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Communicable Diseases Intelligence

Bulletin number 84/5

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Contents:

- . Herpes simplex encephalitis.
- . Nosocomial Norwegian scabies.
- . Aetiology of cat-scratch disease.

VIRUS REPORTING SCHEME - A total of 1417 reports were received this period.

- . Arbovirus infections - The 237 clinical and serologically confirmed cases of Ross River virus infection exhibited a State distribution of New South Wales (97), Queensland (56), South Australia (43), Victoria (20), Western Australia (11), Northern Territory (1) and unspecified (9); a male: female ratio of 1:1.1; and an age range of 1-74 years. Specific IgM against Kunjin virus was detected by the State Health Laboratory, Brisbane, in a female from an unspecified location in New South Wales. Surveys in the Murray-Darling basin showed that virus activity (Ross River, Sindbis) was first detected in November and Culex annulirostris were in plague proportions in December and January this season. However, despite widespread breeding of ducks and swans in response to the breaking of the drought, herons and cormorants were extremely rare in the Riverina, and no breeding colonies were seen. Therefore it may be speculated that the absence of relevant avian species spared the region from an epidemic of Australian encephalitis (I. Marshall, Australian National University, personal communication).
- . Neonatal infections - Echovirus type 17 was isolated by Prince Henry Hospital, Sydney, from faeces, saliva, urine and liver biopsy from a fatal case of lower respiratory tract infection and hepatic disease in a male neonate. Hepatitis has been associated with enterovirus infections, particularly group B coxsackieviruses, usually as part of severe multisystem disease in neonates. Herpes simplex virus type 1 was isolated by Fairfield Hospital, Melbourne, from saliva, mouth swabs and skin lesions of a ten day old neonate. The mother was undergoing methadone treatment for drug addiction. The infant was given acyclovir, but her symptoms did not develop beyond skin lesions on the chest, ulcers in the mouth and slight fever. Vaginal swabs taken of the mother were negative for genital herpes, and no extraneous source of infection could be established. The hospital also isolated herpes simplex virus type 2 from three pregnant mothers with active genital lesions at 36 weeks gestation. Other neonatal infections included Chlamydia trachomatis inclusion conjunctivitis, one diagnosed by the State Health Laboratory, Brisbane, and the other by the State Health Laboratory Services, Perth. A single CF antibody titre against

(continued on page 5)

(Contributed by M. Bucens, State Health Laboratory Services, Perth).

Following spontaneous onset of labour at 35 weeks gestation, a 2000 g baby was delivered to a 26 year old mother. The boy was the woman's first child. The baby thrived after birth, was breast-fed, and was discharged one week after delivery.

At two weeks, the infant became mildly unwell (not eating and drinking as before). He was admitted to hospital, when he was noted to be small for gestational age, pale and quiet. He had thrush of the mouth and palate, weighed 2095 g and had normal temperature. A lumbar puncture on admission revealed 134 white blood cells/cmm of which 99% were mononuclear. Within 24 hours the child became desparately ill and started to have intractable seizures. He rapidly became comatose. An ultra-sound performed on the second day showed that the lateral ventricles were very compressed (consistent with marked cerebral oedema). There was no evidence of intracranial haemorrhage.

Another week later a further ultra-sound showed haemorrhage in the thalamic region, normal sized ventricles and reduction of brain matter. Two days before death the infant developed vesicular lesions on the skin - on his back, abdomen, soles of feet etc. Herpes simplex virus (HSV) type 2 was isolated from these vesicles. The child died two weeks after admission, aged four weeks.

The mother gave no history of previous genital herpes infection and no attempt was made to take specimens from her. At post mortem the infant's brain had been reduced to a shell of cortex around normal "ventricles". HSV was not isolated from post mortem material, but this was not surprising because of the advanced stage of necrosis. A diagnosis of acute, diffuse, necrotic encephalitis, presumeably due to HSV, was made.

Editorial Comment

The risks to the newborn of primary and recurrent HSV-1 and HSV-2 infections during pregnancy have yet to be completely defined, since the prevalence of infection at term, the transmission rate to the neonate, and the effect, if any, of low grade or asymptomatic neonatal infections on the future development of the child remain unknown⁽¹⁾. The virus is most commonly transmitted to the newborn during delivery, but transplacental⁽²⁾ and postnatal nosocomially acquired infections⁽³⁾ have been reported. The clinical presentation of neonatal herpes infection ranges from localised cutaneous infection to disseminated herpetic infection involving the central nervous system.

Data from the National Institute of Allergy and Infectious Disease Collaborative Antiviral Study Group together with a review of over 400 published cases of neonatal HSV infections⁽⁴⁾ showed that only 10% of mothers had clinical manifestations of genital infection. Of the asymptomatic women in the study, 13% gave a history of genital HSV infection, 20% reported infection in their sexual partners, and more than 50% had no clinical or historical evidence of infection. Indeed, widespread screening for HSV of all women at delivery has produced low prevalence of positive diagnoses⁽⁵⁾. As a result it is impossible to know whether the benefits of caesarian section for subclinical HSV infection outweigh the

risks of its anaesthetic and postoperative complications. Most of the current recommendations focus on a group of high risk women, generally defined as those with a documented or probable episode of herpetic vulvovaginitis in the 12-18 months before conception, women who develop herpetic vulvovaginitis during gestation, women with frequent episodes of herpetic labialis or cutaneous herpes, women whose sexual consorts experience genital herpes or oral infections, and women with active lesions on thighs and buttocks. If there is evidence of active viral shedding up to one week prior to term, caesarian section should be considered. However, since even weekly viral cultures among women with recurrent herpes would diagnose asymptomatic viral shedding in only 25% of women at the time of delivery, and since a rapid, sensitive, inexpensive diagnostic test is not available, present diagnostic techniques are unlikely to reduce the incidence of neonatal herpes.⁽⁶⁾

Babies born to mothers with confirmed or suspected HSV infection must be carefully observed for at least two weeks to detect possible neonatal infection. Swabs should be taken from any skin lesions, and from the eyes, mouth, throat, nose and rectum of the baby. Urine, buffy coat and CSF if available should also be cultured for virus. As the incubation period of neonatal HSV infection is variable, it is advisable to repeat these investigations during the first 14 days of life. Virus should be typed, and if virus from the mother or other contacts is available, the isolates should be finger-printed by DNA restriction endonuclease analysis to attempt to identify the source of the neonatal infection.

Recommendations for the prevention of nosocomial transmission of HSV are based on isolation procedures. If genital HSV is present at term, the mother should be given a single room, and attendant personnel should apply source isolation techniques using gowns and gloves⁽⁷⁾. Despite the high incidence of cold sores and asymptomatic infection among hospital staff, neonatal HSV type 1 infections are rare.

References

1. J. Hosp. Inf. (1983) 4: 217
2. Pediatrics (1980) 66: 495
3. CDWR (1982) 8: 241
4. Visintine, A.M., Nahmias, A.J., Whitley, R.J. and Alford, C.A. (1980). The natural history and epidemiology of neonatal herpes simplex virus infection. In: The Human Herpes Viruses. Ed. Nahmias, A.J., Dowdle, W.R. and Schinagi, R.F. Appendix 1. p. 599. Elsevier: New York.
5. Am. J. Obstet. Gynecol. (1979) 135: 547
6. JAMA (1983) 250: 3094
7. JAMA (1980) 243: 157.

NOSOCOMIAL NORWEGIAN SCABIES

(Based on CDR (1984) 84/07 and CDWR (1984) 10/2).

A parasitosis such as scabies arouses considerable emotional reaction among staff, administration and patients when outbreaks occur in hospital or nursing home environments. The CDR reference quoted above detailed an outbreak in an orthopaedic ward in a Shrewsbury hospital, UK, following the admission of an 89 year old woman with a fractured femur. Retrospective enquiry at the old persons' home where she stayed showed that some staff and residents also had clinical symptoms and/or signs indicative of scabies. At least 28 adults were affected, and the outbreak persisted for over two months before

it was recognized. The Canadian CDWR reference detailed two outbreaks, one in a nursing home following the admission of an 82 year old woman, and the other in a hospital centre. The index case was not determined in the second outbreak.

Scabies is not always easy to identify, and as it sometimes does not present with the characteristic itching rash associated with skin burrows, it may not be readily recognized. Norwegian scabies ("Norwegian itch") typically presents with a more extreme form of bulbous lesions or, as in the index case for the Shrewsbury outbreak, with thick crusts over the skin; it is accompanied by abundant mites but often only slight itching about which the patient may not complain especially if elderly or mentally inhibited. The causative parasite is Sarcoptes scabiei, indistinguishable from strains found in more typical skin infestation. Transmission is usually by prolonged skin-to-skin contact, and the parasite is rarely transmitted by social contact. Survival of the parasite away from the human body appears to be only a few days. Incubation period can be up to two months, so that itching may not be experienced until some days or even weeks after contracting infection, and is said to be caused by irritation by toxic secretions and excretions associated directly with burrowing. Atopic eczema may precede infestation of the index case and mask diagnosis. Mites may be extracted from skin lesions by means of a sharp-edged Hagedorn needle, or alternatively the parasite may be displaced by softening the overlying skin with the careful application of a 40% solution of potassium hydroxide, and then identifying the ascaris or eggs in skin scrapings.

Control of outbreaks requires close collaboration between nursing and hospital staff, family practitioners and house staff at nursing homes for the elderly. The policy adopted at the Shrewsbury outbreak was as follows: topical application of benzyl benzoate was advised for those affected together with all members of their family living in the same house and anyone who had shared a bed with an individual with a rash, whether or not they had an itch or rash. The advised procedure was: after a hot bath and drying, 25% benzyl benzoate (benzyl benzoate application) was applied over the whole body surface, omitting the head and neck, and left on overnight. On the following day, but without bathing, a further application of emulsion was applied and then washed off by bathing 24 hours later. Some authorities advise two applications of emulsion on the first day and a third 24 hours later together with a repeat course ten days later to destroy any mites which may hatch from surviving eggs. For children, a 1:2 dilution of benzyl benzoate was recommended, and a 1:3 dilution for infants; for the latter the head and neck needs to be painted also but with care to avoid the mouth and eyes. Patients were warned that they may continue to itch for several days after the treatment. After completing treatment with benzyl benzoate, all clothing and bed linen were changed for newly laundered clothing or linen. All used linen and sheets were washed; that from hospital staff and patients sent for laundering in alginate bags labelled "infected linen". It was not necessary for outer clothing to be cleaned provided it was not worn for three weeks, otherwise it was cleaned or dry-cleaned and the cuffs ironed (to destroy mites and eggs). Blankets and other bedding were not treated provided sheets were used.

THE AETIOLOGY OF CAT-SCRATCH DISEASE

(Based on California Morbidity (1984) No. 1).

Cat Scratch disease (CSD) is a subacute, self-limited, presumed infectious disease characterised by malaise, granulomatous lymphadenitis and variable patterns of fever. It is often preceded by a cat scratch which results in a local lesion that is followed by regional lymph node involvement which may progress to suppuration. The aetiology of CSD has remained unknown despite extensive efforts of medical scientists. Since the first recognition of this clinical entity in 1931 there have been numerous publications of laboratory studies seeking its aetiology.

Because extensive efforts to demonstrate a bacterial cause were unsuccessful a viral aetiology was assumed, but subsequent viral culture efforts and serological studies similarly failed to yield definitive results. However, a recent study has proposed a bacterium as the cause of CSD⁽¹⁾. Small, Gram-negative, silver-staining bacteria were detected in lymph node tissue from a single case in 1981, and in a prospective histopathological study of additional CSD cases, bacilli morphologically identical to those in the original case were found in lymph nodes of 34 of 39 patients studied but in none of the lymph nodes from 91 patients with other diseases. The occurrence of these small, pleomorphic, Gram-negative, non-acid fast intracellular bacilli in capillary walls and in macrophages in lymph node tissue of involved areas, their disappearance as the disease process resolved, and immunofluorescent staining of the bacilli by fluorescein-tagged convalescent serum from CSD patients appear to provide solid evidence for their aetiological role in the disease.

The diagnosis of CSD at present depends upon evidence such as the following; a bacterially-sterile, histologically characteristic lymphadenitis; malaise and fever; an inflammatory skin lesion at the inoculation site (in about one third of the cases); history of a scratch or bite by a cat (in most but not all cases); a positive skin test with a non-standardised, non-commercially available antigen prepared from human lymph node aspirates; and no evidence of any other cause.

Discovery of the aetiological agent should open the way to development of sensitive culture methods, preparation of purified antigens for skin tests and diagnostic serological tests, antibiotic sensitivity tests and determination of appropriate treatment, more accurate epidemiological studies, and knowledge of the agent's natural source and means of survival and propagation.

Reference

1. Science (1983) 221: 1403.

(continued from page 1)

Mycoplasma pneumoniae was also detected by the Institute of Medical and Veterinary Science, Adelaide, in a one month old infant with pneumonia. M. pneumoniae is an uncommon cause of pneumonia in young children, when most infections are apparently asymptomatic.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

 REPORTING PERIOD - 16/2/84 - 29/2/84 BULLETIN NUMBER .84/5
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR		PHH/	FAIR-			STATE	STATE	Total	
	(NSW)/ WVH (ACT)	RAHC (NSW)	POW (NSW)	FIELD (VIC)	RCH (VIC)	IMVS (SA)	LAB (QLD)	LAB (WA)		
0100 ADENOVIRUS NOT TYPED.....		1		4		3	1	5	1	15
0101 ADENOVIRUS TYPE 1.....							2			2
0102 ADENOVIRUS TYPE 2.....					1		2			8
0103 ADENOVIRUS TYPE 3.....						6	1			7
0104 ADENOVIRUS TYPE 4.....							1			1
0105 ADENOVIRUS TYPE 5.....						3	2			5
0106 ADENOVIRUS TYPE 6.....						1	1			2
0108 ADENOVIRUS TYPE 8.....				2						2
0111 ADENOVIRUS TYPE 11.....					1					1
0118 ADENOVIRUS TYPE 18.....							2			2
0119 ADENOVIRUS TYPE 19.....	5									5
0131 ADENOVIRUS TYPE 31.....							1			1
0199 ADENOVIRUS TYPING PENDING.....				1		9	4			14
0201 INFLUENZA A VIRUS.....	1				1			1		3
0301 PARAINFLUENZA VIRUS TYPE 1.....	1					5			2	8
0302 PARAINFLUENZA VIRUS TYPE 2.....						1				1
0303 PARAINFLUENZA VIRUS TYPE 3.....	2					5	2	4	4	17
0399 PARAINFLUENZA VIRUS TYPING PENDING.....						2				2
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)....						1			1	2
0500 RHINOVIRUS (ALL TYPES).....	3				5	9	1	6	2	26
0600 MYCOPLASMA PNEUMONIAE.....	10	1	3	15	5	29	11	5		79
0700 CRNITHOSIS-PSITTACOSIS.....				1						1
0802 COXSACKIEVIRUS A2.....								3		3
0809 COXSACKIEVIRUS A9.....								1		1
0899 COXSACKIEVIRUS GROUP A TYPING PENDING.....								3		3
0902 COXSACKIEVIRUS B2.....						1				1
0903 COXSACKIEVIRUS B3.....							1			1
0905 COXSACKIEVIRUS B5.....	1									1
1000 ECHOVIRUS NOT TYPED.....								7		7
1003 ECHOVIRUS TYPE 3.....	1									1
1004 ECHOVIRUS TYPE 4.....								1		1
1006 ECHOVIRUS TYPE 6.....							1			1
1009 ECHOVIRUS TYPE 9.....					1	3	2	2		8
1014 ECHOVIRUS TYPE 14.....						1		2	1	4
1017 ECHOVIRUS TYPE 17.....				2						2
1022 ECHOVIRUS TYPE 22.....								1		1
1101 POLIOVIRUS TYPE 1.....	3									3
1102 POLIOVIRUS TYPE 2.....	2						1			3
1103 POLIOVIRUS TYPE 3.....	2							1		3
1104 POLIOVIRUS-VACCINAL STRAIN.....				3		1				4
1200 MUMPS VIRUS.....	4	1			3					8
1300 HERPES VIRUS GROUP-NOT TYPED.....	22			4	3		6		4	39
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....		4			2			1		7
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	7	1				1			7	16
1303 VARICELLA-ZOSTER VIRUS.....	6			1						7
1306 HERPES SIMPLEX TYPE 1.....	17				33		23	24	17	114
1307 HERPES SIMPLEX TYPE 2.....	100				58		23	66	57	304
1399 HERPES VIRUS TYPING PENDING.....					4	4	3			11
1401 COXIELLA BURNETI.....	1							4		5
1502 PICORNA VIRUS-NOT TYPED.....	7	2		8						17
1521 MEASLES VIRUS.....					1	1			3	5
1522 RUBELLA VIRUS.....					1		3	6	1	11
1532 HEPATITIS B ANTIGEN.....	65			12	32		6	16	13	144
1535 HEPATITIS A ANTIBODY.....				2	4	1	5	3	5	20
1541 CHLAMYDIA A - C TRACHOMATIS.....	29			8			1	34	58	130
1543 CHLAMYDIA A - LGV TYPE.....									1	1
1556 CMV - CYTOMEGALOVIRUS.....	3			2	14	1		5	8	33
1564 ROTAVIRUS.....		3		5	6	13	5			32
1599 ENTEROVIRUS TYPING PENDING.....				6		12	1	1		20
9901 ARBO. GROUP A.(UNSPECIFIED).....					21		47			68
9902 POXVIRUS GROUP NOT TYPED.....									1	1
9992 ROSS RIVER VIRUS.....				7			1	148	13	169
9993 ASTROVIRUS.....								1		1
9994 SMALL VIRUS (LIKE) PARTICLE.....		1								1
9997 KUNJIN VIRUS.....								1		1
Total.....	292	14	70	207	94	178	358	204		1,417

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 16, 2, 84 to 29, 2, 84

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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	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ muc memb
0100 ADENOVIRUS NOT TYPED.....		1									
0101 ADENOVIRUS TYPE 1.....		1									
0102 ADENOVIRUS TYPE 2.....	1	3		1			3				
0103 ADENOVIRUS TYPE 3.....		3					1				1
0104 ADENOVIRUS TYPE 4.....	1										
0105 ADENOVIRUS TYPE 5.....		3				1	1				
0106 ADENOVIRUS TYPE 6.....							2				
0118 ADENOVIRUS TYPE 18.....		1					1				
0131 ADENOVIRUS TYPE 31.....							1				
0201 INFLUENZA A VIRUS.....		2									
0301 PARAINFLUENZA VIRUS TYPE 1....		8									
0302 PARAINFLUENZA VIRUS TYPE 2....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	15							1		1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		2									
0500 RHINOVIRUS (ALL TYPES).....		21				1	1				2
0600 MYCOPLASMA PNEUMONIAE.....	12	66				1			1		2
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0802 COXSACKIEVIRUS A2.....							1				
0809 COXSACKIEVIRUS A9.....				1							
0902 COXSACKIEVIRUS B2.....							1				
0903 COXSACKIEVIRUS B3.....		1									
0905 COXSACKIEVIRUS B5.....				1							
1004 ECHOVIRUS TYPE 4.....							1				
1006 ECHOVIRUS TYPE 6.....							1				
1009 ECHOVIRUS TYPE 9.....		2			3		2				
1014 ECHOVIRUS TYPE 14.....	1	2					1				
1017 ECHOVIRUS TYPE 17.....		1		1				1			
1022 ECHOVIRUS TYPE 22.....		1									
1101 POLIOVIRUS TYPE 1.....	2						1				
1102 POLIOVIRUS TYPE 2.....	2	1									
1103 POLIOVIRUS TYPE 3.....	3										
1104 POLIOVIRUS-VACCINAL STRAIN....		1					3				
1200 MUMPS VIRUS.....	3			1							
1300 HERPES VIRUS GROUP-NOT TYPED..			1								
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1								1	4
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..	5	2									1
1303 VARICELLA-ZOSTER VIRUS.....											5
1306 HERPES SIMPLEX TYPE 1.....	4	2						1		1	49
1307 HERPES SIMPLEX TYPE 2.....	9				1						55
1401 COXIELLA BURNETI.....	1	1									
1502 PICORNA VIRUS-NOT TYPED.....			1						1		
1521 MEASLES VIRUS.....		1									5
1522 RUBELLA VIRUS.....	1	1									8
1532 HEPATITIS B ANTIGEN.....	91	2					2	46			1
1535 HEPATITIS A ANTIBODY.....	7					1		13			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	1									
1556 CMV - CYTOMEGALOVIRUS.....	4	2		1	1	2				2	
1564 ROTAVIRUS.....							31				
1599 ENTEROVIRUS TYPING PENDING....							1				
9901 ARBO. GROUP A.(UNSPECIFIED)...	7										30
9902 POXVIRUS GROUP NOT TYPED.....											1
9992 ROSS RIVER VIRUS.....	55										44
9993 ASTROVIRUS.....											1
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
9997 KUNJIN VIRUS.....											1
Total.....	211	150	2	10	1	6	56	61	3	4	211

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 16/2/84 to 29/2/84 ...

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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	Gen-ital	Endo/sal gland	RES	Muscle/joint	Con-genital	PUO	Fever/mal-aise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....							1			
0102 ADENOVIRUS TYPE 2.....							1			
0103 ADENOVIRUS TYPE 3.....	2						1			
0108 ADENOVIRUS TYPE 8.....	2									
0111 ADENOVIRUS TYPE 11.....									1	
0119 ADENOVIRUS TYPE 19.....	5	1								
0201 INFLUENZA A VIRUS.....							1			
0500 RHINOVIRUS (ALL TYPES).....				2			3	1		1
0600 MYCOPLASMA PNEUMONIAE.....					1		1	5		
0802 COXSACKIEVIRUS A2.....								1		1
1009 ECHOVIRUS TYPE 9.....							1			
1104 POLIOVIRUS-VACCINAL STRAIN....							1			
1200 MUMPS VIRUS.....				4						
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1						1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..				7				3	1	
1303 VARICELLA-ZOSTER VIRUS.....	1								1	
1306 HERPES SIMPLEX TYPE 1.....	5	44				1		2	8	
1307 HERPES SIMPLEX TYPE 2.....		241						2	1	
1399 HERPES VIRUS TYPING PENDING...		1								
1401 COXIELLA BURNETI.....								4		
1522 RUBELLA VIRUS.....					2			1		
1532 HEPATITIS B ANTIGEN.....					2			1	3	
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	126								
1543 CHLAMYDIA A - LGV TYPE.....		1								
1556 CMV - CYTOMEGALOVIRUS.....		8		1		2	2	1	9	
1564 ROTAVIRUS.....									1	
9901 ARBO. GROUP A.(UNSPECIFIED)...					35	8		2		
9992 ROSS RIVER VIRUS.....					98			3		
9997 KUNJIN VIRUS.....					1					
Total.....	18	423	14		139	11	12	27	25	2