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Communicable Diseases Intelligence

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Contents:

- Haemophilus influenzae surveillance.
- Arbovirus surveillance - NSW(1983-84).
- Arbovirus infection in horses - Victoria(1984).
- S. mississippi surveillance.

VIRUS REPORTING SCHEME - A total of 1598 reports were processed this period. Although there is little evidence of widespread influenza activity in the community at present, 31 reports of influenza A and six of influenza B infection were received. The reports from Fairfield Hospital, Melbourne, included ten isolates of influenza A, subtype H_3N_2 resembling A/Philippines/2/82, from all age groups; and one isolate of subtype H_1N_1 (A/England/333/80-like) from a five year old boy. The 12 untyped influenza A isolates reported from the Royal Children's Hospital, Melbourne, and six of the seven isolates from the State Health Laboratory Services, Perth, were all from young children. Twenty-two influenza A strains (15 of H_1N_1; 7 of H_3N_2) have also been isolated at the OIC WHO Influenza Reference Centre, Melbourne. Most of the strains have been recovered from university students. Preliminary antigenic analyses have indicated that the H_3N_2 strains resemble A/Philippines/2/82 and the H_1N_1 strains exhibit some identity with A/England/333/80 and A/India/6263/80. Two influenza B strains isolated from Melbourne cases have been characterised as B/USSR/100/83-like, but an isolate referred from Brisbane bore closer resemblance to B/Singapore/222/79.

Interpretation of influenza laboratory data is always hampered by the problems of patients' age and the site chosen for specimen collection. Specimens are not usually taken from the elderly, and in the case of young children throat swabs are more easily obtained than blood samples. Consequently, for the older age group, which is usually most vulnerable to severe influenza infection and excess mortality, there is usually a paucity of available laboratory-based data. Conversely for the younger age groups, the number of reported virus isolates relative to seroconversions may be higher than for adults. In addition, if there is difficulty growing the virus, influenza activity will be underestimated.

. Enteric infections - Adenovirus type 40 was isolated in monkey kidney cells at Fairfield Hospital from faeces of a 31 year old male with gastroenteritis. Routine cell culture usually has had little success in isolating these fastidious adenoviruses (see CDI (1984) 84/8). Rotavirus was detected by ELISA at the Institute of Clinical Pathology and Medical Research, Sydney, in stools from a 55 year old female who had been eating oysters.

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HAEMOPHILUS INFLUENZAE SURVEILLANCE

(Contributed by N. Sonenberg and J.A. Gillians, Goulburn Valley Base Hospital, Shepparton, Victoria).

Two cases of meningitis caused by β-lactamase producing strains of Haemophilus influenzae were admitted recently to the hospital, representing 33% of all H. influenzae meningitis cases seen over the past year, and the first β-lactamase producers isolated since records were first kept 16 years ago.

CASE 1 - A 2 1/2 year old girl was admitted on 18 December 1983 with a two day history of fever, lethargy, anorexia and vomiting. Treatment with Bactrim syrup (trimethoprim - sulphamethoxazole) had been instituted for suspected otitis media. Examination revealed fever, photophobia, and neck stiffness. Lumbar puncture showed opaque CSF with 2000 neutrophils per c.mm. and numerous Gram-negative rods. Culture yielded a profuse growth of H. influenzae type b, which produced β-lactamase when tested with chromogenic cephalosporin (Nitrocephin, Oxoid). The strain was resistant to ampicillin, co-trimoxazole, tetracycline; and sensitive to cephalothin, gentamicin and chloramphenicol. Treatment initially was with intravenous penicillin, chloramphenicol and gentamicin, but after one day only chloramphenicol was continued for a total of ten days. The patient made a complete recovery. No epidemiological studies were performed.

CASE 2 - A two year old boy was admitted on 7 July 1984 with a one week history of upper respiratory tract infection with recent vomiting, ataxia and signs of meningism. Lumbar puncture revealed opaque CSF with 2900 neutrophils, 65 lymphocytes, and 200 erythrocytes per c.mm. Numerous Gram-negative rods were seen, and culture yielded a profuse growth of β-lactamase H. influenzae type b. The strain was resistant to ampicillin and cephalothin; and sensitive to chloramphenicol, tetracycline, gentamicin and co-trimoxazole. Treatment with chloramphenicol for seven days intravenously and a further five days orally produced some clinical improvement, although fever and ataxia persisted. A CAT scan two weeks after admission failed to find a localised lesion, and recovery is continuing. Throat swabs were taken from both parents and two siblings, but these failed to grow any significant organisms. No prophylaxis was given, and no secondary cases of meningitis have developed.

Despite the recent incidence of β-lactamase producing strains of H. influenzae isolated from the CSF, none of the respiratory isolates cultured at the hospital have been penicillinase producers. Neither of the two patients had a history of a previous long-term antibiotic usage, but in both cases the use of chloramphenicol, singly or in combination, provided adequate cover until the results of sensitivity testing were available.

Editorial Comment

Systemic H. influenzae type b infections are of major clincial and public health importance, with an estimated 20-30,000 cases occurring annually in the USA.(1) The most frequent manifestation of the bacteraemic infections is meningitis, followed by epiglottitis, arthritis, cellulitis and pneumonia.(2) Nearly all cases of disease occur in children younger than five years; half of all cases occur in infants.(3) Meningitis is associated with a mortality of 5-10% and is estimated to leave permanent sequelae in 30% of patients or more.(4) Two issues with systemic infections have emerged over recent years; the evolution and rapid spread

of antibiotic resistance (most notably to ampicillin) determined by plasmid or chromosomal genes causing special problems in treatment[5,6]; and the increased risk (between 2-6%) of secondary disease among household child contacts and in child day-care facilities[7,8].

Initial hopes for preventing both primary and secondary disease by immunisation with a vaccine based on the capsular polysaccharide of H. influenzae type b[9,10] have been frustrated by the fact that good protective efficacy is only evident in children at least 18 months of age[11,12]. This finding has placed severe limitations on the use of the vaccine, since the highest incidence of H. influenzae meningitis occurs in children about one year of age, or in some populations in even younger children[13,14]. However, a recent review of 956 bacteraemic H. influenzae infections occurring in Finland over a five year period showed that 60% of infections were in children between the ages of 18 months and nine years[12]. Thus with a vaccine efficacy of 90%, up to 50% of all invasive Haemophilus infections of children could be prevented. The development of improved second-generation vaccines[15-18] that would prevent the disease at an earlier age are still some way off.

In the USA, the problem of secondary disease among household child contacts and those that attend child day-care facilities has been addressed with the recommendation for the prophylactic use of rifampicin[19,20]. However, the study on which the recommendations were based also showed that prophylaxis alone would only reduce the endemic incidence of disease by 1.2%.[21] Interpretation of the efficacy of rifampicin chemoprophylaxis has also been questioned, and further studies have been advocated.[22] Nevertheless, even with the availability of improved vaccines, consideration must still be given to the use of prophylactic measures for unimmunised children and for infants younger than six months who might have had insufficient time to respond to vaccination and who are exposed to a patient with systemic Haemophilus disease. Regardless, chemoprophylaxis is not a substitute for parent education; exposed children need careful observation and should be examined by a physician at the earliest signs of an unexplained febrile illness.

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ARBOVIRUS SURVEILLANCE, NEW SOUTH WALES (1983-84)

(Contributed by H.M. Naim, R.A. Hawkes, C.R. Boughton, B. Myrick and B. Cameron, Arbovirus Research Unit, University of New South Wales, Kensington, Sydney).

In early December 1983, as an adjunct to an extensive study in humans, sentinel flocks of seronegative chickens were stationed at nine sites in southern New South Wales and seven sites in the north of the State. The southern flocks (at Albury, Berrigan, Deniliquin, Griffith, Hay, Leeton, Narrandera, Wagga Wagga and Wentworth) were bled at weekly intervals and tested immediately as part of the Australian encephalitis virus early-warning program. The northern flocks (at Bourke, Broken Hill, Menindee, Wilcannia, Narrabri, Gunnedah and Kempsey) were bled at monthly intervals and tested retrospectively. There were approximately 15 birds (14-20) in each flock and all were tested by the haemagglutination-inhibition (HI) method for antibodies to the alphaviruses - Sindbis and Ross River, and to the flaviviruses - Murray Valley encephalitis, Kunjin, Alfuy, Kokobera, Stratford, and Edge Hill. The final sampling of the flocks was done in April 1984. A summary of the seroconversions is given in Table 1.

TABLE 1. Seroconversions to arboviruses in sentinel chickens - New South Wales (1983-84)

Town	Sindbis seroconversions	Week of first seroconversion	Flavivirus seroconversions	Week/month* of first seroconversion
Albury	7	16 January	2	19 March
Berrigan	15	9 December	0	-
Deniliquin	7	9 January	0	-
Griffith	8	3 January	1	12 March
Hay	8	19 December	0	-
Leeton	13	3 January	2	2 March
Narrandera	15	3 January	3	12 March
Wagga Wagga	7	14 February	2	19 March
Wentworth	16	3 January	4	12 March
Bourke*	11	13 December	5	13 March
Broken Hill*	6	24 January	0	-
Menindee*	11	17 January	5	3 April
Wilcannia*	10	3 April	10	3 April
Narrabri*	9	7 March	0	-
Gunnedah*	12	20 February	1	2 April
Kempsey*	1	27 March	0	-

Sindbis seroconversions occurred in all flocks, beginning at Bourke and most of the southern flocks soon after the establishment of the study. In most flocks more than half of the birds became infected, the notable exception being Kempsey (one bird only). This finding may be associated with the relatively low frequency of Sindbis antibodies detected in humans on the New South Wales coast (unpublished observations). Predictably, Ross River virus antibodies were not detected in the chickens despite frequent high Sindbis titres.

Flavivirus seroconversions occurred in only ten of the 15 flocks, and not until much later in the season (i.e. between 2 March - 3 April). It was noteworthy that six widely distributed flocks seroconverted in the same period (12-19 March; Wentworth, Narrandera, Griffith, Bourke, Albury and Wagga Wagga). The proportion of each flock showing seroconversion was generally lower than those shown by Sindbis virus. The pattern of HI titres indicated that Kunjin was

probably the infecting virus in most (25/35) instances. This was confirmed in three chickens by neutralisation tests (I.D. Marshall, Australian National University, Canberra). In eight chickens, the HI response was too broad to permit presumptive identification of the infecting virus. An unusual antibody pattern was seen in three chickens, one each at Griffith, Albury and Wagga Wagga. In these birds, HI titres were monospecifically or predominantly to Kokobera antigen, and neutralisation tests will be carried out on these sera. There was no serological evidence of MVE in 1983-84.

In mid- February, a three month old female dog at Bourke developed an acute neurological disorder of about one week's duration characterised by collapse and limb rigidity (front and hind limbs), with eventual recovery. Accompanying this was the development of a broadly reactive HI response to all six flavivirus antigens, highest titres being to MVE, Kunjin and Stratford. It seems likely that this episode represents a clinical infection by an as yet undetermined flavivirus; and a pilot study using sentinel dogs is planned in Bourke in 1984-85.

The recent wet conditions were presumably responsible for the great contrast in seroconversion rates between the summer of 1983-84 and that of 1982-83, in which no seroconversions were detected (see CDI 83/22). The explanation for alphavirus infections preceding flavivirus infections by 2-3 months is currently unknown, and highlights our relative ignorance of the basic vector-host cycles of these viruses. The relatively specific HI responses of chickens for flaviviruses again in 1981-82 (see CDI 82/22) enabled presumptive identification of Kunjin as the predominant virus infecting these chickens.

ARBOVIRUS INFECTION IN HORSES - VICTORIA 1984

(Contributed by R.T. Badman, Regional Veterinary Laboratory, Bendigo; and J. Campbell and J. Aldred, Attwood Veterinary Research Laboratory, Westmeadows, Melbourne).

EQUINE ROSS RIVER VIRUS INFECTION - The occurrence of an unusual equine nervous disease has lead to the recent recognition of a high prevalence of infection with Ross River virus (RRV) in horses in northern and central Victoria. In the period January-April 1984, veterinary practitioners from northern Victoria attended many horses displaying locomotor disturbances. The clinical signs noted included disinclination to move, incoordination, staggery gait, cutaneous hyperaesthesia and fasciculation in the major appendicular muscles. Joint involvement was not seen and recovery occurred within 2-5 days. A viral aetiology was suspected and serological testing for a range of viruses, including RRV, was undertaken on both affected and unaffected horses in the same area. Of 53 clinically affected horses, 42 had haemagglutination inhibition (HI) titres against RRV, with 24 exceeding titres of 1/640. In 25 normal horses, 18 were positive in the HI test for RRV and nine of these showed distinct inhibition zones with the more sensitive plaque inhibition test. One of these "normal" horses subsequently became affected with the locomotor disturbance.

The disease outbreak this year repeated a pattern established in 1982 when clinically affected horses were first seen in January in the Mildura area. Further cases followed in February and March along the Murray and Goulburn Valleys. Attempts have been made to isolate viruses from some of the affected horses, but all have been unsuccessful. The aetiology of the disease therefore remains unknown, but its occurrence in a period of active infection with RRV suggests that this agent may be involved.

ENCEPHALOMYELITIS IN HORSES - As a consequence of the efforts to isolate RRV from clinically affected horses, two yearlings with severe nervous signs necessitating euthanasia were submitted for pathological examination. Both horses initially showed signs of colic. Within 12 hours posterior paralysis was noted; lateral recumbency, cutaneous anaesthesia, marked dullness and convulsions ensued. A severe non-suppurative encephalomyelitis was present in both horses, with lesions predominating in the thalamus, mid brain, pons and medulla.

Kunjin virus was isolated from the cervical spinal cord of one horse. This animal had HI titres of 1/80 to Sindbis and 1/640 to MVE and Kunjin. It was negative by HI and plaque inhibition for RRV. No viruses were isolated from the second horse which had an HI titre of 1/40 to RRV, but serological testing for other viruses was not done. This finding is considered to be the first isolation of Kunjin virus from a horse with encephalomyelitis, and further confuses the role of arboviruses in the sporadic encephalomyelitides of animals in Australia.

SALMONELLA MISSISSIPPI SURVEILLANCE - TASMANIA

(Contributed by A. Ball, Public Health Laboratory, Royal Hobart Hospital, Tasmania).

Reports of human S. mississippi infections have generally been confined to Tasmania, or have been traced back to patients who have been there. During the past two months, a survey of wild animals has been undertaken in search of a reservoir of the serotype on the island. The animals were trapped and released after faecal samples were obtained, and the isolations are given in Table 1.

TABLE 1. S. mississippi isolations from Tasmanian fauna (1984)

Animal	No. tested	No. isolations
Quoll (Dasyurus viverrinus)	13	7
Tasmanian Devil (Sarcophillus harrisii)	11	3
Possum (Trichosurus vulpecula)	1	0
Bandicoot (Peramales gunii)	1	0
Feral cat	1	0

S. mississippi was isolated from seven Quolls and three Tasmanian Devils. No other serotypes were encountered apart from a rough Salmonella with the same H-antigens as S. mississippi, which was isolated from one of the Quolls carrying S. mississippi. The epidemiology of infection from these carnivorous marsupials to humans is under investigation. To date, no isolations have been made from Moore's swabs of creeks in the trapping areas.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 2/8/84 - 15/8/84 BULLETIN NUMBER 84/17

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	1		4		10	3	12	1	31
0101 ADENOVIRUS TYPE 1	2	1		3		5			11
0102 ADENOVIRUS TYPE 2	1			1		1		1	4
0103 ADENOVIRUS TYPE 3				4		1			5
0105 ADENOVIRUS TYPE 5						4			4
0106 ADENOVIRUS TYPE 6			1			1		1	3
0107 ADENOVIRUS TYPE 7						1		1	2
0108 ADENOVIRUS TYPE 8				1				2	3
0118 ADENOVIRUS TYPE 18				1					1
0119 ADENOVIRUS TYPE 19				2				4	6
0137 ADENOVIRUS TYPE 37								13	13
0140 ADENOVIRUS TYPE 40				1					1
0199 ADENOVIRUS TYPING PENDING		4			1	5			10
0201 INFLUENZA A VIRUS			1		12			7	20
0202 INFLUENZA A VIRUS SUBTYPE H3N2				10					10
0203 INFLUENZA B VIRUS	3		1				1	1	6
0206 INFLUENZA A VIRUS SUBTYPE H1N1				1					1
0301 PARAINFLUENZA VIRUS TYPE 1	2			1	1	8	4		16
0302 PARAINFLUENZA VIRUS TYPE 2	2			6	1	13	1	1	24
0303 PARAINFLUENZA VIRUS TYPE 3					2	7	2	3	14
0399 PARAINFLUENZA VIRUS TYPING PENDING		1		1		2			4
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	30	20	7	29	31	65	33	32	247
0500 RHINOVIRUS (ALL TYPES)	2		1	7	12	14	12	4	52
0600 MYCOPLASMA PNEUMONIAE	9	2	5	2		3	1	1	23
0700 ORNITHOSIS-PSITTACOSIS	1			2					3
0816 COXSACKIEVIRUS A16	2			1					3
0902 COXSACKIEVIRUS B2						1			1
0905 COXSACKIEVIRUS B5	1			1		3	1		6
1000 ECHOVIRUS NOT TYPED							2		2
1009 ECHOVIRUS TYPE 9				4					4
1017 ECHOVIRUS TYPE 17				1					1
1101 POLIOVIRUS TYPE 1	3	1					2	1	7
1102 POLIOVIRUS TYPE 2				1		1	1		3
1103 POLIOVIRUS TYPE 3						2			2
1104 POLIOVIRUS-VACCINAL STRAIN			3				1		4
1200 MUMPS VIRUS	3		1	7			1		12
1300 HERPES VIRUS GROUP-NOT TYPED	26		2	8		3		3	42
1301 HERPES SIMPLEX VIRUS NOT-TYPED	2	3		5					10
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5	2			1			16	24
1303 VARICELLA-ZOSTER VIRUS	2	1		1		3		3	10
1306 HERPES SIMPLEX TYPE 1	12			33		32	33	12	122
1307 HERPES SIMPLEX TYPE 2	87			63		40	77	34	301
1399 HERPES VIRUS TYPING PENDING			7		2	1			10
1401 COXIELLA BURNETI	1					1	3	1	6
1502 PICORNA VIRUS-NOT TYPED	5		4				1		10
1521 MEASLES VIRUS	1	2			1			1	5
1522 RUBELLA VIRUS	1			1		5	2	4	13
1532 HEPATITIS B ANTIGEN	45		9			53	7	8	122
1534 HEPATITIS B ANTIGEN AND ANTIBODY	2								2
1535 HEPATITIS A ANTIBODY	1		1			3	2		7
1541 CHLAMYDIA A - C TRACHOMATIS	24		4				10	57	95
1556 CMV - CYTOMEGALOVIRUS	10		2	18	4	12	5	9	60
1562 REOVIRUS (ALL TYPES)					1				1
1564 ROTAVIRUS	30	6	39	5	12	40	3	12	147
1599 ENTEROVIRUS TYPING PENDING		2	14		6	2			24
9901 ARBO. GROUP A.(UNSPECIFIED)				2					2
9992 ROSS RIVER VIRUS							4	15	19
9994 SMALL VIRUS (LIKE) PARTICLE		4							4
9995 DENGUE							2		2
9997 KUNJIN VIRUS							1		1
Total	316	49	106	223	97	335	224	248	1,598

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 2/8/84 to 15/8/84 .... 84/17

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.....	1	8					2				
0102 ADENOVIRUS TYPE 2.....	1	1					1				
0103 ADENOVIRUS TYPE 3.....		3									
0105 ADENOVIRUS TYPE 5.....		2					2				
0106 ADENOVIRUS TYPE 6.....	1						2				
0107 ADENOVIRUS TYPE 7.....							1				
0140 ADENOVIRUS TYPE 40.....							1				
0201 INFLUENZA A VIRUS.....		18									
0202 INFLUENZA A VIRUS SUBTYPE H3N2		9					2				
0203 INFLUENZA B VIRUS.....	1	2				1					
0206 INFLUENZA A VIRUS SUBTYPE H1N1		1									
0301 PARAINFLUENZA VIRUS TYPE 1....		16									
0302 PARAINFLUENZA VIRUS TYPE 2....	1	22		1			1				
0303 PARAINFLUENZA VIRUS TYPE 3....		13					1				
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	4	234					4				1
0500 RHINOVIRUS (ALL TYPES).....		55									
0600 MYCOPLASMA PNEUMONIAE.....	4	16							1		
0700 ORNITHOSIS-PSITTACOSIS.....		3									
0816 COXSACKIEVIRUS A16.....				1							2
0902 COXSACKIEVIRUS B2.....							1				
0905 COXSACKIEVIRUS B5.....		5					1				
1009 ECHOVIRUS TYPE 9.....		1		2							
1017 ECHOVIRUS TYPE 17.....		1									
1101 POLIOVIRUS TYPE 1.....	2	2					3				
1102 POLIOVIRUS TYPE 2.....		2					1				
1103 POLIOVIRUS TYPE 3.....		1					1				
1104 POLIOVIRUS-VACCINAL STRAIN....							3				
1200 MUMPS VIRUS.....	3	2	1	3							
1300 HERPES VIRUS GROUP-NOT TYPED..											1
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1									2	6
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	6	4									1
1303 VARICELLA-ZOSTER VIRUS.....											8
1306 HERPES SIMPLEX TYPE 1.....	4	8			1					2	56
1307 HERPES SIMPLEX TYPE 2.....	14						1				50
1401 COXIELLA BURNETI.....	1										
1502 PICORNA VIRUS-NOT TYPED.....							4				
1521 MEASLES VIRUS.....	1					1					2
1522 RUBELLA VIRUS.....	2					1					4
1532 HEPATITIS B ANTIGEN.....	66						2	29			1
1534 HEPATITIS B ANTIGEN AND ANTIBODY.....								1			
1535 HEPATITIS A ANTIBODY.....	2							4			
1541 CHLAMYDIA A - C.TRACHOMATIS...	2										
1556 CMV - CYTOMEGALOVIRUS.....	7	17			1			1	1	4	
1564 ROTAVIRUS.....	11						128				1
9901 ARBO. GROUP A.(UNSPECIFIED)...											2
9992 ROSS RIVER VIRUS.....	6	1				1					3
9994 SMALL VIRUS (LIKE) PARTICLE...							3				1
9995 DENGUE.....	2										
9997 KUNJIN VIRUS.....		1									
Total.....	143	449	1	7	2	4	168	35	2	8	139

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 2,8,84 to 15,8,84 ... 84/17

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
0102 ADENOVIRUS TYPE 2.....								1		
0103 ADENOVIRUS TYPE 3.....	1		1					2		
0107 ADENOVIRUS TYPE 7.....	1									
0108 ADENOVIRUS TYPE 8.....	1	2								
0118 ADENOVIRUS TYPE 18.....	1									
0119 ADENOVIRUS TYPE 19.....	3	3								
0137 ADENOVIRUS TYPE 37.....	4	9								
0201 INFLUENZA A VIRUS.....					1		1			
0202 INFLUENZA A VIRUS SUBTYPE H3N2								4		
0203 INFLUENZA B VIRUS.....							1	2		
0206 INFLUENZA A VIRUS SUBTYPE H1N1								1		
0303 PARAINFLUENZA VIRUS TYPE 3....							1			
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....							1	2	2	2
0500 RHINOVIRUS (ALL TYPES).....						1	2	1		
0600 MYCOPLASMA PNEUMONIAE.....								1	2	
1009 ECHOVIRUS TYPE 9.....								1		
1104 POLIOVIRUS-VACCINAL STRAIN....										1
1200 MUMPS VIRUS.....			6					2	1	
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1			2	1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			9	1			1	5	1	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	5	44						1	2	
1307 HERPES SIMPLEX TYPE 2.....		235			1				1	
1401 COXIELLA BURNETI.....			1				1	3		
1521 MEASLES VIRUS.....									1	
1522 RUBELLA VIRUS.....				1	7			1		
1532 HEPATITIS B ANTIGEN.....		1							24	
1534 HEPATITIS B ANTIGEN AND ANTIBODY.....									1	
1535 HEPATITIS A ANTIBODY.....									1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	91							1	
1556 CMV - CYTOMEGALOVIRUS.....		9	3			4	2		11	
1562 REOVIRUS (ALL TYPES).....									1	
1564 ROTAVIRUS.....									6	
9901 ARBO. GROUP A.(UNSPECIFIED)...					2					
9992 ROSS RIVER VIRUS.....					9			2		
9994 SMALL VIRUS (LIKE) PARTICLE...								1		
9997 KUNJIN VIRUS.....					1					
Total.....	17	394	20	3	21	5	12	31	56	3