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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME: A total of 1 367 reports were processed for this period.

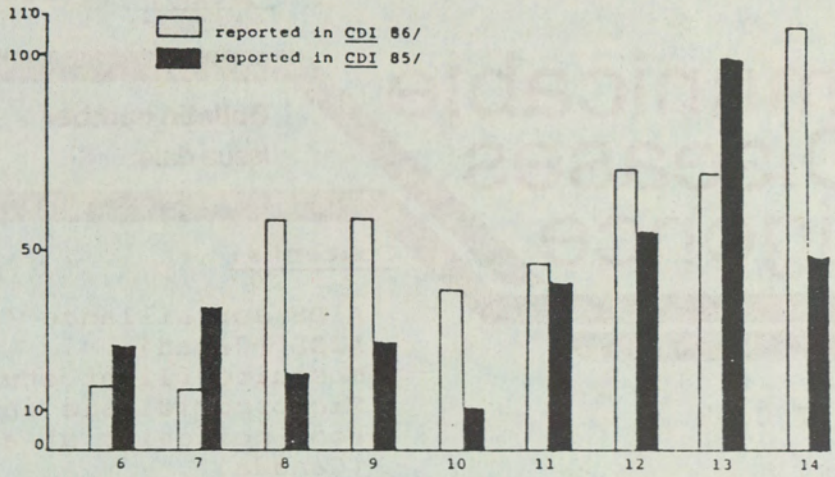
Herpes Simplex virus type 2 was isolated from the perianal ulcerations of two HIV (human immunodeficiency virus) antibody positive males aged 39 and 62 respectively.

Since the report in CDI 86/8 of an apparent increase in rotavirus infections, the activity of this virus has been monitored regularly in young children presenting with infantile gastroenteritis. One hundred and two cases of rotavirus associated diarrhoea have been reported for this period. The demographics of these cases are detailed below:

	SEX			
AGE	Female	Male	Unknown	TOTAL
0-1	32	43	2	77
2-5	7	11	-	18
6-10	-	3	-	3
Adults	-	4	-	4
TOTAL	39	61	2	102

Rotavirus infections are expected to predominate during the winter months with an incubation period of 2-4 days. Symptomatic infections, including diarrhoea, fever, abdominal pain and vomiting, leading to dehydration are most commonly seen in children between ages 6 months and 10 years, and transmission appears to be by faecal-oral route. Nocosomial infections are also frequent. A monitoring of this virus activity for the past quarter is compared with the corresponding period in 1985 (Figure).

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.



AIDS SURVEILLANCE - AUSTRALIA

To 3 July 1986, 240 cases of AIDS fulfilling the criteria of case definition have been reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification and by risk group are shown below:-

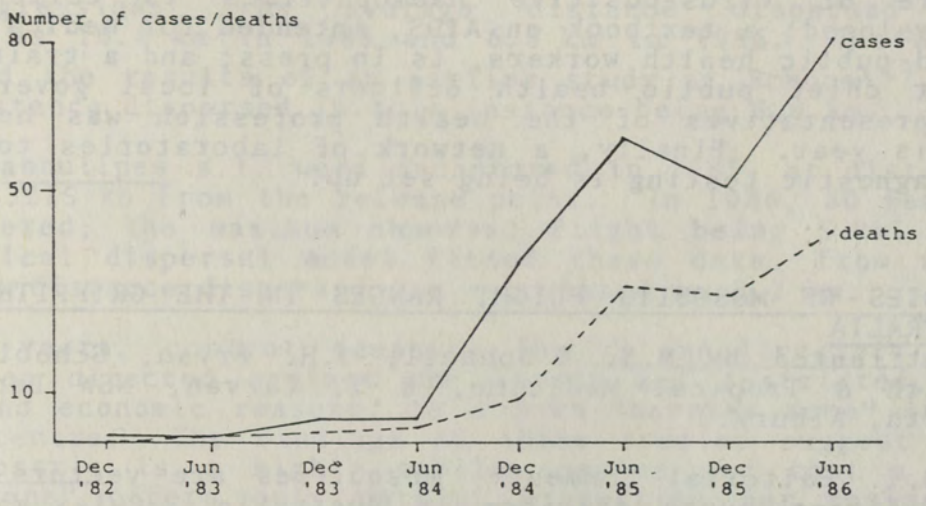
Table 1: AIDS patients by State or Territory of notification

STATE/TERRITORY	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
NSW	159	6	165	77	5	82
VIC	37	-	37	15	-	15
QLD	19	2	21	12	1	13
WA	9	2	11	5	-	5
SA	2	-	2	-	-	-
NT	2	-	2	1	-	1
TAS	1	-	1	1	-	1
ACT	1	-	1	-	-	-
	230	10	240	111	6	117

Table 2: AIDS patients by risk category

RISK GROUP	CASES	DEATHS
Homo-/Bi-sexual	210	94
IV drug abuser	1	-
Homo-/Bi-sexual IV drug abuser	3	2
Blood transfusion recipient	20	16
Person with haemophilia	3	3
Heterosexual transmission	2	2
None of the above	1	-
	240	117

Figure 1: Half yearly reports of new cases and deaths showing the disease progress



AIDS IN JAPAN
(Based on WER (1986) 23: 179)

To 31 January 1986, a total of 14 cases of AIDS had been reported in Japan.

In 1984 an AIDS Surveillance Committee was established to collect information on various aspects of the disease and to initiate and co-ordinate surveillance activities.

Since the discovery of the first case in March 1985, a number of prevention and control activities have been introduced. For example:

- all preparations of Factors VIII and IX are heat treated and the source of imported blood products certified free from HIV antibody;
- efforts are also directed towards the testing of blood donations and the exclusion of donors belonging to high-risk groups;
- information leaflets on the disease are available to the general public and to people at increased risk;
- guidelines for the daily life of virus-positive haemophiliacs are soon to be issued;
- consultation services for persons particularly concerned have been established in one centre and will be extended to others. These centres will be staffed by specially trained physicians and will be equipped with appropriate diagnostic testing facilities;
- measures are also being taken to ensure that physicians and other health workers who may come into contact with AIDS receive the information they require: case definitions and diagnostic guidelines were circulated nationwide in 1983; guidelines for the prevention of secondary spread from

infected persons have already been distributed to public health service authorities, while a procedures manual for the care of virus-positive haemophiliacs is currently being developed; a textbook on AIDS, intended for medical personnel and public health workers, is in press; and a training course for chief public health officers of local governments and representatives of the health profession was held earlier this year. Finally, a network of laboratories to carry out diagnostic testing is being set up.

STUDIES OF MOSQUITO FLIGHT RANGES IN THE GRIFFITH AREA, NSW AUSTRALIA

(Contributed by M.S. O'Donnell, J.H. Bryan, School of Public Health & Tropical Medicine, & T. Carvan, NSW Department of Health, Albury.)

[C.D.I. Editorial Comment: Mosquitoes are vectors of several significant human diseases in Australia. Culex annulirostris is the major vector of the viruses causing Australian Encephalitis (Murray Valley Encephalitis flavivirus and Kunjin flavivirus) and Epidemic Polyarthritis (Ross River alphavirus). Cx annulirostris is particularly important as the primary vector during epidemics of these diseases. Anopheles annulipes is a suspected, but unproven vector of malaria in Australia(1). An annulipes has recently been shown to be a sibling species complex, and in this article, An annulipes s.l. (Sensu lato) indicates that the taxon was treated as a whole and not analysed to the member species.

It is the female mosquitoes' requirement for a blood meal to provide protein for the maturation of each successive egg batch that has allowed these species to act as vectors for transmission of viruses and parasites from one blood meal host to a later host. In general, the pathogen must undergo some development in the vector before it can be transmitted. This is generally over a period of about 10-14 days. Females will disperse considerable distances from the breeding site in search of blood meals, and this dispersal has profound implications for the planning and execution of vector control and disease prevention programs. This report emphasises the fact that the currently accepted 5 km buffer zone around communities within which mosquito control programs are carried out may be inadequate to protect the residents from disease during an epidemic.]

The disperal of Culex annulirostris, a major arbovirus vector in Australia, and Anopheles annulipes were studied during February of 1985 and 1986 in Griffith and its environs. Female mosquitoes were caught in the field, labelled with fluorescent powders and released. Their subsequent movements were followed by a retrapping program using E.V.S. (Encephalitis Vector Surveillance) traps.

In 1985, 215 labelled Cx annulirostris were recovered, of which 19 had dispersed 5 km or more. The maximum flight recorded was 8.65 km. In 1986, 378 females were recaptured, of which 16 had travelled 5 km or more and 5 had travelled 10 km or more. The maximum flight range observed was 12 km, the limit of the retrapping grid. In both years, some individuals dispersed up to 7 km in a single day.

An empirical model of dispersal[2] gave a good fit to both sets of recapture data. Applying Hawkes'[3] approach to the fitted regressions, the average distance dispersed was calculated as 10.4 km in 1985 and 6.8 km in 1986. The model also fitted the results of an earlier study at Echuca[4], the average distance dispersed in this instance being 8.0 km.

Only 8 An annulipes s.l. were recaptured in 1985, at distances of up to 4.225 km from the release point. In 1986, 80 females were recovered, the maximum observed flight being 5 km. The same empirical dispersal model fitted these data, from which the average distance dispersed was calculated as 3.2 km.

In recent years, control measures for Cx annulirostris have usually been directed against the larvae and restricted, for logistic and economic reasons, to a 5 km "barrier zone" around an urban centre. The findings of these studies suggest that Cx annulirostris is a highly mobile species and that a 5 km "barrier zone" offers only partial protection for residents, especially where adult numbers are high and/or environmental conditions favour movement of females. An annulipes s.l. was found to be less mobile, so that, if needed, control measures within this limit should prove to be more effective.

1. Trans, Roy. Soc. Trop. Med. Hyg. (1983) 77:278-279
2. Ecol. Ent. (1987) 3:63-701
3. J. Appl. Ecol (1972) 9:617-632
4. Gen. Appl. Ent. (1986) 18:in press

MICROSPORIDIOSIS IN AIDS

(Based on CDR (1986) 21: 3-4)

Diarrhoea is a common feature in AIDS patients and is often responsible for rapid weight loss and death. Many organisms have been associated with the diarrhoea, including microsporidia.

Microsporidia are intracellular parasites reported in both vertebrates and invertebrates. Infection is thought to occur by the ingestion of tiny spores each containing a coiled polar filament. In the digestive tract of a host the filament is extruded and is thought to be involved in injecting the infective body, or sporoplasm, into host cells. Division by binary or multiple fission is followed by formation of sporonts. These divide into sporoblasts, which develop into mature spores capable of invading new host cells.

Microsporidial infection was reported recently in 6 AIDS cases.

The first patient was a 29 year old Haitian man with a 5 month history of diarrhoea, weight loss and epigastric pain, who denied any homosexual activity, drug addiction or blood transfusion. He had diffuse lymphadenopathy and acquired ichthyosis, and was shown to have a low T4:T8 lymphocyte ratio and no response to specific or non-specific mitogens. Intestinal function tests showed a low D-xylose absorbance, raised faecal fat, and raised alpha-1-antitrypsin, indicating malabsorption. He had herpes simplex facial ulcers, Mycobacterium kansasii, Pneumocystis carinii and

cytomegalovirus lung infection, pulmonary aspergillosis, Kaposi's sarcoma of the inguinal lymph nodes, fibrosis and necrosis of the adrenal glands and intestinal Giardia lamblia. Duodeno-jejunal and ileal biopsies showed partial villous atrophy, a mild inflammatory infiltration of the lamina propria, and 1-5 um round or ovoid basophilic intracellular bodies which were identified as microsporidia by electron microscopy. Treatment with pyrimethamine, sulphadiazine and metronidazole failed to eradicate the microsporidia or improve the intestinal function tests, although it did eliminate the Giardia. The patient died 16 months after the onset of symptoms.

The second patient was a homosexual man who had diarrhoea, weight loss and lymphadenopathy, and had a reduced T4:T8 ratio. He had P. carinii pneumonia, cryptococcal sepsis, perirectal cytomegalovirus, and intestinal G. lamblia. There were mild increases in the numbers of plasma cells and macrophages seen in the intestinal biopsies and electron microscopy of an intestinal biopsy showed intracellular microsporidia with some structural features similar to those in the first patient. The patient died two months after the diagnosis of microsporidiosis was made. An autopsy was not performed.

The third patient was a homosexual man in his 40s who had chronic secretory diarrhoea and cytomegalovirus infection. Microsporidia were demonstrated in intestinal biopsies by electron microscopy and there was damage to microvilli and some epithelial sloughing. The patient died 6 months after onset of his illness.

The fourth patient was a homosexual man in his 30s with chronic secretory diarrhoea, cytomegalovirus infection and Kaposi's sarcoma of the stomach. Intestinal microsporidia were demonstrated by Brown and Bran Gram stain and electron microscopy. The patient died three months after presentation.

The fifth patient was a homosexual man in San Francisco who had AIDS and intestinal microsporidiosis.

The sixth patient was a 20 year old man who admitted to one homosexual contact one year before admission. He had generalised muscle weakness with contractures for seven months, generalised lymphadenopathy, weight loss, purulent nasal discharge, testicular atrophy, and gynecomastia. He had a reduced T4:T8 ratio, decreased in vitro lymphocyte transformation, and delayed skin tests were nonreactive. The muscles were firm but not inflamed, and electromyographic and nerve conduction findings indicated diffuse myopathy. Testicular biopsy was refused but biopsies were taken from both the quadriceps and deltoid muscles. There was scarring and fibrosis with intense inflammatory reaction of plasma cells, lymphocytes, and histiocytes. Atrophic and degenerating muscle fibres were infiltrated by spores in clusters of at least 12. Spores were 2.8 um by 3.2-3.4 um on haematoxylin and eosin stain, and had a dark centre and relatively unstained ends, with a dot or line in the anterior unstained end. Many spores were acid fast by Ziehl-Nielsen stain, and had a periodic-acid-schiff positive granule at one end. The polar filament was seen inside spores by electron microscopy.

FOOD POISONING AT A SUMMER CAMP

(Based on Diseases Surveillance Vol 7/No 7, 18 January 1986)

In August 1985, an outbreak of food poisoning occurred in a summer camp operated by Boy Scouts of Canada. The camp began on a Sunday afternoon and the first symptoms of illness were experienced that evening. Of 41 persons at risk, 39 were interviewed, and of these, 24 persons experienced varying symptoms of nausea, vomiting, diarrhoea, cramps and headaches. The mean incubation period was 24 hours and ranged from one hour to 36 hours.

Food specific attack rates, calculated for all food items (ham, cold roast beef, scalloped potatoes, peas, coleslaw, bread and butter, milk and squares) available at the only meal served prior to the onset of symptoms, implicated ham as the suspected vehicle. In this instance the preparation and cooking of ham did not conform to the safe food handling practices. The three hams had been transferred from the freezer to the oven, arranged closely together in one pan, and cooked for approximately nine hours at a low temperature. No meat thermometer was used to ensure safe internal temperatures of the hams.

The drinking water source was subsequently switched from the untreated lake water which was used up until during the outbreak, to the drilled well, serving the camp caretaker's residence. No faecal coliform organisms were found in the lake water samples but total coliform counts exceeded the prevailing Drinking Water Standards. A bacteriological examination of the well water proved negative for both total and faecal coliforms. Chemical water quality from both sources was within the standards but the camp was required to install an approved waterworks system in accordance with the Health Act.

Six stool samples were cultured yielding 2 specimens with heavy growth of Clostridium perfringens, one with moderate growth of Staphylococcus aureus and one with growth of both organisms. No food poisoning organisms were isolated in the other two specimens, one of which was from the asymptomatic food handler. However, food from the meal was not available for testing.

Of the 24 persons reported ill, 23 had headaches. However, headaches are not a common symptom consistent with illness caused by C. perfringens or S.aureus. Some headaches were recurring and had onset related to times spent in the main pavilion, which suggested the possibility of carbon monoxide poisoning. A Draeger multi-gas detector was used to test the pavilion for the presence of CO but no levels were detected. No other possible sources capable of producing CO in the pavilion were identified. A further review of Camp medical records was undertaken in an attempt to determine possible causes of handaches, other than food poisoning.

TOXIC SHOCK SYNDROME (TSS) - UPDATE

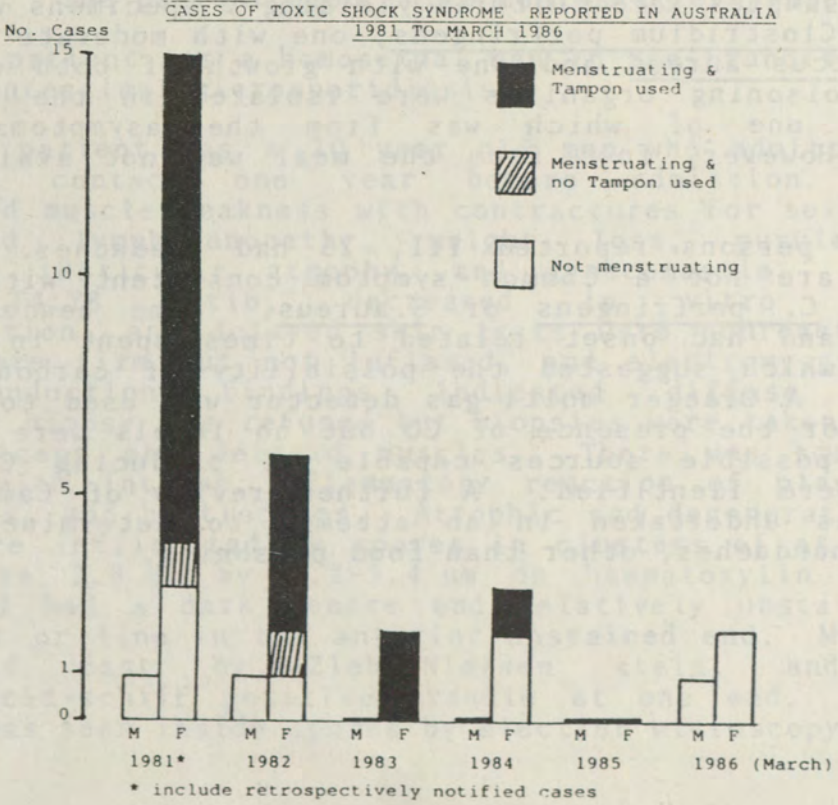
The Toxic Shock Syndrome (TSS) was first described in 1978 as a condition characterised by toxaemia associated with Staphylococcus aureus[1]. In 1980, TSS was reported in the United States in cases involving mostly menstruating women, thus implicating TSS with tampon use[2].

TSS has an abrupt onset of high fever, vomiting, diarrhoea, myalgias, a scarlatiniform rash, and hypotension with cardiac and renal failure in the most severe cases. Toxic shock syndrome occurs often within 5 days of the onset of menses in young women who use tampons, but it also occurs in children and in men with postoperative staphylococcal wound infections. The Syndrome can recur and is tentatively attributed to staphylococcal toxin. TSS-associated staphylococci has been found on tampons, in wounds, or in the throat but virtually never in the bloodstream. These organisms usually produce enterotoxin F and exotoxin C which may be antigenically related or identical[3].

Following the widespread publicity given to TSS in Australia in 1980-81, the first cases were reported in Melbourne and Launceston in January 1981. Subsequently 11 cases were reported for that year, including 1 case who had a recurrence four months later, and 4 cases (1 male) which were diagnosed retrospectively back to 1974. Figure 1 collates the number of cases reported since 1981.

No cases were reported in 1985. It is possible that cases of severe toxaemia were not diagnosed as TSS when there was no link with tampon use. Secondly, although tampons have been linked with TSS, cases are usually admitted through intensive care units, not through gynaecology wards.

FIGURE 1
CASES OF TOXIC SHOCK SYNDROME REPORTED IN AUSTRALIA 1981 TO MARCH 1986



* include retrospectively notified cases

Although the cases reported in the early years were predominantly in women and related to tampon use, the 6 cases reported since 1984 did not show these characteristics:

- only 1 case was associated with tampon use
- 1 case was male
- 3 cases were associated with surgery
- 2 of 4 cases occurred mid-menstrual cycle with no apparent precipitating cause.

Of a total of 31 cases reported since 1981, two cases have been fatal. Both women were in their early forties, but only one case was associated with tampon use. Such a mortality rate is statistically higher than that reported from the United States. However, in one other TSS case involving a 23 year old male, a routine surgery to the knee performed in 1983 resulted in a 2 year illness with severe residual neurological damage and a permanent handicap.

Since the reporting of TSS cases has been voluntary, reminders were issued in late 1985 to State Health Authorities and major public and private hospitals throughout Australia, seeking information on cases of TSS admitted since 1984. Responses to date are as follows:

	Reminders issued	Responses (as at 30/5/86)
State Authorities	8	6
Teaching Hospitals	36	29
Large private hospitals	24	11
Large public hospitals	55	44

TSS in Australia has not become as significant a problem as in the United States. The advice given on tampon use, the changing patterns of menstrual care and the use of the small adhesive pads may all be factors in maintaining the low level of tampon related TSS incidence among Australian women. Nevertheless there is no apparent reason to justify relaxing either the advice or the concern on quality and safety of tampons, and attention should be paid to the seriousness of the condition by including it in the differential diagnosis of any sudden unexplained febrile illness with shock, whether related to tampon use or not. The Department of Health is still interested to hear of cases of TSS occurring in Australia. Reports should be forwarded to:-

Dr Cathy Mead
Medical Services Adviser
Women's Health and Family Planning
Commonwealth Department of Health
PO Box 100
WODEN ACT 2606

REFERENCES

1. The Lancet (1978) Nov. 25:1116-18
2. MMWR (1981) 30:563-70
3. Infect Immun (1983) 40:1023

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 23/6/86 - 6/7/86 BULLETIN NUMBER 86/14
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	8	1	4		1	5	9		28
0101 ADENOVIRUS TYPE 1				1		1		2	4
0102 ADENOVIRUS TYPE 2			1	1					2
0104 ADENOVIRUS TYPE 4						1			1
0105 ADENOVIRUS TYPE 5						1			1
0106 ADENOVIRUS TYPE 6		1							1
0108 ADENOVIRUS TYPE 8	1		2	4					7
0199 ADENOVIRUS TYPING PENDING			2		1				3
0203 INFLUENZA B VIRUS								1	1
0301 PARAINFLUENZA VIRUS TYPE 1				1	8	1	6		16
0302 PARAINFLUENZA VIRUS TYPE 2	2	1		2	11	3			19
0303 PARAINFLUENZA VIRUS TYPE 3	1			2	5	6	1		15
0399 PARAINFLUENZA VIRUS TYPING PENDING		1						1	2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	16	14	5	4	20	3	18	1	81
0500 RHINOVIRUS (ALL TYPES)	1			2	9	14	1		27
0600 MYCOPLASMA PNEUMONIAE	7							1	8
0700 ORNITHOSIS-PSITTACOSIS	1					2			3
1002 ECHOVIRUS TYPE 2								1	1
1005 ECHOVIRUS TYPE 5								1	1
1006 ECHOVIRUS TYPE 6						1			1
1007 ECHOVIRUS TYPE 7	1	1							2
1011 ECHOVIRUS TYPE 11	1							11	12
1013 ECHOVIRUS TYPE 13	1								1
1014 ECHOVIRUS TYPE 14	1								1
1101 POLIOVIRUS TYPE 1						2			2
1102 POLIOVIRUS TYPE 2						1			1
1200 MUMPS VIRUS	1			1				5	7
1300 HERPES VIRUS GROUP-NOT TYPED	29		1	5		1			36
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1							1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5		2					8	15
1303 VARICELLA-ZOSTER VIRUS	1		1	1				1	4
1306 HERPES SIMPLEX TYPE 1	31			48		28	32	14	153
1307 HERPES SIMPLEX TYPE 2	100			77		24	77	57	335
1399 HERPES VIRUS TYPING PENDING					4				4
1401 COXIELLA BURNETI	1								1
1502 PICORNA VIRUS-NOT TYPED	3		1				15	1	20
1514 MOLLUSCUM CONTAGIOSUM								2	2
1521 MEASLES VIRUS	2			2	1				5
1522 RUBELLA VIRUS								8	8
1532 HEPATITIS B ANTIGEN	48	1	10	19		39	8	11	136
1535 HEPATITIS A ANTIBODY	4			9		2		27	42
1541 CHLAMYDIA A - C TRACHOMATIS	20		2	51		57	23	25	178
1556 CMV - CYTOMEGALOVIRUS	2	5	2	16	3	8	9	6	51
1564 ROTAVIRUS	31	3	4	6	2	55		1	102
1571 ENTEROVIRUS TYPE 71 (BRCR)				2					2
1599 ENTEROVIRUS TYPING PENDING		1	8		4				13
9992 ROSS RIVER VIRUS								1	1
9993 ASTROVIRUS	1								1
9994 SMALL VIRUS (LIKE) PARTICLE	1	8							9
Total	321	38	45	254	69	255	199	186	1,367

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 23/6/86 - 6/7/86
Viral Identifications by Clinical Information Table I.
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					3				
0101 ADENOVIRUS TYPE 1.....		1				1	2				
0102 ADENOVIRUS TYPE 2.....							2				1
0104 ADENOVIRUS TYPE 4.....		1									
0105 ADENOVIRUS TYPE 5.....							1				
0106 ADENOVIRUS TYPE 6.....							1				
0199 ADENOVIRUS TYPING PENDING.....		1									
0203 INFLUENZA B VIRUS.....		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		16									
0302 PARAINFLUENZA VIRUS TYPE 2....	1	18									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	14									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	78							1		
0500 RHINOVIRUS (ALL TYPES).....	1	26									
0600 MYCOPLASMA PNEUMONIAE.....	1	5							1		
0700 ORNITHOSIS-PSITTACOSIS.....		2									
1002 ECHOVIRUS TYPE 2.....							1				
1005 ECHOVIRUS TYPE 5.....		1									
1006 ECHOVIRUS TYPE 6.....					1						
1007 ECHOVIRUS TYPE 7.....	1										1
1011 ECHOVIRUS TYPE 11.....	1	2				5	4				
1013 ECHOVIRUS TYPE 13.....		1									
1014 ECHOVIRUS TYPE 14.....							1				
1101 POLIOVIRUS TYPE 1.....	1						1				
1200 MUMPS VIRUS.....		5						1			1
1300 HERPES VIRUS GROUP-NOT TYPED..			1								
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	5	2				1		1			
1303 VARICELLA-ZOSTER VIRUS.....											3
1306 HERPES SIMPLEX TYPE 1.....	3	9					1	1		2	71
1307 HERPES SIMPLEX TYPE 2.....	10							1		1	73
1502 PICORNA VIRUS-NOT TYPED.....	1	6				2	2				
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....		2	1						1		1
1522 RUBELLA VIRUS.....	1										6
1532 HEPATITIS B ANTIGEN.....	30							73			
1535 HEPATITIS A ANTIBODY.....	19							23			
1541 CHLAMYDIA A - C.TRACHOMATIS...	10										
1556 CMV - CYTOMEGALOVIRUS.....	8	13		1		3		2	1	2	
1564 ROTAVIRUS.....							100				
1571 ENTEROVIRUS TYPE 71 (BRCR)....							1				1
9993 ASTROVIRUS.....							1				
9994 SMALL VIRUS (LIKE) PARTICLE...							9				
Total.....	96	205	2	2		12	130	102	4	5	160

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 23/6/86 - 6/7/86
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
0108 ADENOVIRUS TYPE 8.....	7									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....									1	
0600 MYCOPLASMA PNEUMONIAE.....								1		
0700 ORNITHOSIS-PSITTACOSIS.....								1		
1011 ECHOVIRUS TYPE 11.....								2		
1102 POLIOVIRUS TYPE 2.....										1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			3	3			1	1	1	
1303 VARICELLA-ZOSTER VIRUS.....								1		
1306 HERPES SIMPLEX TYPE 1.....	3	61						1	3	1
1307 HERPES SIMPLEX TYPE 2.....		252							2	
1401 COXIELLA BURNETI.....									1	
1502 PICORNA VIRUS-NOT TYPED.....	1									1
1522 RUBELLA VIRUS.....					2			1	1	
1532 HEPATITIS B ANTIGEN.....									33	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	165							2	
1556 CMV - CYTOMEGALOVIRUS.....		5				5	1	2	11	
1564 ROTAVIRUS.....										1
1571 ENTEROVIRUS TYPE 71 (BRCR)....								2		
9992 ROSS RIVER VIRUS.....					1					
Total.....	12	483	3	3	3	5	2	12	55	4

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 2 - 25 January 1986 to 21 February 1986

Bulletin ...86/14

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			3				5	7
Ankylostomiasis				1			N.N.		1	* 2
Anthrax										-
Arbovirus infection	22	6	37		1				66	145
Brucellosis		1	1						2	5
Campylobacter infections	76	N.N.	N.N.	99	6	N.N.	9	N.N.	190	432
Chancroid				N.N.		N.N.				-
Cholera										-
Congenital rubella syndrome		N.N.	N.N.			N.N.		N.N.		-
Diphtheria							2		2	5
Donovanosis		N.N.		N.N.	2	N.N.			2	7
Giardiasis	23	N.N.	N.N.	63	3	N.N.	N.N.	N.N.	89	* 164
Genital herpes	109	N.N.	3	16	N.N.	N.N.			128	212
Gonococcal ophthalmia neonatorum		N.N.	N.N.		N.N.	N.N.		N.N.		-
Gonorrhoea	95		43	49	129	8	46	5	375	734
Hepatitis A (infectious)	33	15	16	29	45		3	2	143	267
Hepatitis B (serum)	43	19	34	7	25		1	12	141	265
Hepatitis - unspecified	7	2	5			N.N			14	26
Hydatid disease										1
Lassa Fever		N.N	N.N.			N.N.	N.N.	N.N.		-
Legionnaires' disease	2	1	N.N.	14		N.N.		N.N.	17	27
Leprosy										3
Leptospirosis	3	4				2			9	24
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.				-
Malaria	17	6	28	1	1	1	2	3	59	* 121
Marburg Disease		N.N.	N.N.			N.N.	N.N.	N.N.		-
Meningococcal infections	2	1			1	N.N.			4	6
Non-specific urethritis	315	N.N.	2	100	1	N.N.		N.N.	418	743
Ornithosis				2					2	* 6
Pertussis (whooping cough)	50	7	N.N.	15	18	N.N.		N.N.	90	* 223
Plague										-
Poliomyelitis										-
Q. fever	2		3	2	1		N.N.		8	23
Rabies		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	75	16	38	24	27	9	36		225	* 459
Shigella infections	16		13	8	13		22		72	143
Smallpox										-
Syphilis	41		5	17	22	1	68		154	262
Tetanus				1					1	1
Trachoma		N.N.				N.N.	N.N.			4
Tuberculosis (all forms)	10	20		6	13			1	50	106
Typhoid fever	4	1	1						6	* 9
Typhus (all forms)										-
Vibrio parahaemolyticus infections	1	N.N.	N.N.			N.N.		N.N.	1	3
Yellow Fever										-
Yersinia enterocolitica infections	7	N.N.	N.N.			N.N.		N.N.	7	11

(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:

Ankylostomiasis	+ 1	South Australia
Giardiasis	+ 4	South Australia
Malaria	+ 1	South Australia
Ornithosis	+ 1	South Australia
Pertussis (whooping cough)	+ 14	South Australia
Salmonella infections	+ 1	South Australia
Typhoid fever	+ 1	South Australia

IMPORTANT NOTICE

Our ref 86/4465

MAILING LIST UPDATE - 1986

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