ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 85/18
Issue date: 6 September 1985

Contents:

VIRUS REPORTING SCHEME - A total of 1248 reports was processed this period. It is evident from the last two periods that influenza A virus activity in the community has been declining (Table 1). One hundred infections have been reported in this generation (15 subtyped H_3N_2, one H_1N_1 and 84 untyped)

TABLE 1

	1984	1985
Influenza A reports, generation 14, 20 June-3 July	-	41
Influenza A reports, generation 15, 4 July-17 July	6	121
Influenza A reports, generation 16, 18 July-31 July	-	246
Influenza A reports, generation 17, 1 Aug-14 Aug	31	151
Influenza A reports, generation 18, 15 Aug-28 Aug	47	100

Two cases of Q fever were reported. Both patients were associated with the meat industry and neither had been involved in the Q fever vaccine field trial being conducted in South Australia.

AIDS SURVEILLANCE - AUSTRALIA

To 4 September 1985, 110 cases of AIDS fulfilling the criteria of case definition have been reported to the AIDS Task Force.

	Cases	Deaths
New South Wales	72	29
Victoria	17	8
Queensland	13	9
South Australia	-	-
Western Australia	6	3
Tasmania	1	1
Australian Capital Territory	1	-
Total	110	50

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

PENICILLIN SENSITIVE, SPECTINOMYCIN RESISTANT NEISSERIA GONORRHOEAE - WESTERN AUSTRALIA

(Contributed by Morris M. Gollow, Consultant Venereologist, M. Blums, and A. Ismail, Health Department of Western Australia)

A female patient aged 26 attended the Special Clinic in Perth, Western Australia for a routine check up, following a recent episode of sex with multiple partners.

Immediate microscopy revealed Gram-negative intracellular diplococci in the urethral and endocervical specimens, whereupon she was treated with 2g amoxycillin and 1g probenecid as a stat dose. Two days later, culture confirmed the presence of Neisseria gonorrhoeae. Minimum inhibitory concentrations (MIC) of 0.03 micrograms per millilitre for penicillin and greater than 800 micrograms per millilitre for spectinomycin were determined. The patient's proof of cure swabs were negative for N. gonorrhoeae. In view of the spate of β-lactamase producing organisms reported world-wide and the recommendations of some centres for the use of spectinomycin as the primary line of treatment, this could result in failure of cure and spread of disease in the interim period.

The implications of this finding to those centres advocating spectinomycin as drug of first choice are of importance.

Editorial Comment

The contributors of this report are unaware, to date, of any penicillin sensitive, spectinomycin resistant N. gonorrhoeae having been reported in Australia. CDI is interested in hearing of any Australian cases.

AMOEBIASIS

Amoebiasis denotes the condition of harbouring Entamoeba histolytica with or without clinical manifestations[1]. The causative parasite is a unicellular protozoon whose normal habitat is the crypts of the caecum and ascending colon where the faecal contents are liquid. The disease is known to occur in every part of the world, and it is estimated to affect 10% of the world's population, although its prevalence and severity may differ from area to area or increase in special circumstances. Seven deaths (13 cases) from amoebic dysentery were reported recently in Colorado, USA, from infections arising from colonic irrigation at a chiropractic clinic[2]. While amoebiasis is endemic in Australia, the native strains seem to be of low invasiveness, and extraintestinal lesions are rare[3]. A State breakdown for amoebiasis based on notifiable disease returns is given is Table 1.

TABLE 1 Notifiable disease statistics for amoebiasis, Australia 1979-84.

Year	State								
	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	Australia
1984	13	-	4	20	7	-	1	1	46
1983	6	-	19	26	3	-	2	1	57
1982	6	1	14	10	-	-	1	1	33
1981	NN	7	30	18	4	-	-	3	62
1980	NN	1	22	14	4	1	-	11	53
1979	NN	3	1	4	5	-	-	2	15

NN = not notifiable

The spread of amoebiasis is by faecal-oral contamination, usually of uncooked food such as salads and vegetables. It is a disease associated with poor hygiene, and as such is more common in the tropics, and areas where human faeces are used for fertilisation of crops. Infection is contracted by ingestion of cysts; trophozoites being destroyed in the stomach. Cysts are resistant to the usual chlorination of water, but are rapidly destroyed by drying or deep freezing, and are killed if heated to 50°C for five minutes.

The clinical presentation of amoebic infection of the large bowel varies with the extent of the infection. Indeed, amoebiasis is one of the great tropical mimics,[4] and must be differentiated from the bacillary dysenteries, ulcerative colitis, Crohn's disease and nonspecific and other diarrhoeas, and from carcinoma of the colon. Extraintestinal amoebiasis must be considered in the differential diagnosis of abdominal tumours, malignant, parasitic and inflammatory diseases of the liver; of empyema of the gall bladder; of appendicitis and peptic ulcer; in the chest as a cause of empyema, lung abscess; and of ulcerating granuloma around the anus.

Most patients with amoebae have no symptoms; the organism living quietly in the bowel lumen and shedding as cysts in the stool. Diagnosis depends on the demonstration of the parasite, although the available serological tests are adequate for most clinical situations. Circulating antibody levels, which are directed mainly against surface determinants of the amoeba, are of particular value in confirming the diagnosis of invasive lesions of the gut[4]. Strong seropositivity (by immunofluorescent antibody tests and amoebic gel diffusion) also appears to be an indication of potentially invasive organisms in symptom-free individuals[5]. Local antibody levels in patients with lumenal amoebiasis are not known, although a transient rise in IgA coproantibodies has been reported[6]. If the intestinal milieu is favourable, and the strain of E. histolytica is pathogenic, invasion of the mucosa may take place. This is characterised by massive tissue injury with no evidence to date that the host immune response is involved in preventing infection or modifying the course of the disease. Repeated invasion of the colon followed by secondary infection may result in the formation of a granuloma - the amoeboma[7].

Hepatic involvement is a consequence of the amoebae penetrating tributaries of the portal vein. In the liver, cellular necrosis occurs with destruction of liver tissue and subsequent abscess formation. Liver abscesses may rupture into the peritoneum, the stomach, the intestines, the pericardium, and the kidney. Approximately 15% of the world's population infected with E. histolytica will have systematic amoebiasis[6]. In Mexico, where the incidence of amoebic liver abscess is very high, the disease is found in about 2% of all adult patients and in 3-4% of autopsies performed in general hospitals[8].

From a practical standpoint, the Australian practitioner sees amoebiasis in one of three different clinical patterns:-

- Amoebiasis in middle income or affluent groups who have acquired the disease during a trip to foreign areas that have poor environmental hygiene;

- Amoebiasis in migrants and refugees who have brought their infections with them;

. Amoebiasis in homosexual males.

Proper treatment initiated early in acute, severe disease, whether colonic or hepatic, is usually followed by a gratifying response and cure when there is little risk of reinfection[9]. Although the first report published in 1968 suggesting sexual transmission of enteric protozoa among homosexual males was largely ignored[10], more recent surveys have shown that the prevalence of amoebiasis has substantially increased in this group because of their sexual practices which enhance faecal-oral contamination[11,12]. Studies have shown prevalence rates of 36% among homosexual males in San Franscisco[13], 20%[14] and 31%[15] in New York, 20%[16] and 27%[17] in Toronto, 12%[18] in London, and 21%[19] in Göteburg, Sweden. Giardia lamblia cysts were frequently found in conjunction with such E. histolytica infections. Despite the high prevalence of pathogenic and non-pathogenic intestinal protozoa in homosexual men, there is usually a lack of correlation between symptoms and the presence of cysts. It has been suggested that only certain groups of E. histolytica are associated with clinical amoebiasis[20], and homosexuals appear to carry the non-pathogenic varieties[21]. However, symptomatic amoebiasis is more likely to occur in immunodeficient people, and certain groups of homosexual males have been shown to have an appreciable suppression of their immune function[22]. E. histolytica has also been found to have an immunosuppressive activity[23]. Consequently, amoebiasis has been proposed as having a role in the acquired immune deficiency syndrome (AIDS)[24].

The disparity between the number of individuals passing cysts and those showing overt clinical symptoms has led to the concept of virulent and avirulent strains[20]. The isolation of E. histolytica from a host is accomplished by inoculating faeces or aspirated liver pus into culture[25], followed by subculture into similar culture medium. Conventional techniques of cultivating E. histolytica involve growing the amoebae in association with one or more species of microorganism. Whatever medium is selected, it must have the ability to support the growth of suitable bacteria for several days, and contain rice starch as an easily assimilative carbohydrate source. Axenic cultures provide a source of amoebae free of the influences of the microbial associates, whereas monoxenic cultures are amoeba-trypanosomatid flagellate cultures. Axenically cultivated E. histolytica is non-infective to guinea-pigs, but the infectivity and degree of virulence are restored when amoebae are reassociated with mixed bacterial flora[26]. It has been suggested that the restoration is due to the stimulation of the amoeba's electron transport system in the microaerobic conditions; the bacteria acting as broad-range scavengers for oxidised molecules and metabolites through their contribution of enzymatic systems, components or products[27]. Whether these observed differences of pathogenicity are phenotypic or genotypic is not known.

Markers for the pathogenicity of a population of amoebae have also been demonstrated using the mobilities of four enzymes (glucose phosphate isomerase; L-malate: $NADP^+$ oxidoreductase; phosphoglucomutase; hexokinase) on thin-layer starch gel electrophoresis[20]. Some 20 distinct zymodemes with distinct geographical distributions[28] are now recognised, of which seven have been associated with clinical evidence of tissue invasiveness[29]. The markers for pathogenicity are the absence of the band together with the presence of the β

band in phosphoglucomutase. Advanced bands in the hexokinase normally confirm the phosphoglucomutase result. Mixtures of non-pathogenic zymodemes of E. histolytica have been found in clinical specimens, but never a mixture of pathogenic zymodemes. Prior to the isolation of zymodeme XX from the pus in a confirmed case of amoebic liver abscess[30], the strain was "made" in the laboratory in June 1983 following the cocultivation of zymodemes II and XIV[28]. This result has raised the question of possible sexual genetic exchange, an hypothesis that has remained dormant for over a decade[31]. Sexuality has also been proposed in Trypanosoma cruzi[32].

The reasons why the non-pathogenic zymodemes are non-invasive remain unanswered, although there is some evidence that they are susceptible to complement-mediated lysis whereas pathogenic strains are resistant[33]. Although E. histolytica has been found to be parasitised by viruses[34], their presence has not been related to pathogenicity[35,36].

The main damage caused by the amoeba resides in the destruction of host tissues. The virulent trophozoite kills cells by a rapid process that involves a contact-dependent cytolethal stage followed by phagocytosis. Experiments using artificial planar lipid bilayers have demonstrated increased conductance resulting from the spontaneous incorporation of amoeba-derived ion channels making the membrane permeable to Na, K, Ca and protons. These marked changes in cation permeability lead to cellular loss of ATP and the depolymerisation of microtubuli. The ion channel forming fraction is a small mass protein named amoebapore[37]. In a fully dissociated form the amoebapore is a basic hydrophobic protein with a molecular weight of 13-14,000 daltons, but exists within the trophozoite in a highly aggregated state as a small diameter highly dense particle[38]. It is exteriorised by a stimulus-mediated process. Therefore it has been suggested that immunological control of invasive amoebiasis might be possible by raising adequate blocking antibodies to pure amoebapore, or seeking cationic molecules that could block the ion channel e.g. polar guanidines and amidines[39].

E. histolytica invasive reinfections are extremely unusual (0.3%) in patients cured of amoebic hepatic abscess[40], and studies have shown that circulating antibodies, activation of the alternate pathway of complement[41], and cell-mediated immunity do participate in host defence against invasion[42]. However, the protective effect of circulating antibodies is intimately related with the immune avoidance properties of the amoeba's plasma membrane. The membrane exists in a dynamic state, and pinocytosis rates are such that the total membrane is internalised roughly every 20 minutes[43]. Addition of heat-inactivated immune sera to intact trophozoites induce rapid redistribution of immune complexes on the cell surface forming aggregates. The capped material is then either shed or internalised.[44] A major part of the surface redistribution involves strongly antigenic lipid and lipopeptidophosphoglycan components. The general concensus about acquired immunity in humans is that high titres of humoral antibodies present in patients with dysentery and invasive amoebiasis but do not alter the course of the disease nor do they prevent reinfection. It is not clear whether commensal carriers, many of whom are asymptomatic, remain apparently healthy because of acquired resistance.

References

1. Amoebiasis; Report of a WHO Expert Committee (1969). WHO Technical Report Series No. 421
2. MMWR (1981) 30 : 101
3. MJA (1983) 2 : 592
4. Medical Clinics of North America (1982) 66 : 689
5. Lancet (1985) 1 : 716
6. Rev. Inf. Dis. (1982) 4 : 1154
7. BMJ (1971) 3 :382
8. Arch. Inv. Med. (Mexico) (1971) 2 (Suppl. 1) : 327
9. NEJM (1984) 310 : 298
10. Am. J. Trop. Med. Hyg. (1968) 17 : 333
11. Cutis (1981) 27 : 278
12. Comp. Ther. (1980) 6 : 34
13. Sex. Trans. Dis. (1984) 11 : 59
14. Sex. Trans. Dis. (1978) 5 : 155
15. Br. J. Vener. Dis. (1979) 55 : 375
16. NEJM (1981) 305 : 603
17. CAMJ (1980) 123 : 512
18. Br. J. Vener. Dis (1984) 60 : 193
19. Scand. J. Inf. Dis. (1984) 16 : 199
20. Lancet (1982) 1 : 1386
21. Br. J. Vener. Dis. (1983) 59 : 193
22. NEJM (1982) 307 : 729
23. J. Immunol (1981) 126 :2084
24. Lancet (1983) 2 : 51
25. Trans. Roy. Soc. Trop. Med. Hyg. (1968) 62 : 285
26. Am. J. Trop. Med. Hyg. (1970) 19 : 755
27. J. Exp. Med. (1984) 160 : 353
28. Trans. Roy. Soc. Trop. Med. Hyg. (1984) 78 : 96
29. Trans. Roy. Soc. Trop. Med. Hyg. (1985) 79 : 86
30. Lancet (1985) 1 : 719
31. Nature (1971) 232 : 256
32. Trans. Roy, Soc. Trop. Med. Hyg. (1983) 77 : 76
33. Trans. Roy. Soc. Trop. Med. Hyg. (1983) 77 : 248
34. J. Virol (1977) 23 : 685
35. Lancet (1984) 2 : 528
36. Lancet (1984) 2 : 818
37. EMBO. Journal (1982) 1 : 801
38. Phil. Trans. Roy. Soc. Lond. (1984) 307 : 73
39. Vaccine (1984) 2 : 227
40. Arch. Invest. Med (Mex) (1970) 1 :(Suppl.) : S205
41. Clin. Exp. Immunol. (1978) 34 : 10
42. Rev. Inf. Dis. (1982) 4 : 1247
43. J. Exp. Med. (1976) 68 : 665
44. Martinez Palomo, A. (1982) The biology of Entamoeba histolytica. In: Tropical medicine research studies series (ed. K.N. Brown), vol. 2, Chichester:Research Studies.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 15/8/85 to 28/8/85 BULLETIN NUMBER 85/18
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		6		5			2	16
0101 ADENOVIRUS TYPE 1		1		1		5		2	9
0102 ADENOVIRUS TYPE 2	1	1				5			7
0103 ADENOVIRUS TYPE 3						1			1
0105 ADENOVIRUS TYPE 5								1	1
0106 ADENOVIRUS TYPE 6	1								1
0107 ADENOVIRUS TYPE 7	1								1
0108 ADENOVIRUS TYPE 8	1								1
0110 ADENOVIRUS TYPE 10		1							1
0199 ADENOVIRUS TYPING PENDING		2			2				4
0201 INFLUENZA A VIRUS	33	1	23			8		19	84
0202 INFLUENZA A VIRUS SUBTYPE H3N2				8	7				15
0203 INFLUENZA B VIRUS	2			12	20	9		10	53
0206 INFLUENZA A VIRUS SUBTYPE H1N1				1					1
0299 INFLUENZA VIRUS		3							3
0301 PARAINFLUENZA VIRUS TYPE 1				1	1	3			5
0302 PARAINFLUENZA VIRUS TYPE 2						2			2
0303 PARAINFLUENZA VIRUS TYPE 3	1			3	5			1	10
0399 PARAINFLUENZA VIRUS TYPING PENDING					2				2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	10	9	9	12	23	74		30	167
0500 RHINOVIRUS (ALL TYPES)	1			3	2	1			7
0600 MYCOPLASMA PNEUMONIAE	7		3			2		1	13
0700 ORNITHOSIS-PSITTACOSIS	3							2	5
0816 COXSACKIEVIRUS A16				1					1
0904 COXSACKIEVIRUS B4						2			2
1007 ECHOVIRUS TYPE 7				3					3
1009 ECHOVIRUS TYPE 9								1	1
1021 ECHOVIRUS TYPE 21				1					1
1100 POLIOVIRUS NOT TYPED			3						3
1101 POLIOVIRUS TYPE 1		1		1		1			3
1102 POLIOVIRUS TYPE 2		1							1
1103 POLIOVIRUS TYPE 3						1			1
1200 MUMPS VIRUS	2								2
1300 HERPES VIRUS GROUP-NOT TYPED	16		1	1		3		1	22
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1						2	3
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	2					2		4	8
1303 VARICELLA-ZOSTER VIRUS	6		1					1	8
1306 HERPES SIMPLEX TYPE 1	31			28				26	85
1307 HERPES SIMPLEX TYPE 2	103			61				58	222
1399 HERPES VIRUS TYPING PENDING					4	39			43
1401 COXIELLA BURNETI						2			2
1502 PICORNA VIRUS-NOT TYPED	12		2						14
1521 MEASLES VIRUS	1		1						2
1522 RUBELLA VIRUS			1	5				6	12
1532 HEPATITIS B ANTIGEN	37		7	16	1	20		20	101
1535 HEPATITIS A ANTIBODY			4	3		15		3	25
1541 CHLAMYDIA A - C TRACHOMATIS	24		4			22		57	107
1556 CMV - CYTOMEGALOVIRUS	10	3	2	31	2	9		3	60
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	43	10	7	1	23	8		1	93
1599 ENTEROVIRUS TYPING PENDING		1	1		8				10
9992 ROSS RIVER VIRUS								1	1
9994 SMALL VIRUS (LIKE) PARTICLE	1	1							2
Total	352	36	75	193	105	234		253	1,248

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 15/8/85 to 28/8/85 ....
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.....		5	1				4				
0101 ADENOVIRUS TYPE 1.....		3				1	3				
0102 ADENOVIRUS TYPE 2.....		5					2				
0103 ADENOVIRUS TYPE 3.....							1				
0105 ADENOVIRUS TYPE 5.....		1									
0106 ADENOVIRUS TYPE 6.....							1				
0107 ADENOVIRUS TYPE 7.....		1									
0110 ADENOVIRUS TYPE 10.....		1									
0201 INFLUENZA A VIRUS.....	14	49				1			4	1	2
0202 INFLUENZA A VIRUS SUBTYPE H3N2		14									
0203 INFLUENZA B VIRUS.....	1	44		1		1					
0206 INFLUENZA A VIRUS SUBTYPE H1N1		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		5									
0302 PARAINFLUENZA VIRUS TYPE 2....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	8									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	156									1
0500 RHINOVIRUS (ALL TYPES).....		7									
0600 MYCOPLASMA PNEUMONIAE.....	2	6				1			1		
0700 ORNITHOSIS-PSITTACOSIS.....		4									1
0816 COXSACKIEVIRUS A16.....											1
0904 COXSACKIEVIRUS B4.....							2				
1007 ECHOVIRUS TYPE 7.....		3									
1009 ECHOVIRUS TYPE 9.....		1									
1100 POLIOVIRUS NOT TYPED.....							3				
1101 POLIOVIRUS TYPE 1.....		2							1		
1102 POLIOVIRUS TYPE 2.....		1									
1103 POLIOVIRUS TYPE 3.....							1				
1200 MUMPS VIRUS.....	2										
1300 HERPES VIRUS GROUP-NOT TYPED..	4	1	1								7
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1							
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	2	2					1	1			
1303 VARICELLA-ZOSTER VIRUS.....	1					1			1		5
1306 HERPES SIMPLEX TYPE 1.....	3	12						1	2	1	23
1307 HERPES SIMPLEX TYPE 2.....	8		1								44
1502 PICORNA VIRUS-NOT TYPED.....	3	2		1			2		2		1
1522 RUBELLA VIRUS.....	3										2
1532 HEPATITIS B ANTIGEN.....	49						1	29			
1535 HEPATITIS A ANTIBODY.....	2							18			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1										
1556 CMV - CYTOMEGALOVIRUS.....	8	21		1		1		4	1	4	
1563 CORONAVIRUS.....		1									
1564 ROTAVIRUS.....		1					92				
1599 ENTEROVIRUS TYPING PENDING....		1									
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
Total.....	106	360	3	4		6	115	53	12	6	88

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 15/8/85 to 28/8/85 ...
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0100 ADENOVIRUS NOT TYPED.....									2	
0101 ADENOVIRUS TYPE 1.....									2	
0102 ADENOVIRUS TYPE 2.....							1			
0108 ADENOVIRUS TYPE 8.....	1									
0201 INFLUENZA A VIRUS.....				1			5	10	2	
0202 INFLUENZA A VIRUS SUBTYPE H3N2								2		
0203 INFLUENZA B VIRUS.....					1		2	8		2
0303 PARAINFLUENZA VIRUS TYPE 3....								1		1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....				2				5	4	
0500 RHINOVIRUS (ALL TYPES).....	1									
0600 MYCOPLASMA PNEUMONIAE.....			1				1		1	
1007 ECHOVIRUS TYPE 7.....								2		
1021 ECHOVIRUS TYPE 21.....								1		
1300 HERPES VIRUS GROUP-NOT TYPED..	1	6						1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED									1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			1	1				1		
1306 HERPES SIMPLEX TYPE 1.....	2	38						3	3	
1307 HERPES SIMPLEX TYPE 2.....		170								
1401 COXIELLA BURNETI.....							1		1	
1502 PICORNA VIRUS-NOT TYPED.....							2		1	
1521 MEASLES VIRUS.....							1	1		
1522 RUBELLA VIRUS.....						3		1	4	
1532 HEPATITIS B ANTIGEN.....								1	21	
1535 HEPATITIS A ANTIBODY.....									5	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	105								
1556 CMV - CYTOMEGALOVIRUS.....			1			3	5	1	18	
9992 ROSS RIVER VIRUS.....					1					
Total.....	6	319	3	4	2	6	18	38	65	3

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA
Period 5
20 April 1985 to 17 May 1985

Bulletin 85/18

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	2	14
Ankylostomiasis	–	–	–	2	–	–	–	–	2	18
Anthrax	–	–	–	–	–	–	–	–	–	–
Arbovirus infection	14	–	–	–	–	–	–	–	14	40
Brucellosis	–	–	1	–	–	–	–	–	1	5
Campylobacter infections	99	N.N.	N.N.	88	N.N.	N.N.	–	N.N.	187	968 *
Chancroid	–	–	–	N.N.	–	N.N.	–	–	–	–
Cholera	–	–	–	–	–	–	–	–	–	–
Congenital rubella syndrome	–	N.N.	N.N.	–	N.N.	N.N.	N.N.	N.N.	–	–
Diphtheria	–	–	–	–	–	–	–	–	–	–
Donovanosis	–	N.N.	2	N.N.	5	N.N.	1	–	8	39
Giardiasis	19	N.N.	N.N.	76	N.N.	N.N.	N.N.	N.N.	95	475 *
Genital herpes	44	N.N.	39	20	N.N.	N.N.	2	–	104	668
Gonococcal ophthalmia neonatorum	–	N.N.	–	–	N.N.	N.N.	2	N.N.	2	5
Gonorrhoea	168	82	104	40	170	8	74	7	653	3268
Hepatitis A (infectious)	10	2	13	7	6	1	3	–	42	268
Hepatitis B (serum)	39	2	18	14	34	–	3	4	114	594
Hepatitis - unspecified	8	–	–	1	2	N.N	1	–	12	47
Hydatid disease	2	–	–	–	–	–	–	–	2	4
Lassa Fever	–	–	N.N.	–	–	N.N.	N.N.	N.N.	–	1
Legionnaires' disease	–	–	N.N.	–	N.N.	N.N.	N.N.	N.N.	–	9
Leprosy	2	2	–	1	–	–	–	–	5	10
Leptospirosis	3	1	11	1	–	1	–	–	17	90
Lymphogranuloma venereum	–	N.N.	N.N.	N.N.	N.N.	N.N.	1	–	1	3
Malaria	10	7	23	7	3	–	–	2	52	288
Marburg Disease	–	–	N.N.	–	–	N.N.	N.N.	N.N.	–	–
Meningococcal infections	1	–	–	2	–	N.N.	–	–	3	15
Non-specific urethritis	324	N.N.	–	89	N.N.	N.N.	–	N.N.	413	1717
Ornithosis	–	–	–	–	1	–	–	–	1	3
Pertussis (whooping cough)	19	6	N.N.	1	N.N.	N.N.	N.N.	N.N.	26	222
Plague	–	–	–	–	–	–	–	–	–	–
Poliomyelitis	–	–	–	–	–	–	–	–	–	–
Q. fever	3	–	9	6	N.N.	–	N.N.	–	18	66
Rabies	–	N.N.	N.N.	–	–	N.N.	N.N.	N.N.	–	–

2.

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	92	7	25	38	17	5	24	–	208	1443 *
Shigella infections	8	4	12	7	14	–	23	–	68	363
Smallpox	–	–	–	–	–	–	–	–	–	–
Syphilis	26	1	24	14	29	2	81	–	177	859
Tetanus	–	–	–	–	–	–	–	–	–	2
Trachoma	–	N.N.	–	–	N.N.	N.N.	–	–	–	2
Tuberculosis (all forms)	36	21	13	3	12	–	1	1	87	377 *
Typhoid fever	1	1	–	–	–	–	–	–	2	14
Typhus (all forms)	–	–	–	–	–	–	–	–	–	–
Vibrio parahaemolyticus infections	–	N.N.	N.N.	–	N.N.	N.N.	N.N.	N.N.	–	4
Yellow Fever	–	–	–	–	–	–	–	–	–	–
Yersinia enterocolitica infections	6	N.N.	N.N.	2	N.N.	N.N.	N.N.	N.N.	8	14

(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 to the Cumulative Total since last report:

Campylobacter infections	+1	South Australia
Giardiasis	-1	South Australia
Salmonella infections	-2	South Australia
Tuberculosis (all forms)	+2	South Australia

Erratum: CDI 85/18 p1.

AIDS Surveillance - Australia: There have been no reports of AIDS in the Australian Capital Territory.
The table should read

	Cases	Deaths
Australian Capital Territory	-	-
Northern Territory	1	-