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Communicable Diseases Intelligence

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Contents:

This is the final issue of CDI for 1983, and includes the subject index for the year. The next issue will be published on 13 January 1984. The editorial staff takes this opportunity to extend seasonal greetings to all readers, with best wishes for the New Year.

VIRUS REPORTING SCHEME - A total of 1478 reports were received this period.

. Arbovirus infections - Specific IgM antibody against dengue type 1 was detected by the State Health Laboratory, Brisbane, in a 78 year old male from Ipswich, approximately 30Km south-west of Brisbane. The patient, who had no history of overseas travel, presented with thrombocytopenia, headache, lethargy fever and myalgia. A broad cross-reactivity against MVE, Kunjin and Alfuy antigens was also detected in a 37 year old male with arthralgia from Woodstock, Queensland.

As a component of the 1983-84 Victorian arbovirus surveillance program, 200 sero-negative chickens, apportioned into ten sentinel flocks, were established at Mildura, Robinvale, Swan Hill, Kerang, Echuca, Barmah, Shepparton, Cobram, Rutherglen and Wodonga on 21 November 1983. Of those flocks tested to 9 December, all chickens were negative in HI tests for antibody against MVE, Kunjin and Sindbis antigens.

Other reports of interest include :-

. Prior to the report in CDI83/22 of three cases of systemic candidiasis after intravenous abuse of heroin, a similar incident was published in BMJ (1983) 287 : 861 (T.C. Sorrell, Westmead Hospital, Sydney; personal communication). Seven young males presented to Westmead Hospital in September 1982 with disseminated candidiasis after a single episode of intravenous heroin abuse. The patients had a distinctive syndrome of hepatitis followed by eye lesions, skin lesions, osteomyelitis and costochondritis.

. A 60 year old woman who returned recently from India was admitted to Fairfield Hospital, Melbourne, with severe

(continued on page 7)

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CREUTZFELDT-JAKOB DISEASE (CJD) SURVEILLANCE

Subsequent to the review of CJD in CDI 83/3, data on Australian cases collated by two study groups in Perth and Sydney are published below. As the earlier review contained some misconceptions of this transmissible dementia, an update is also included.

CJD SURVEILLANCE - WESTERN AUSTRALIA

(Contributed by C.L. Masters, Department of Neuropathology, Royal Perth Hospital, Western Australia.)

In those countries where efforts have been made to document CJD, suspected approximate annual incidences of 1-2 cases/million have been recorded[1]. Subsequent intensive efforts at careful case identification in France, Chile, Japan, England and Italy have not yielded substantially higher incidence rates.

The first adequately documented cases of CJD in Australia were described by Rischbeith in Adelaide in 1963[2]. In 1979, a postal survey of all neurologists and neuropathologists in Australia and New Zealand was conducted, and although their responses indicated that pathologically verified cases of CJD occurred throughout the two countries, the number of cases identified was small.

A careful retrospective review of all the diagnosed cases in Western Australia is now being done. The first two cases were diagnosed in 1971 and 1975, and in addition to pathological verification, virus causing spongiform encephalopathy (based on primate inoculation experiments performed at the National Institutes of Health, USA) was isolated from both cases. In the 12 years since 1971, ten cases of CJD (7 males and 3 females, with a mean age at death of 55 years) have been recorded. Diagnosis has been verified pathologically in seven cases, and two cases are pending. Two additional cases diagnosed clinically were excluded after pathological review. Table 1 gives the estimated incidence rates for Western Australia.

TABLE 1 Incidence of CJD in Western Australia(1971-83)*

	Annual incidence (per million population)
Based on whole population of Western Australia	0.71
Based on population of Perth Metropolitan Statistical Division	0.99
Based on whole population of Western Australia aged ⩾ 40 years	2.26
Based on population of Perth Metropolitan Statistical Division aged ⩾ 40 years	3.14

* Ten cases over of 12 years using 1976 Census figures.

Although the crude annual incidence is 0.71 cases/million, all the cases resided within the Perth Metropolitan Statistical Division, so that 1 case/million/year is a more appropriate estimate. The age specific rate exceeds 3 cases/million/year.

A complete assessment of the clinical and pathological features of these cases is also in progress, but few unusual aspects have emerged to date. One case presented a diagnostic problem because of a clinical evolution similar to progressive supranuclear palsy (Steele-Richardson syndrome). No familial

cases have yet been identified, but this may be attributed to the small numbers examined[3]. Investigations of occupational, social, residential and past medical and surgical histories are also in progress, but preliminary results have again shown no evidence of temporal-spatial clustering, nor any occupational risk factors among the cases. However, travel histories of some cases have allowed the firm statement that infection must have been acquired within Western Australia, since they had never travelled outside the State. In addition, review of the medical and surgical histories has shown ample opportunity for possible iatrogenic transmission for most cases, but no common source of infection for any of these cases has been identified to date.

Because Perth has a geographically isolated population, an epidemiological study of CJD in this community has several distinct advantages. Even a study within Australia and New Zealand provides the unique opportunity to exclude one major hypothesis of CJD natural transmission i.e. the zoonotic transmission of scrapie virus from sheep, goats or other animals. With the exception of a possible outbreak in Victoria in 1950-52[4], scrapie is unknown in Australia. Therefore, most CJD patients in Australia must have acquired their infection by one of two mechanisms:-

- Activation of an endogenous (latent) virus through an environmental trigger or some genetically determined mechanism.
- Person-to-person transmission (which includes lateral contact transmission, vertical or familial transmission and transmission resulting from iatrogenic contamination.).

Of these two mechanisms, the available evidence favours some form of person-to-person transmission. Iatrogenic spread of CJD is the only mechanism that has been clearly demonstrated, albeit in a very small proportion of all cases, and the known biological and physio-chemical properties of CJD virus are consistent with an iatrogenic spread in sporadic cases through inadvertent contamination of surgical instruments, scalp electrodes, venipuncture needles etc. However, the uncovering of this form of transmission in a disease with an incubation period exceeding three decades poses formidable epidemiological problems.

A strong case could be made for making CJD a notifiable disease, since with the co-operation of neurologists, neuropathologists, psychiatrists and general physicians, and the existing criteria for diagnosis, the true incidence in Australia could be ascertained. In addition the geographic isolation of the country should allow possible reconstruction of most of the significant epidemiological features, particularly since it is expected that the true incidence of CJD will increase over the next decade. However, proper case identification and subsequent attention to adequate decontamination of instruments and other materials which may be sources of secondary infection may lead to a reduction in the incidence of the disease. Finally, the demonstration of infectivity of the CJD virus in goats, may indicate a possibility for a reverse zoonotic spread of human CJD virus to sheep with subsequent economic loss to the sheep industry.

References

1. Ann. Neurol. (1979) 5 : 177
2. Proc. Australian Assoc. Neurologists (1965) 3 : 11

3. Brain (1981) 104 : 535
4. Aust. Vet. J. (1958) 34 : 213
5. Ann.Neurol. (1980) 8 : 628

CJD SURVEILLANCE - NEW SOUTH WALES

(Contributed by J.M. Worthington, Base Hospital, Dubbo; and S.M. Stone, Rachel Forster Hospital, Redfern, Sydney).

In Australia, as elsewhere, CJD affects a wide range of ages from 23-83 years, although it does have a particular predilection for the 55-70 year age group. Since identifying initial infection among patients is extremely rare, the mode of spread and inoculation period are uncertain. The only proven means of transmitting CJD is by inoculation or ingestion of infected material[1]. After close study of Kuru among the Fore people of Papua New Guinea and observations of animal colonies, there is no evidence that these spongiform encephalopathies are communicable.

A comprehensive search for cases of CJD in New South Wales during 1970-80 has revealed over 30 cases (Worthington and Stone, in preparation). The incidence of 0.66 cases/million/year is similar to that found in many countries e.g. USA (0.26-0.43), France (0.59-1.29), England and Wales (0.09), Chile (0.73), Czechoslavakia (0.28), Hungary (0.18), Japan (1.10) and Italy (0.05).

Reference

1. Epidemiological Reviews (1980) 2 : 113

CJD - UPDATE

(Based on material contributed by J.M. Worthington, Base Hospital, Dubbo; and S.M. Stone, Rachel Foster Hospital, Redfern, Sydney).

CJD is an inevitably fatal, rapidly progressive dementia with focal neurological signs, a characteristic EEG and the potential for iatrogenic spread[1]. A transmissible slow virus infection, CJD belongs to the acute spongiform encephalopathies comprising kuru of man, scrapie of sheep and goats and transmissible mink encephalopathy. These degenerative central nervous system (CNS) diseases possess similar infective agents, natural history and pathology. Although subacute sclerosing panencephalitis and progressive multifocal leukoencephalopathy are diseases with similar slowly progressive courses, their clinical features are quite distinct from those of CJD.

Pathology - Pathological changes are restricted to the CNS where there is a notable absence of inflammatory changes. Affected brains show an exclusive triad of neuronal loss, astrocytosis and spongiform change. Unique membrane - bound vacuoles form in the neuropil of affected brain. The vacuoles contain membrane fragments and give histological sections their sponge-like appearance. Cortical regions, the cerebellum and the pyramidal and extrapyrimidal systems may all be affected. As cell loss and glial scarring progress, unbound vacuolation of the extracellular brain substance may occur, giving rise to the appearance of status spongiosus.

Clinical Features - The most common presenting symptoms of CJD are dementia, ataxia, behavioural, visual and speech changes, vertigo, weakness, malaise, weight loss, tremor and seizures. Dysphagia is not common. As the disease progresses, dementia worsens and diverse neurological defects appear.

Transient or permanent mono- or hemiparesis, aphasias, apraxias, agnosias, sensory deficits and symptoms, altered muscle tone, incoordination, rigid posturing, opisthotonos, ataxia and choreiform and athetoid movements may all be seen in patients with CJD. The existence of an amyotrophic form of CJD is controversial. Startle reactions and the abrupt sudden muscle movements of myoclonus are dramatic features often seen in the illness. Although common, the spontaneous, epileptiform movements and the elicited startle reactions are not pathognomonic for CJD nor necessary to the diagnosis.

Patients with CJD become increasingly vegetative, and usually succumb to intercurrent infections. The average duration of illness among Australian cases is ten months, but the disease may progress rapidly to death within seven weeks. There is no known effective treatment. Brain biopsy is the only definitive ante-mortem diagnostic investigation. There is no serological or biochemical test diagnostic of CJD; CSF indices are unhelpful, and even electroencephalographic changes are not specific or mandatory. Characteristically, there are diffuse paroxysmal sharp wave complexes on the EEG trace, occurring at intervals of 0.5 to a few seconds. These periodic complexes are superimposed on an abnormal background of theta and delta activity. As the clinical state progresses the amplitude of the slow wave activity declines and the interval between spike wave complexes may increase.

Handling Procedures - Resistant as the CJD agent is, it can be inactivated in usual samples by autoclaving at 121°C for one hour and by sodium hypochlorite solutions (0.5-5%). It has been dealt with in the laboratory for many years using these techniques. Only specially prepared sonicated materials seem to present any problems[3]. Table 1 tabulates the inactivation times for CJD and scrapie by various techniques.

Caution is required in the clinical setting. Health professionals do not appear to have a high incidence of CJD. Nonetheless, the disease is transmissible iatrogenically, and has been reported in a neurosurgeon, two physicians, six nurses, a nurses' aide and four dental professionals[4]. No person-to-person transmissions have been identified among the health worker cases. Various handling procedures are outlined in Table 2[3,5], and mortuary workers and funeral directors as well as pathologists and paramedical staff should be aware of them. CJD is not a communicable disease, and isolation of patients seems of little value. Barrier techniques, routine biohazard techniques and needle precautions are adequate. Abrasion of hands by vigorous brushing should be avoided during hand washing. Inoculation is the probable mode of spread. Gown, gloves and masks are recommended for invasive procedures including autopsy. Body fluids and contaminated fluids, including fixatives, should all be autoclaved or disinfected before disposal. Infected tissues, including brain, CSF, spinal cord, kidney, lens, liver, lung, lymph node, spleen and cornea are infective, even when formol-fixed. One gram of brain alone can contain 10^8 lethal doses of CJD. Transplantation of organs from patients with suspected CJD must be avoided. Proposed organ donation from any demented patient should be reviewed carefully.

References

1. CDI (1983) 83/3 : 2
2. Neurology (1981) 31 : 538
3. Infectious Diseases (1982) 145 : 683
4. Annals Neurology (1978) 5 : 177
5. NEJM (1982) 306 : 1279

TABLE 1 Chemical inactivation of CJD and scrapie virus*

CHEMICAL	CONC**	TIME	EFFECTIVENESS+	RATING	REF.
Sodium hypochlorite	1.3% W/V	1 hour	++++	R	C
Sodium hypochlorite	0.5% W/V	1 hour	++++	R	C,S
Sodium metaperiodate	0.01 M	1 hour (in dark)	+++	NI	S
Chlorine dioxide	50ppm V/V	1 hour	++	NR	S
Lysol (Phenol 0.5%)	10% V/V	1 hour	++	NR	S
Iodine	2% W/V	1 hour	++	NR	S
Potassium permanganate	0.4% W/V	45 min	+++	NI	C
Potassium permanganate	0.1% W/V	1 hour	++	NR	C,S
Formaldehyde	3.7% W/W	4 hours	++	NR	S
Formaldehyde (fixed brain)	3.7% W/W	-	-	NR	C
Hydrogen peroxide	3.0% V/V	1 hour	++	NR	S
Ethylene oxide	88% V/V	1 hour	++	NR	C
Triton X-100	1% W/V	-	-	NR	C
Sodium deoxycholate	5% W/V	-	-	NR	C
Histological processing		-	++	NR	S

Key
R - Recommended
NI - Not ideal
NR - Not recommended
S - Scrapie
C - CJD
* Autoclaving should be used as an adjunct where possible
** Concentration of antiseptic; not the final concentration in the preparation
+ effective reduction of infectious titres in brain homogenates

TABLE 2 Handling procedures for CJD

CONTAMINATION	
Large surfaces	SH
Fabrics	A
Sensitivity	A
Durable instruments, glassware, plastics etc. including dental instruments	SH and A
LP needles, IV lines, venipuncture needles, catheters and manometers	SH and A,I
CSF/blood counting chambers	SH
Solutions including fixatives	A or SH and D
TISSUES AND BODY FLUIDS	
Body secretions	N1, HW
Blood*, CSF, tissues, wound drainage etc.	T1, ND, A or SH
Percutaneous exposure	SH
Fixed tissue	T1, A, SH, I

Key

SH - Cleansing with 0.5-1.3% sodium hypochorite.
A - Autoclaving 121°C for 1 hour. Note that many autoclaves do not perform according to specification. Washing and other handling should follow autoclaving of material.
I - Incineration
D - Disposal, should not be reused
HW - Thorough hand-washing with usual hospital soaps and detergents. Avoid abrasion of hands by vigorous brushing.
N1 - Probably not infectious
* - Probably not infectious
ND - Not suitable for blood product or organ donation
T1 - Treat as infectious

(continued from page 1)

Shigella flexneri dysentery. Although the patient recovered, the strain isolated from her faeces was resistant to all antibiotics which are know to be clinically effective for the treatment of severe shigella dysentery. Multi-resistant strains of shigella and salmonella are prevalent in India, S.E. Asia, Middle East, Africa and South America. For uncomplicated salmonella gastroenteritis and for mild cases of shigella dysentery antibiotics are unnecessary, but antibiotic treatment is important, and may be life-saving, in cases of severe shigella dysentery and for systemic salmonella infections.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24/11/83 - 7/12/83 BULLETIN NUMBER . 83/25

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	3	6		5	3	8		28
0101 ADENOVIRUS TYPE 1	1			2		4		2	9
0102 ADENOVIRUS TYPE 2	3	1				2		4	10
0103 ADENOVIRUS TYPE 3				4		1			5
0104 ADENOVIRUS TYPE 4						2			2
0105 ADENOVIRUS TYPE 5	2					1			3
0106 ADENOVIRUS TYPE 6								1	1
0107 ADENOVIRUS TYPE 7		6							6
0115 ADENOVIRUS TYPE 15	1								1
0119 ADENOVIRUS TYPE 19	1							2	3
0199 ADENOVIRUS TYPING PENDING		2	2		3	2			9
0201 INFLUENZA A VIRUS	2		3				1	3	9
0301 PARAINFLUENZA VIRUS TYPE 1	1			2	2	1			6
0303 PARAINFLUENZA VIRUS TYPE 3	13	1	2	3	21	21	6		67
0399 PARAINFLUENZA VIRUS TYPING PENDING.					1	1			2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)			6			2		10	18
0500 RHINOVIRUS (ALL TYPES)	4		1	11	16	20	3		55
0600 MYCOPLASMA PNEUMONIAE	18		9		21	82	17	8	155
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED								2	2
0816 COXSACKIEVIRUS A16							1		1
0901 COXSACKIEVIRUS B1		1							1
0902 COXSACKIEVIRUS B2		1		1	6	6	1	1	16
0903 COXSACKIEVIRUS B3								2	2
0905 COXSACKIEVIRUS B5			1						1
0999 COXSACKIEVIRUS GROUP B TYPING PENDING							1		1
1000 ECHOVIRUS NOT TYPED					2				2
1003 ECHOVIRUS TYPE 3								1	1
1005 ECHOVIRUS TYPE 5				1					1
1006 ECHOVIRUS TYPE 6	1								1
1007 ECHOVIRUS TYPE 7								1	1
1009 ECHOVIRUS TYPE 9	5			1		1	3		10
1011 ECHOVIRUS TYPE 11	2								2
1014 ECHOVIRUS TYPE 14							1		1
1016 ECHOVIRUS TYPE 16	2								2
1017 ECHOVIRUS TYPE 17	2	1							3
1022 ECHOVIRUS TYPE 22		3					1		4
1101 POLIOVIRUS TYPE 1						2		2	4
1102 POLIOVIRUS TYPE 2							1		1
1103 POLIOVIRUS TYPE 3								1	1
1104 POLIOVIRUS-VACCINAL STRAIN			2			2			4
1200 MUMPS VIRUS	3		1	2	1		2		9
1300 HERPES VIRUS GROUP-NOT TYPED	27			2		2		2	33
1301 HERPES SIMPLEX VIRUS NOT-TYPED		4		1				1	6
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	9				3			9	21
1303 VARICELLA-ZOSTER VIRUS	4	1	2	2		4	1		14
1306 HERPES SIMPLEX TYPE 1	31			41	1	21	25	15	134
1307 HERPES SIMPLEX TYPE 2	136		1	55	1	24	48	53	318
1399 HERPES VIRUS TYPING PENDING			20		3	1			24
1401 COXIELLA BURNETI	2						4		6
1502 PICORNA VIRUS-NOT TYPED			3				1	1	5
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS		1		6	2	4			13
1522 RUBELLA VIRUS	1		2	2		6	20	2	33
1532 HEPATITIS B ANTIGEN	20		4	40	1	11	11	3	90
1535 HEPATITIS A ANTIBODY			2	1		7	1	8	19
1541 CHLAMYDIA A - C TRACHOMATIS	53		9				19	72	153
1556 CMV - CYTOMEGALOVIRUS	6			23	6	3	9	7	54
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	16	6	10		7	8		1	48
1599 ENTEROVIRUS TYPING PENDING		4	5		10		1		20
ROSS RIVER VIRUS						1	8	6	15
ASTROVIRUS		1							1
SMALL VIRUS (LIKE) PARTICLE	1			1					2
DENGUE							1		1
PARAMYXOVIRUS						4			4
ARBO. GROUP B.							1		1
Total	370	36	91	201	112	249	196	223	1,478

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24/11/83 to 7/12/83 .... 83/25

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0100 ADENOVIRUS NOT TYPED.....		1					3				
0101 ADENOVIRUS TYPE 1.....	2	3									
0102 ADENOVIRUS TYPE 2.....	2	4		1			1				
0103 ADENOVIRUS TYPE 3.....	1	2									1
0104 ADENOVIRUS TYPE 4.....		2									
0105 ADENOVIRUS TYPE 5.....				1							
0106 ADENOVIRUS TYPE 6.....		1									
0107 ADENOVIRUS TYPE 7.....		5					4				
0201 INFLUENZA A VIRUS.....	2	4							1		
0301 PARAINFLUENZA VIRUS TYPE 1....		5				1					1
0303 PARAINFLUENZA VIRUS TYPE 3....	3	62		2							1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	3	12							1		
0500 RHINOVIRUS (ALL TYPES).....	3	48	1	1							1
0600 MYCOPLASMA PNEUMONIAE.....	7	138		1			1		1		4
0816 COXSACKIEVIRUS A16.....											1
0901 COXSACKIEVIRUS B1.....		1									
0902 COXSACKIEVIRUS B2.....	1	7		2			8		1		1
0903 COXSACKIEVIRUS B3.....				2							
0905 COXSACKIEVIRUS B5.....				1							
1005 ECHOVIRUS TYPE 5.....											1
1006 ECHOVIRUS TYPE 6.....	1										
1007 ECHOVIRUS TYPE 7.....							1				
1009 ECHOVIRUS TYPE 9.....	1	1		7		1					
1011 ECHOVIRUS TYPE 11.....		2									
1014 ECHOVIRUS TYPE 14.....		1									
1017 ECHOVIRUS TYPE 17.....	1			1			1				
1022 ECHOVIRUS TYPE 22.....	1	2					1				
1101 POLIOVIRUS TYPE 1.....		2									
1102 POLIOVIRUS TYPE 2.....							1				
1103 POLIOVIRUS TYPE 3.....	1										
1104 POLIOVIRUS-VACCINAL STRAIN....							3				
1200 MUMPS VIRUS.....	1		1	2			1				
1300 HERPES VIRUS GROUP-NOT TYPED..											1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	1	1				1				2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	4	3	1	1	1		1	1			
1303 VARICELLA-ZOSTER VIRUS.....	1	4	1			1	1				8
1306 HERPES SIMPLEX TYPE 1.....	5	2		3						1	61
1307 HERPES SIMPLEX TYPE 2.....	7										53
1401 COXIELLA BURNETI.....	2	1									
1502 PICORNA VIRUS-NOT TYPED.....							1				
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....		4				1					11
1522 RUBELLA VIRUS.....	3										26
1532 HEPATITIS B ANTIGEN.....	43							39			
1535 HEPATITIS A ANTIBODY.....	2							17			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1						2				
1556 CMV - CYTOMEGALOVIRUS.....	7	11		2				1		4	
1563 CORONAVIRUS.....		1									
1564 ROTAVIRUS.....	1	2					47				
9992 ROSS RIVER VIRUS.....	3										7
9993 ASTROVIRUS.....							1				
9994 SMALL VIRUS (LIKE) PARTICLE...	1						1				
9996 PARAMYXOVIRUS.....	1	3									
Total.....	112	335	5	27	1	4	80	58	4	5	181

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24/11/83 to 7/12/83 ... 83/25

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						2			
0102 ADENOVIRUS TYPE 2.....								1		1
0103 ADENOVIRUS TYPE 3.....	2							1		
0105 ADENOVIRUS TYPE 5.....						1		1		
0115 ADENOVIRUS TYPE 15.....	1									
0119 ADENOVIRUS TYPE 19.....	1	2								
0201 INFLUENZA A VIRUS.....					1				1	
0303 PARAINFLUENZA VIRUS TYPE 3....									1	1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1	1	
0500 RHINOVIRUS (ALL TYPES).....		1						1		
0600 MYCOPLASMA PNEUMONIAE.....	1		1				1	7	2	
1003 ECHOVIRUS TYPE 3.....					1					
1007 ECHOVIRUS TYPE 7.....								1		
1009 ECHOVIRUS TYPE 9.....							1			
1016 ECHOVIRUS TYPE 16.....							1	1		
1101 POLIOVIRUS TYPE 1.....						1				1
1104 POLIOVIRUS-VACCINAL STRAIN....							1			
1200 MUMPS VIRUS.....			5					1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			9	1				1	1	
1306 HERPES SIMPLEX TYPE 1.....	6	55			1			3		
1307 HERPES SIMPLEX TYPE 2.....		258								
1401 COXIELLA BURNETI.....						1		2		
1521 MEASLES VIRUS.....								1		
1522 RUBELLA VIRUS.....			1		5		1		1	
1532 HEPATITIS B ANTIGEN.....	1				1				6	
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	147								
1556 CMV - CYTOMEGALOVIRUS.....		8		1		5	1	8	11	2
9992 ROSS RIVER VIRUS.....					10			1		
9995 DENGUE.....					1			1		
9998 ARBO. GROUP B.					1					
Total.....	16	473	16	2	21	8	8	32	24	5

CDI 83/25

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

(Weeks 37-40)
(10 September to 7 October 1983)

Bulletin 83/25

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Amoebiasis			2	1			1	1	5	39
Ankylostomiasis			2	8					10	65
Anthrax									—	—
Arbovirus infection									—	4
Brucellosis									—	13
Campylobacter infections	25	N.N.	N.N.	93	N.N.	N.N.	N.N.	N.N.	118	1141
Chancroid				N.N.	2	N.N.	1		3	12
Cholera									—	4
Congenital rubella syndrome		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—
Diphtheria									—	—
Donovanosis		N.N.	2	N.N.	4	N.N.			6	78
Giardiasis	17	N.N.	N.N.	61	N.N.	N.N.	N.N.	1	79	744
Genital herpes	90	N.N.	6	22	N.N.	N.N.		N.N.	118	1541
Gonococcal ophthalmia neonatorum		N.N.			N.N.	N.N.	3	N.N.	3	13
Gonorrhoea	131	107	60	56	99	4	61	6	524	8665
Hepatitis A (infectious)	8	18	8	21	9	1	16	2	83	801
Hepatitis B (serum)	29	26	7	8	2				72	719
Hepatitis - unspecified	5	1			9	N.N.	5	2	22	259
Hydatid disease			1						1	7
Lassa Fever			N.N.			N.N.	N.N.	N.N.	—	—
Legionnaires disease		1	N.N.		N.N.	N.N.	N.N.	N.N.	1	17
Leprosy			3		3				6	45
Leptospirosis		6	5	3					14	119
Lymphogranuloma venereum	2	N.N.	N.N.	N.N.	N.N.	N.N.			2	7
Malaria	10	4	24	5		1	1	1	46	468
Marburg Disease			N.N.			N.N.	N.N.	N.N.	—	—
Meningococcal infections	3		4			N.N.			7	84
Non-specific urethritis	268	N.N.	N.N.	97	N.N.	N.N.	N.N.	N.N.	365	3994
Ornithosis				1					1	16
Pertussis (whooping cough)	17	7	N.N.	2	N.N.	N.N.	N.N.	N.N.	26	242
Plague									—	—
Poliomyelitis									—	—
Q. fever	1	2	9	1	N.N.		N.N.		13	138
Rabies		N.N.	N.N.			N.N.	N.N.	N.N.	—	—

DISEASE	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Salmonella infections	116	13	17	21	4	5	9	1	186	2520
Shigella infections	8	5	3	2	4		15		37	487
Smallpox									—	—
Syphilis	44	5	17	10	46	2	47	1	172	2040
Tetanus									—	7
Trachoma		N.N.	1		N.N.	N.N.			1	4
Tuberculosis (all forms)		20	13	9	14		2	2	60	921
Typhoid fever									—	18
Typhus (all forms)			4						4	19
Vibrio parahaemolyticus infections		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—
Yellow Fever									—	—
Yersinia enterocolitica infections		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—

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

N.N. Not Notifiable

ADJUSTMENTS

Campylobacter Infections	-	2 SA
Giardiasis	-	4 SA
Gonorrhoea	-	1 NT
Hepatitis A	-	2 SA
	+	1 SA
Leptospirosis	+	1 SA
Malaria	-	5 SA
Pertussis	+	2 SA
Salmonella infections	-	1 Vic
	-	2 SA
	-	1 Qld
Shigella infections	+	3 SA
Syphilis	+	6 Vic
	+	1 NT
Tuberculosis	+	38 NSW

Entries indicate Issues : Page number.

Underlined entries refer to longer articles.

(c) refers to corrigenda of previous article(s).