING						2			2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1	5	2					2	10
0500 RHINOVIRUS (ALL TYPES)	1		1	5	8	1	8		24
0600 MYCOPLASMA PNEUMONIAE	6			2		1	4	4	17
0700 ORNITHOSIS-PSITTACOSIS	1			1					2
0809 COXSACKIEVIRUS A9							1		1
0904 COXSACKIEVIRUS B4	1	1	1	2			3	1	9
1006 ECHOVIRUS TYPE 6				2					2
1017 ECHOVIRUS TYPE 17							2		2
1022 ECHOVIRUS TYPE 22			3				1		4
1030 ECHOVIRUS TYPE 30						1			1
1101 POLIOVIRUS TYPE 1						1			1
1102 POLIOVIRUS TYPE 2								2	2
1103 POLIOVIRUS TYPE 3							1		1
1104 POLIOVIRUS-VACCINAL STRAIN	1		1						2
1200 MUMPS VIRUS	3			4			1		8
1300 HERPES VIRUS GROUP-NOT TYPED	21			2		7		2	32
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2		3				39	44
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	12					1			13
1303 VARICELLA-ZOSTER VIRUS	4			2		3	1		10
1306 HERPES SIMPLEX TYPE 1	5			12		15	13		45

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 2

REPORTING PERIOD - 12/11/81 - 25/11/81 BULLETIN NUMBER - 81/24

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
1307 HERPES SIMPLEX TYPE 2	54			33		16	26		129
1399 HERPES VIRUS TYPING PENDING			10		7	7			24
1401 COXIELLA BURNETI	15			1		1	8		25
1502 PICORNA VIRUS-NOT TYPED								4	4
1512 VACCINIA VIRUS							1		1
1514 MOLLUSCUM CONTAGIOSUM						1		2	3
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)								1	1
1521 MEASLES VIRUS	16	2		3	7	1			29
1522 RUBELLA VIRUS				16	2	2	24	1	45
1532 HEPATITIS B ANTIGEN	9		5	20		9	6	3	52
1535 HEPATITIS A ANTIBODY	4		2			2	6	4	18
1541 CHLAMYDIA A - C TRACHOMATIS	32					2		35	69
1556 CMV - CYTOMEGALOVIRUS	5		2	18	2	1	5	9	42
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS	1	5	2	1	6	1	1	1	18
1599 ENTEROVIRUS TYPING PENDING			3		4	1			8
AUSTRALIAN ENCEPHALITIS								2	2
ROSS RIVER VIRUS							1	2	3
SMALL VIRUS (LIKE) PARTICLE	2					1			3
DENGUE							3		3
Total	209	16	40	140	44	105	141	123	818

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

3

PERIOD : 12/11/81 to 25/11/81 .... 81/24

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir atory	Encepha litis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....		1					4				
0102 ADENOVIRUS TYPE 2.....		3					3				
0105 ADENOVIRUS TYPE 5.....		4					1				1
0201 INFLUENZA A VIRUS.....	1	4	1	1							1
0203 INFLUENZA B VIRUS.....		3									
0301 PARAINFLUENZA VIRUS TYPE 1....		3									
0302 PARAINFLUENZA VIRUS TYPE 2....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....	2	27				2					
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		10									
0500 RHINOVIRUS (ALL TYPES).....		23									2
0600 MYCOPLASMA PNEUMONIAE.....	3	13	1								1
0700 ORNITHOSIS-PSITTACOSIS.....	1	1									
0809 COXSACKIEVIRUS A9.....	1										
0904 COXSACKIEVIRUS B4.....	1	2		3		1	1				
1006 ECHOVIRUS TYPE 6.....				1							
1022 ECHOVIRUS TYPE 22.....		1					3				
1030 ECHOVIRUS TYPE 30.....				1							
1101 POLIOVIRUS TYPE 1.....							1				
1102 POLIOVIRUS TYPE 2.....		1									
1103 POLIOVIRUS TYPE 3.....							1				
1104 POLIOVIRUS-VACCINAL STRAIN....	1						1				
1200 MUMPS VIRUS.....	1	1									1
1300 HERPES VIRUS GROUP-NOT TYPED..											1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1										28
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		1						1			2
1303 VARICELLA-ZOSTER VIRUS.....	2										7
1306 HERPES SIMPLEX TYPE 1.....	2	4	2								17

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 4

PERIOD : 12/11/81 to 25/11/81 .... 81/24

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

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
1307 HERPES SIMPLEX TYPE 2.....											8
1401 COXIELLA BURNETI.....	10								1		
1514 MOLLUSCUM CONTAGIOSUM.....											3
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....	5	3									21
1522 RUBELLA VIRUS.....	3										39
1532 HEPATITIS B ANTIGEN.....	22					1		30			
1535 HEPATITIS A ANTIBODY.....	1							17			
1556 CMV - CYTOMEGALOVIRUS.....	15	5		2				2		4	
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....						1	16				
AUSTRALIAN ENCEPHALITIS											1
ROSS RIVER VIRUS		1									1
SMALL VIRUS (LIKE) PARTICLE	1						2				
DENGUE											3
Total.....	73	112	4	8		5	34	50	1	4	138

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

5

PERIOD : 12 / 11 / 81 to 25 / 11 / 81 ...

81/24

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		
0105 ADENOVIRUS TYPE 5.....								1		
0115 ADENOVIRUS TYPE 15.....	1									
0201 INFLUENZA A VIRUS.....								2		
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0500 RHINOVIRUS (ALL TYPES).....								1		
0600 MYCOPLASMA PNEUMONIAE.....			1							
0904 COXSACKIEVIRUS B4.....			1					1		
1006 ECHOVIRUS TYPE 6.....			1					1		
1017 ECHOVIRUS TYPE 17.....							1	1		
1102 POLIOVIRUS TYPE 2.....										1
1200 MUMPS VIRUS.....			5		1			1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED		17							1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			5	1	1			6	2	
1303 VARICELLA-ZOSTER VIRUS.....		1	1							
1306 HERPES SIMPLEX TYPE 1.....	1	18					1	1		
1307 HERPES SIMPLEX TYPE 2.....	1	120								
1401 COXIELLA BURNETI.....							3	10	2	
1512 VACCINIA VIRUS.....	1									
1521 MEASLES VIRUS.....									2	
1522 RUBELLA VIRUS.....			2		5	1		2		
1541 CHLAMYDIA A - C TRACHOMATIS...	1	68								
1556 CMV - CYTOMEGALOVIRUS.....	1	5		2		4	2	2		1
1564 ROTAVIRUS.....										1
AUSTRALIAN ENCEPHALITIS								2		
ROSS RIVER VIRUS					3					
SMALL VIRUS (LIKE) PARTICLE								1		
DENGUE					2					
Total.....	6	229	16	3	12	5	7	34	7	3

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

.10th4 Weekly Period for...1981.

Bulletin ..81/24

(6 9 81 to 3 10 81 inclusive)

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Amoebiasis	N.N.			3					3	48
Ankylostomiasis	N.N.			N.N.					–	93
Anthrax	1								1	1
Arbovirus infection				N.N.					–	19
Brucellosis				1					1	27
Campylobacter infections	N.N.	1	N.N.	17	N.N.	N.N.	3	N.N.	21	236
Chancroid			1	N.N.		N.N.	N.N.		1	19
Cholera									–	2
Congenital rubella syndrome	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	–	–
Diphtheria									–	10+1 CARRIER
Donovanosis		N.N.	4	N.N.	1	N.N.	5		10	49
Giardiasis	N.N.	N.N.	N.N.	21	N.N.	N.N.	N.N.	N.N.	21	534
Genital herpes	N.N.	N.N.	N.N.	18	N.N.	N.N.	N.N.	N.N.	18	284
Gonococcal ophthalmia neonatorum		N.N.		N.N.	N.N.	N.N.	N.N.	N.N.	–	–
Gonorrhoea	360	180	113	37	158	8	63	7	926	*8378
Hepatitis A (infectious)	35	25	32	11	3	2	1		109	1157
Hepatitis B (serum)	15	11	6	7	3		2	1	45	380
Hepatitis - unspecified	N.N.	N.N.		N.N.	11	N.N.	2		13	82
Hydatid disease	1								1	17
Lassa Fever	N.N.		N.N.	N.N.		N.N.	N.N.	N.N.	–	–
Leptonnaires disease	N.N.		N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	–	16
Leprosy		2	1	1			1		5	35
Leptospirosis	1	3			1				5	47
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.			–	–
Malaria	6	5	10	3	1		2		27	335
Marburg Disease	N.N.		N.N.	N.N.		N.N.	N.N.	N.N.	–	–
Meningococcal infections	N.N.	1	2	1		N.N.	1		5	53
Non-specific urethritis	N.N.	N.N.	N.N.	90	N.N.	N.N.	N.N.	N.N.	90	1011
Ornithosis		1		3					4	12
Pertussis (whooping cough)	N.N.	6	N.N.	1	N.N.	N.N.	N.N.	N.N.	7	124
Plague									–	–
Poliomyelitis									–	–
Q. fever	1		16	6	N.N.		N.N.		23	331
Rabies	N.N.	N.N.	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	38	34	23	28	10	1	18		152	2006
Shigella infections	N.N.	3	10	4	6		13		36	345
Smallpox									–	—
Syphilis	88	14	36	7	40		45		230	2316
Tetanus		1							1	10
Trachoma	N.N.	N.N.		N.N.	N.N.	N.N.			—	1
Tuberculosis (all forms)	37	32	20	8	18		1		116	* 1110
Typhoid fever		1c							1c	7 + 1 CARRIER
Typhus (all forms)									–	—
Vibrio parahaemolyticus infections	N.N.	N.N.	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.	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

* Corrections made to the Cumulative Total since last Report

Gonorrhoea +14 cases for S.A.
– 2 cases for N.T.

Tuberculosis + 1 case for S.A.