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Contents:

VIRUS REPORTING SCHEME - A total of 1053 reports were processed this period. A further ten isolates of echovirus type 7 (including one dual isolation with coxsackievirus B5 from faeces of a one year old boy with gastroenteritis) were reported by the State Health Laboratory, Brisbane. Six of the isolates were from young children. The summer peak of rubella infections appears to have decreased, with 15 reports compared with 65, 93 (two periods) and 97 for the previous four periods.

- Arbovirus infections - The 12 epidemic polyarthritis reports originated from Queensland (7: Townsville (3), Rockhampton (3), Miles (1)); Western Australia (2: Onslow (1); metropolitan Perth (1)); Northern Territory (2: Darwin (1), Katherine (1)); and New South Wales (Tamworth (1)). The Brisbane laboratory also reported specific IgM against Kunjin virus in a 56 year old male from Trinity Beach, and cross-reacting IgM against Kokobera and Alfuy viruses in a 40 year old female from Townsville. No further arbovirus seroconversions have been detected in the Victorian sentinel chicken program to 21 January. Of the alphavirus reactions, three transient HI responses in the Robinvale flock, and six seroconversions at Mildura and one at Echuca have been detected (corrigendum of CDI 85/2). Serum from the chicken at Rutherglen which exhibited an antibody response of 1/320 by HI against MVE and Kunjin has been shown to be negative for MVE antibody by ELISA, and negative for both antibodies by plaque inhibition and plaque reduction tests. Edge Hill virus is the probable infecting agent (c.f. CDI 85/2).

- A non-menstrual case of Toxic Shock Syndrome was reported recently in a 40 year old female who was readmitted to a Perth hospital with typical signs three days after a breast biopsy. Staphylococcus aureus was recovered from the wound, but not from any other sites.

References from page 6

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HUMAN SALMONELLOSIS SURVEILLANCE

(Contributed by J. Taplin and L.K. Scott, Microbiological Diagnostic Unit (MDU), University of Melbourne).

A total of 726 salmonella (69 serotypes), 172 shigella and 329 campylobacter reports from human cases in Australia were collated during July-September 1984. This compares with the total of 1459 reports for the same period in 1983, of which 168 were shigella and 264 were campylobacter species.

TYPHOID - S. typhi phage type 25 was isolated from blood and faeces of an 11 month old girl in New South Wales. The carrier was found to be her 59 year old grandmother who lived with the family, and who had come from Kampuchea some years previously. A degraded strain was isolated from the grandmother on follow-up. S. typhi degraded was also cultured from faeces of a 32 year old male who had been ill for two weeks. He had no history of overseas travel. In Queensland, S. typhi A was grown from blood cultures of a man with a history of fever, headache and green mucoid stools.

Four S. typhi isolations were from refugees. S. typhi D1-N was grown from faeces of a 69 year old female Vietnamese refugee on routine screening; S. typhi untypable was isolated from faeces of a 30 year old female Timorese refugee from Indonesia who had arrived in Darwin two weeks previously; and S. typhi O was isolated from blood, and later faeces, of a 48 year old male refugee from Iran. S typhi B2 was also cultured from blood and faeces of his six year old son, but from no other family member. The family had come to Australia via India.

PARATYPHOID - A sudden increase of S. paratyphi B serotype java phage type 1 variant 6 reports was recorded during the quarter in Victoria (26), New South Wales (12), Queensland (2) and South Australia (1) (see CDI 84/23). The outbreak started in Victoria in early July, and continued in August and September. Isolations in New South Wales began at the end of August and continued in September. The South Australian case occurred in August. No cause was found for the sudden spate of cases. Previously, this phage type had only been seen in Queensland (9 isolations) and once in South Australia in 1981.

S. paratyphi B Dundee was isolated from faeces of a 25 year old female in Queensland, and from faeces of a six year old boy in Victoria with a one week history of diarrhoea. The child had had diarrhoea while in Lebanon five weeks earlier. S. paratyphi B Taunton was isolated from faeces of a 56 year old female, from whom the same strain was isolated from a leg abscess in March.

FAMILY OUTBREAKS - Family outbreaks affecting at least two members of a family (mainly young children) involved; S. havana, S. java 1 var 6, S. typhimurium phage types 6, 55 and 108, S.4, 12:D:-:, and S.flexneri in New South Wales; S. adelaide, S. java 1 var 6 and S. typhimiurium phage types 9 and 141 in Victoria; C. jejuni and S. typhimiurium phage types 5 and 58 in Western Australia; S. bovis-morbificans and S. eastbourne in Northern Territory; S. kottbus and S. typhimurium phage type 8 in South Australia; and S. virchow in Queensland.

SEROTYPE FLUCTUATIONS - A steep rise in isolations of S. havana was evident in the Northern Territory compared with the seven reports in the previous quarter. The 30 reports included an outbreak on a settlement near Alice Springs involving eight

people (four in one family); three were in children aged less than five years, and the remainder were in adults. There were eight other patients from surrounding areas of Alice Springs (six in children ≤ five years). Two cases were in Darwin. Since 1980, 27% of all isolates of S. havana have come from the Northern Territory. A similar rise was noted for S. orion in Western Australia (13 isolates). Nine cases were in children ≤ 3 years; ten patients were from metropolitan Perth and two from Fremantle. S. typhimurium phage type 135 is still the most common phage type (33 reports), and was isolated in all States except the Australian Capital Territory. Eleven isolates came from South Australia, mainly from Adelaide suburbs, with six cases in children ≤ three years. Five of the nine Victorian isolates were also in children ≤ three years. The first report of S. typhimurium phage type 25 in 1984 was recorded in a 2 1/2 year old girl from Sydney. This phage type was responsible for a large food poisoning outbreak in New South Wales in 1983, when over 100 cases were identified (see CDI (1983) 83/21: 3).

URINE ISOLATIONS - Isolations from urine comprised serotypes S. bareilly, S. oranienburg, S. singapore and S. typhimurium phage types 6, 16, 135 and untypable.

BLOOD ISOLATIONS - Cases of septicaemia involved salmonella serotypes S. birkenhead, S. bovis-morbificans, S. bredeney, S. cholerae-suis var. Kunzendorf (fatal case in a 78 year old female), S. derby, S. heidelberg, S. java 1 var 6 (from a five day old boy whose mother had diarrhoea due to the serotype three days prior to birth), S. muenchen, S. typhi A, 25 and untypable, and S. typhimurium phage types 6 and 135. S. boydii 14 was isolated from blood and faeces of an eight month girl dehydrated after an attack of gastroenteritis contracted in Lebanon. The culture was aerogenic and failed to ferment mannitol. S. dysenteriae 1 was cultured from a 23 year old female with a several day history of diarrhoea and vomiting after visiting India. The organism was resistant to chloramphenicol and sulphonamides. A Campylobacter species was isolated in South Australia from a 50 year old female with leukaemia.

Other isolations of interest include S. adelaide from bone; S. agona from pus drained from a subphrenic abscess; S. enteritidis from gall bladder; and S. birkenhead from a rectal abscess which also grew Klebsiella and Citrobacter species. A laboratory-acquired infection of S. singapore was also recorded in a 26 year old female.

New reports for the National Salmonella Surveillance Scheme were S. java 1 var 3 (Victoria), S. typhi 25 (New South Wales), S. typhi D1-N (Victoria) and S. dysenteriae 4 (New South Wales). Organisms isolated for the first time in various States were S. heidelberg in Tasmania from a woman who had returned from Hong Kong and Japan with a four week history of diarrhoea, arthritis and conjunctivitis; S. treforest from an 18 month old boy in Queensland; and S. uganda from a six year old refugee girl from El Salvador in Victoria.

PHYTOPHOTODERMATITIS AMONG GROCERY WORKERS - USA
(Based on MMWR (1985) 34:11)

On 5 July 1984, a 33 year old woman presented to an Ohio medical clinic with a bullous, erythematous, nonpruritic, discrete rash of the left forearm of six days' duration. An occupational history indicated that she was a cashier at a supermarket. Several co-workers were reported to have had similar rashes that were attributed to handling celery.

Investigation revealed that 14 (27%) of the 52 current full- and part-time employees interviewed had papular, well-circumscribed rashes confined to the upper extremities, with residual blistering or hyperpigmentation. Dates of rash onset ranged from April through August, with a peak in July. All cases occurred among cashiers, baggers and produce clerks. None occurred among shelf stockers, delicatessen and meat personnel or managers. Cases were significantly more likely than noncases to have had contact with fresh vegetables (100% compared with 39%; p=0.009) and with fresh flowers (92% compared with 29%; p=0.009). Also, cases were significantly more likely than noncases to have used a tanning salon during the outbreak period (36% compared with 5%; p=0.01). On the basis of history and physical examination, a diagnosis of phytophotodermatitis was made, and employees handling produce were recommended to wash exposed areas of hands, wrists, and forearms regularly, and avoid either tanning salons or excessive exposure to sunlight. No new cases occurred after October, which is typical for the seasonal pattern of this disease.

Skin disorders appear to represent a widespread but largely unrecognised problem among supermarket employees. Many of these rashes among these workers appear to be phytophotodermatitis, a well-circumscribed rash evoked by contact with linear furanocoumarins (psoralens), followed by exposure of the skin to long-wave ultraviolet light (350 nm). It is associated with exposure to a wide variety of fruits, flowers, and vegetables including celery, dill, parsley, oil of Bergamot, and chrysanthemums. Exposure to sunlight is sufficient to provoke phytophotodermatitis following contact with psoralens. However, the use of artificial ultraviolet light in tanning salons appears in the present instance to have enhanced this effect.

In phytophotodermatitis, the reaction is typically confined to the initial site of contact and is characterised by redness and blistering in the absence of itching and by residual hyperpigmentation. (1) This type of reaction differs from an allergic contact dermatitis in that it requires exposure to ultraviolet light and does not require a period of sensitisation. In addition an allergic dermatitis is usually pruritic.

This outbreak resembles a series of episodes investigated in supermarkets throughout the midwest in 1980-81. In these episodes, baggers had the highest attack rates of dermatitis (51%). Frequent contact with unpackaged celery and exposure to sunlight during the work-shift were significantly associated with cases. (2) Also, an investigation of agricultural field workers in Michigan in 1961 found that celery infected with pink rot (Sclerotinia sclerotiorum) was associated with an outbreak of photodermatitis. (3)

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HAEMORRHAGIC SHOCK ENCEPHALOPATHY SYNDROME (HSES) SURVEILLANCE - UNITED KINGDOM

(Based on CDR (1985) 85/1:3)

In July 1983 a "new" syndrome was reported in ten babies presenting to the Hospital for Sick Children, London, between March 1982 and March 1983, seven of whom died [1]. The illness, called Haemorrhagic Shock Encephalopathy Syndrome (HSES), consisted of acute onset of encephalopathy, fever, shock, watery diarrhoea, severe disseminated intravascular coagulation (DIC), renal and hepatic dysfunction. There were no consistent microbiological findings, but low plasma levels of the acute phase reactant a-1 antitrypsin together with high levels of immunoreactive trypsin were found in the affected children.

The aetiology of HSES is unknown but various hypotheses have been put forward. These include: heatstroke due to overwrapping [2,3,4]; exposure to toxic agents [1]; an as yet unidentified viral infection [1]; disturbance of normal bacterial flora due to a viral infection, predisposing to overgrowth of toxin-producing bacteria [5].

A national surveillance scheme [6] has recorded 44 cases during the period 1 January 1982 - 31 December 1984; 13 occurred in 1982, 22 in 1983 and nine in 1984. Of the 39 cases which had sufficient details for analysis, 15 were in females and 24 (62%) in males. Although the age distribution ranged from 17 days to 15 years, the modal age was three months and the median five months, with 34 (87%) cases under one year of age. Cases were fairly evenly distributed over the year. Of the 15 patients for whom ethnic information was provided, 11 were white caucasians, two were black and two of mixed race. Outcome information was provided for 33 patients; 22 died. Mortality among females was 10/12 (83%) and for males was 12/21 (37%). Nine of the 11 survivors were severely neurologically damaged, the nature of any sequelae was unknown in the other two.

Clinical features for the 38 patients for whom the information was provided are shown in Table 1; 22 satisfied all 11 diagnostic criteria.

TABLE 1. Presence of HSES case criteria (N=38)

Feature	Yes	No	Not reported/ investigated
1. Encephalopathy	38	-	-
2. Shock	35	1	2
3. DIC	34	4(1)	-
4. Diarrhoea	34	3	1
5. Falling haemoglobin	30	3	5
6. Falling platelets	31	2	5
7. Acidosis	35	1	2
8. Raised transaminases	36	-	2
9. Renal function impairment	32	5	1
10. Normal NH_3	27	6	5
11. Negative blood and CSF cultures	33	1(2)	4

(1) - one had "hepatic type" coagulopathy.
(2) - one had an enterovirus in the CSF.

Four of 30 patients for whom details of past history and prodromal illness was available had a previous history of neuro-developmental abnormalities. Twenty-two had a variety of

prodromal illness ranging in duration from a few hours to several days, but eight had been completely well before being found collapsed. The admission temperature was reported in 19 patients and was > 39°C in ten of these; one was afebrile and one hypothermic.

In addition to the characteristic post mortem findings described in the first HSES report[1], and which were also observed in the other patients, some centres reported hepatic necrosis (3 cases); adrenal haemorrhage (2); villous atrophy and crypt hyperplasia (1); pulmonary petechial haemorrhages (1). Reye's Syndrome appeared as the certified cause of death in two cases.

No positive findings were found in the special virological and toxicological investigations. In the risk-factor study in which the parents of ten HSES patients were interviewed, a family history of neurological abnormality or unexpected deaths in infancy in half of the cases were recorded. All seven babies who were "typical" HSES with idential histories were clothed identically in "babygros" (four with vests), and all except one were covered by man-made fibre quilts, the other was under two doubled blankets and a sheet. Five were described as "hot" to the touch and two as "cold" when found; all were soaking wet. Of the other three interviewed, two had severe neurodevelopmental abnormalities and developed HSES in hospital and one was a child of seven years.

The HSES reporting scheme has shown that this condition is being recognised in many parts of the UK, a conclusion supported by published observations [7,8]. Just under half of the cases (excluding those in the original report [1]) were, however, reported by the hospital from which HSES was first described, presumably reflecting both the higher diagnostic awareness and readiness to report of a centre with a particular interest in the condition. Conclusions about the epidemiology of HSES based on the reported cases should therefore be viewed with caution because of the selected nature of the sample. Several observations can nevertheless be made: the cases reported from other centres were similar to those in the original study in respect of age and clinical features, although some patients had a raised ammonia and some had normal renal function. The latter could be a function of severity of shock but the former illustrates the "grey area" in the differential diagnosis of HSES from Reye's Syndrome. In one patient, HSES was considered to be the cause of death even though there was some fatty change in the liver. There is a need for a more detailed and systematic post mortem case definition as findings are usually reported in a non-standardised fashion. Histology of the small bowel for example, which is said to undergo characterstic changes in hyperthermia, [9] is rarely undertaken.

No clear aetiological factors have emerged to date either from the laboratory or pilot risk factor studies. There was no evidence of gross overwrapping; there was however a certain consistency in the way in which the seven "typical" cases were clothed and wrapped which suggests that there is a need to examine the possible role of more subtle factors affecting heat loss. The significance of these, together with such factors as past medical and family histories can only be assessed by controlled studies.

Editorial Comment

The CDI would be interested in receiving information on any Australian cases of this newly recognised illness.

References continued on page 1

HUMAN SALMONELLOSIS CASES

Period: July - September 1984

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S. adelaide	11	1		2	1		5		2
S. agona	13	1	2	3	2		3		2
S. albany	1						1		
S. anatum	20		1	4	8		6		1
S. arizonae	4		1		1		1		1
S. barenfeld	1						1		
S. bareilly	2			1			1		
S. birkenhead	12		3		6	3			
S. blockley	2			1		1			
S. bovis-morbificans	31		13	1	1	6	5	1	4
S. braenderup	2		1		1				
S. bredeney	4				4				
S. breukelen	1				1				
S. chester	19		1	2	5	4	3		4
S. cholerae-suis Kunzendorf	1		1						
S. derby	12		4	3		2	3		
S. eastbourne	5			1			1		3
S. emek	2					1	1		
S. enteritidis	7		2	2	2				1
S. give	2		2						
S. havana	45		2		4	3	6		30
S. heidelberg	8		2		3		2	1	
S. houten	1				1				
S. hvittingfoss	7			1	2	1	2		1
S. indiana	1						1		
S. infantis	16		1	2	2	2	3		6
S. java	5		3				1		1
S. java 1 var 3	1			1					
S. java 1 var 6	42		12	26	3	1			
S. java Dundee	1				1				
S. java untypable	1								1
S. kentucky	1			1					
S. kinondoni	1								1
S. kottbus	4					4			
S. lansing	5				5				
S. lexington	3		1	1	1				
S. litchfield	2				1				1
S. london	1		1						
S. mbandaka	3		2	1					
S. meleagridis	2				1		1		
S. mississippi	1							1	
S. montevideo	1			1					
S. muenchen	18				5		6		7
S. muenster	1					1			
S. newington	6				5				1
S. newport	9	2	2	2	3				
S. onderstepoort	1								1
S. oranienburg	11			2	5		3		1
S. orion	18				1		13		4
S. oslo	1		1						
S. panama	5			2			3		
S. paratyphi B Dundee	3			2	1				
S. paratyphi B Taunton	1		1						
S. poona	2				2				
S. potsdam	2				1		1		
S. rubislaw	4						1		3
S. saintpaul	29		2	1	13		8	1	4
S. senftenberg	10		2				4		4

HUMAN SALMONELLOSIS CASES

Period: July - September 1984

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S. singapore	11		5	3	1	2			
S. sofia	2		1			1			
S. stanley	5					4	1		
S. tennessee	9		2	2			5		
S. thompson	2		1			1			
S. treforest	2				2				
S. typhi*	13		3	3	7				
S. typhimurium*	210		55	50	31	34	30	5	5
S. uganda	1			1					
S. untypable 16:LV:-	1								1
S. untypable 4,12:-:-	1						1		
S. untypable 9,12:-:1,5	1			1					
S. untypable rough:K:ENX	1			1					
S. untypable rough:LV:1,7	1				1				
S. urbana	3						3		
S. virchow	20	2		4	14				
S. wandsbek	2					1			1
S. wandsworth	2						2		
S. waycross	1				1				
S. welikade	3				3				
S. weltevreden	8		1	1			1		5
S. 4,12:D:-	2		2						
TOTAL	726	6	133	129	152	72	129	9	96
S. typhi*									
S. typhi 25	2		2						
S. typhi A	1				1				
S. typhi B2	2				2				
S. typhi D1-N	3			3					
S. typhi degraded	1		1						
S. typhi O	2				2				
S. typhi untypable	2				2				
TOTAL	23		3	3	7				
S. typhimurium*									
S. typhimurium	6		1				5		
S. typhimurium UDNC	3		1	1			1		
S. typhimurium untypable	10		1	1	5	1	2		
phage type 2	1		1						
phage type 3	2				2				
phage type 4	3		2	1					
phage type 5	9					2	6		1
phage type 6	8		2	6					
phage type 8	8			1		6			1
phage type 9	5		1	1	1		2		
phage type 10	1			1					
phage type 12	4		2	2					
phage type 12A	13		1	1	3	8			
phage type 16	2		1	1					
phage type 22	2						2		
phage type 24	2				2				

HUMAN SALMONELLOSIS CASES

Period: July - September 1984

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
phage type 25	1		1						
phage type 26	7			5			2		
phage type 31	1			1					
phage type 41	2		1		1				
phage type 44	5		4	1					
phage type 49	3			3					
phage type 55	4		3			1			
phage type 58	2						2		
phage type 64	3			1			2		
phage type 68	1								1
phage type 72	1			1					
phage type 90	1			1					
phage type 101	6		1		1	1		3	
phage type 102	1		1						
phage type 104	1		1						
phage type 108	8		7	1					
phage type 122	1				1				
phage type 124	4		4						
phage type 135	33		5	9	5	11	1	1	1
phage type 141	20		5	7	3		4	1	
phage type 145	2		2						
phage type 170	14		5	3	5	1			
phage type 179	3		2	1					
phage type 185	2					2			
phage type 202	5				2	1	1		1
TOTAL	210		55	50	31	34	30	5	5
Shigellae									
S. boydii 4	2		1	1					
S. boydii 12	1			1					
S. boydii 14	2			2					
S. dysenteriae 1	2		1				1		
S. dysenteriae 3	1		1						
S. dysenteriae 4	2		2						
S. flexneri 1B	2			2					
S. flexneri 2	31				1		30		
S. flexneri 2A	18		1		1			1	15
S. flexneri 2B	2			2					
S. flexneri 4	5		4		1				
S. flexneri 4A	1			1					
S. flexneri 6	51		6	2			31		12
S. flexneri Y	1				1				
S. sonnei	26	1	12		1		12		
S. sonnei biotype A	21			5	1	5		1	9
S. sonnei biotype F	1			1					
S. sonnei biotype G	3		1	2					
TOTAL	172	1	29	19	6	5	74	2	36

HUMAN SALMONELLOSIS CASES

Period: July - September 1984

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
Campylobacter									
C. jejuni	283	1	58	87	27	22	78		10
C. species	46	1	38	1		5			1
TOTAL	329	2	96	88	27	27	78		11
Escherichia coli									
E. coli O112 K66 B11	1			1					
E. coli O114 K90 B	1			1					
E. coli O125 K70 B15	2			2					
E. coli O126 K71 B16	1			1					
E. coli O128 K67 B12	4	1	1	2					
E. coli O55 K59 B5	1	1							
E. coli O86 K61 B7	1			1					
TOTAL	11	2	1	8					

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 17/1/85 - 30/1/85 BULLETIN NUMBER 85/3

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	4		2		1		11		18
0101 ADENOVIRUS TYPE 1				1		3		1	5
0102 ADENOVIRUS TYPE 2				1	2	12			15
0103 ADENOVIRUS TYPE 3				1	1	1		2	5
0105 ADENOVIRUS TYPE 5					1	1		1	3
0106 ADENOVIRUS TYPE 6					3				3
0107 ADENOVIRUS TYPE 7		1				1			2
0137 ADENOVIRUS TYPE 37								2	2
0199 ADENOVIRUS TYPING PENDING		2	1		3	3			9
0201 INFLUENZA A VIRUS	1		1	1		1	2		6
0203 INFLUENZA B VIRUS							2		2
0301 PARAINFLUENZA VIRUS TYPE 1					1	2	3		6
0302 PARAINFLUENZA VIRUS TYPE 2						1	5	1	7
0303 PARAINFLUENZA VIRUS TYPE 3	1			5	11	1	3	1	22
0399 PARAINFLUENZA VIRUS TYPING PENDING						1			1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	2		1		1	1	2	3	10
0500 RHINOVIRUS (ALL TYPES)				3	4	4	6	1	18
0600 MYCOPLASMA PNEUMONIAE	1		1	1			1	3	7
0700 ORNITHOSIS-PSITTACOSIS				1					1
0809 COXSACKIEVIRUS A9					1	1	5		7
0816 COXSACKIEVIRUS A16	2								2
0901 COXSACKIEVIRUS B1		1							1
0905 COXSACKIEVIRUS B5		1					2	2	5
1000 ECHOVIRUS NOT TYPED							6		6
1003 ECHOVIRUS TYPE 3	1								1
1007 ECHOVIRUS TYPE 7							10	1	11
1009 ECHOVIRUS TYPE 9				1					1
1011 ECHOVIRUS TYPE 11					5			1	6
1020 ECHOVIRUS TYPE 20						1			1
1021 ECHOVIRUS TYPE 21								1	1
1022 ECHOVIRUS TYPE 22		1				1	1		3
1024 ECHOVIRUS TYPE 24	1								1
1030 ECHOVIRUS TYPE 30				2					2
1031 ECHOVIRUS TYPE 31				1					1
1099 ECHOVIRUS TYPING PENDING							6		6
1100 POLIOVIRUS NOT TYPED			1				2		3
1101 POLIOVIRUS TYPE 1								1	1
1200 MUMPS VIRUS	3			7	2		1		13
1300 HERPES VIRUS GROUP-NOT TYPED	17			3		1		4	25
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	6		3				1	11
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5			6		2		4	17
1303 VARICELLA-ZOSTER VIRUS	4		1				2	1	8
1306 HERPES SIMPLEX TYPE 1	19			35		19	14	7	94
1307 HERPES SIMPLEX TYPE 2	82			54		21	31	39	227
1399 HERPES VIRUS TYPING PENDING					3	2			5
1401 COXIELLA BURNETI	1			3			8	1	13
1502 PICORNA VIRUS-NOT TYPED	3		6				1		10
1521 MEASLES VIRUS	6	3						1	10
1522 RUBELLA VIRUS	1			1		2	5	6	15
1532 HEPATITIS B ANTIGEN	56		7	21		10	3	9	106
1535 HEPATITIS A ANTIBODY	6			3		2	1	2	14
1541 CHLAMYDIA A - C TRACHOMATIS	94	1		23 *			37	39	194
1556 CMV - CYTOMEGALOVIRUS	7	3		2	6	5	4	6	33
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS			7	1	6	10		1	25
1571 ENTEROVIRUS TYPE 71 (BRCR)				1					1
1599 ENTEROVIRUS TYPING PENDING			8		5	1			14
9992 ROSS RIVER VIRUS							9	3	12
9994 SMALL VIRUS (LIKE) PARTICLE		1							1
9996 PARAMYXOVIRUS						1			1
9997 KUNJIN VIRUS							1		1
9998 ARBO. GROUP B. ...							1		1
Total	319	20	36	182	56	111	184	145	1,053

* Cultures performed at the Microbiological Diagnostic Unit, Melbourne

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 17/1/85 to 30/1/85 .... 85/3

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0101 ADENOVIRUS TYPE 1.....		4									
0102 ADENOVIRUS TYPE 2.....	1	11					2				
0103 ADENOVIRUS TYPE 3.....		3									
0105 ADENOVIRUS TYPE 5.....		2		1							
0106 ADENOVIRUS TYPE 6.....		1									
0107 ADENOVIRUS TYPE 7.....		1					1				
0201 INFLUENZA A VIRUS.....		7									
0203 INFLUENZA B VIRUS.....		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		7									
0302 PARAINFLUENZA VIRUS TYPE 2....		6									
0303 PARAINFLUENZA VIRUS TYPE 3....		22									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		8						1			1
0500 RHINOVIRUS (ALL TYPES).....		15		1							
0600 MYCOPLASMA PNEUMONIAE.....	1	6									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0809 COXSACKIEVIRUS A9.....	2			1			2				
0816 COXSACKIEVIRUS A16.....	1										
0901 COXSACKIEVIRUS B1.....				1							
0905 COXSACKIEVIRUS B5.....							4				
1003 ECHOVIRUS TYPE 3.....						1					
1007 ECHOVIRUS TYPE 7.....				9			1				
1009 ECHOVIRUS TYPE 9.....		1									
1011 ECHOVIRUS TYPE 11.....		1		2		1	1				
1020 ECHOVIRUS TYPE 20.....		1									
1022 ECHOVIRUS TYPE 22.....		2					1				
1024 ECHOVIRUS TYPE 24.....	1										
1030 ECHOVIRUS TYPE 30.....				1							
1031 ECHOVIRUS TYPE 31.....				1							
1200 MUMPS VIRUS.....		1	1	5							
1301 HERPES SIMPLEX VIRUS NOT-TYPED			3								8
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	2	1					1				
1303 VARICELLA-ZOSTER VIRUS.....	1	1									6
1306 HERPES SIMPLEX TYPE 1.....	3	2									50
1307 HERPES SIMPLEX TYPE 2.....	4										34
1401 COXIELLA BURNETI.....	2	1									
1521 MEASLES VIRUS.....	3		1			1					4
1522 RUBELLA VIRUS.....	2	1									11
1532 HEPATITIS B ANTIGEN.....	65							27			
1535 HEPATITIS A ANTIBODY.....								13			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	1								1	
1556 CMV - CYTOMEGALOVIRUS.....	3	9		1			1			2	1
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....				1			19				
1571 ENTEROVIRUS TYPE 71 (BRCR)....				1							
9992 ROSS RIVER VIRUS.....	1										5
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	93	117	5	25		3	35	41		3	120

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 17/1/85 to 30/1/85 ... 85/3
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....								1		
0102 ADENOVIRUS TYPE 2.....										1
0103 ADENOVIRUS TYPE 3.....	2									
0106 ADENOVIRUS TYPE 6.....			1							1
0137 ADENOVIRUS TYPE 37.....		2								
0201 INFLUENZA A VIRUS.....							1			
0203 INFLUENZA B VIRUS.....					1			1		
0302 PARAINFLUENZA VIRUS TYPE 2....									1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....				1					1	
0500 RHINOVIRUS (ALL TYPES).....			1					1		1
0600 MYCOPLASMA PNEUMONIAE.....								1		
0809 COXSACKIEVIRUS A9.....								1		1
0816 COXSACKIEVIRUS A16.....										1
0905 COXSACKIEVIRUS B5.....							1	1		
1007 ECHOVIRUS TYPE 7.....							1	2		
1011 ECHOVIRUS TYPE 11.....									1	1
1021 ECHOVIRUS TYPE 21.....								1		
1030 ECHOVIRUS TYPE 30.....								1		
1101 POLIOVIRUS TYPE 1.....									1	
1200 MUMPS VIRUS.....			6			1				
1301 HERPES SIMPLEX VIRUS NOT-TYPED			1						1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			10					5		
1303 VARICELLA-ZOSTER VIRUS.....								1		
1306 HERPES SIMPLEX TYPE 1.....	4	39								
1307 HERPES SIMPLEX TYPE 2.....		192							3	
1401 COXIELLA BURNETI.....					2		2	8		
1521 MEASLES VIRUS.....								2		
1522 RUBELLA VIRUS.....					2			4	1	
1532 HEPATITIS B ANTIGEN.....									14	
1535 HEPATITIS A ANTIBODY.....								1		
1541 CHLAMYDIA A - C.TRACHOMATIS...		192								
1556 CMV - CYTOMEGALOVIRUS.....		3	2			4	2	2	3	1
1564 ROTAVIRUS.....									1	
9992 ROSS RIVER VIRUS.....					11			4		
9997 KUNJIN VIRUS.....					1			1		
9998 ARBO. GROUP B.								1		
Total.....	6	428	21	1	17	5	7	39	27	7