ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 84/22
Issue date: 2 November 1984

Contents:

VIRUS REPORTING SCHEME - A total of 1508 reports were processed this period. The seasonal decline in respiratory tract infections (276 reports compared with 310, 458 and 526 for the previous three periods) is primarily due to the decrease in respiratory syncytial virus reports (57 compared with 74, 185 and 268), with no significant abatement in influenza, parainfluenza and rhinovirus infections. The reports also indicate the progressive reduction of rotavirus infections but the continued increased incidence of rubella.

. IgA antibody to the viral capsid antigen (VCA) of Epstein-Barr (EB) virus was detected by immunofluoresence in a 57 year old Chinese male with nasopharyngeal carcinoma (NPC). Undifferentiated NPC is rare in most parts of the world, but has a very high incidence among southern Chinese and Eskimos. In addition to the racial predisposition, a weak genetic influence has been described, and there is also good epidemiological evidence incriminating environmental factors. One hundred per cent of undifferentiated NPC carry EB virus DNA in the malignant cells, and these epithelial tumour cells provide the only example apart from B lymphocytes of cells naturally infected with EB virus (Clin. Exp. Immunol. (1983) 53: 257).

. Encephalitis - Two unusual encephalitis cases were reported this period. A CF titre of $\geq$ 1/320 against the 229E strain of coronavirus was detected at the State Health Laboratory Services, Perth, in an 11 year old boy who presented with meningoencephalitis two months previously. Since the consistent and intrinsic feature of human coronaviruses has been the difficulty of recovering them from clinical specimens in vitro, the true picture of their importance in human disease has yet to be delineated. In addition, the State Health Laboratory, Brisbane, reported the detection of IgM antibody to Ross River virus in a male with encephalitis.

(continued from page 5)

References

1. "Rapid laboratory techniques for the diagnosis of viral infections" (1981). Report of a Scientific Group. Technical Report Series, No. 661, WHO, Geneva.
2. PHLS Microbiology Digest (1984) 1 (4) : 44.

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GONOCOCCAL SURVEILLANCE - AUSTRALIA (JANUARY - JUNE 1984)

(Contributed by the Australian Gonococcal Surveillance Program (AGSP). Co-ordinator - J.W. Tapsall, Department of Microbiology, Prince of Wales Hospital, Sydney).

The AGSP has collated and reported the penicillin sensitivities of Neisseria gonorrhoeae isolates since July 1981(1). This report describes the patterns for the period January-June 1984. As in previous reports, the results are recorded quarterly and by region, with Table 1 giving the percentages of gonococcal isolates classified as either (A) sensitive (minimal inhibitory concentration (MIC) value = 0.008 µg/ml) or (B) less sensitive (MIC = 0.12 µg/ml) to penicillin.

TABLE 1. Penicillin sensitivity of N. gonorrhoeae isolates (January-June 1984)

Percentages for the corresponding January-June 1983 quarters are in parentheses

Centre	Percentage of isolates			
	January - March, 1984		April - June, 1984	
	A	B	A	B
Brisbane	34.5 (39.5)	53.0 (48.3)	35.0 (38.0)	53.0 (49.0)
Sydney	15.0 (22.5)	55.0 (68.4)	13.0 (19.0)	64.0 (67.8)
Melbourne	12.0 (48.0)	46.0 (46.0)	27.0 (31.0)	46.0 (53.0)
Adelaide	39.3 (45.0)	48.3 (40.0)	27.0 (39.0)	59.3 (50.0)
Perth	27.0 (37.0)	35.0 (37.8)	23.0 (45.0)	52.0 (38.0)
Strains examined	1363 (1771)		1250 (1204)	

In all centres, strains with a reduced sensitivity to penicillin predominated. Additional data, not shown in Table 1 was also received from other laboratories, with gonococci isolated in lower numbers in Hobart, Canberra and Darwin. Of the MIC determinations performed on the 109 strains from Darwin during the period January-June 1984, 45.8% were fully sensitive and 35.7% showed a decreased sensitivity. Of the 73 strains isolated over the eighteen months to June 1984 in Canberra, 18% were fully sensitive and 55% were less sensitive. The pattern for Hobart was as for the previous reports, with the strains belonging to the sensitive category.

However, the increase both in absolute and percentage terms of penicillinase - producing N. gonorrhoeae (PPNG) strains is of considerable interest and concern (Table 2).

TABLE 2. Percentage of PPNG strains isolated, January-June 1984

Percentages for the corresponding January - June 1983 quarters are in parentheses.

Centre	Percentage of isolates	
	January - March, 1984	April - June, 1984
Brisbane	8.7 (5.7)	4.4 (6.5)
Sydney	18.1 (3.9)	14.4 (6.5)
Melbourne	5.1 (3.7)	2.8 (2.5)
Adelaide	5.2 (4.7)	5.1 (2.2)
Perth	6.0 (5.2)	6.5 (5.8)
PPNG strains (Australia)	131 (81)	85 (50)

Of the 131 and 85 PPNG strains isolated in the two quarters, 57 and 44 respectively were reported from Sydney. Since the AGSP

began collating data three years ago, seasonal variations have been noted, but it has also been evident that has been a steady increase in the number of PPNG strains isolated. In the last quarter of 1983 (see CDI 84/13), a marked increase in PPNG was recorded in Sydney, and attributed to an outbreak among prostitutes. In the two quarters reviewed here, the seasonal decline in the number of PPNG strains seen previously did not occur, and even higher isolation rates were recorded. Investigation has shown that sustained domestic transmission, rather than importation from overseas, is now the principal source of PPNG infection in Sydney. Of the 101 strains isolated in the reported six month period, three infections were acquired in Thailand, three in Manila, and three were unknown. The remainder were locally acquired. However, in the other centres the numbers of PPNG infections acquired locally, although increasing, were still in the minority. PPNG strains were also detected in Hobart, Canberra and Darwin.

Editorial Comment

Although foreign importation still contributes significantly to PPNG infections in Australia, the current proportion of cases linked to domestic transmission in Sydney is cause for concern. The incidence of PPNG strains has been increasing steadily since their emergence in 1976(2), with annual totals of 144, 177, 215 and 220 respectively for the period 1980-1983. Equivalent figures for the years 1982 and 1983 for the UK are 1033 and 1223, and for the USA are 4457 and 3720. Temporal and regional variation in their isolation rates have been noted, and evidence has also been accumulating that increasing numbers had undergone sustained domestic transmission(1).

Domestic PPNG cases usually appear among two population groups that have traditionally had a high prevalence of gonococcal infection; prostitutes and the urban poor(3). Prostitutes habitually have poor communication with the medical profession and usually conceal their occupation, so that public education programs have little permanent effect(4). Of the 70 prostitutes of a Sydney house of prostitution screened on a weekly basis between June 1980 - June 1981, 10% acquired new infections with gonorrhoea each week (53 episodes)(5). Clinical guidelines (symptoms, contact history, physical signs) were found to be unreliable in diagnosis, and regular, rapid and accurate pathology testing was essential. During this study, no PPNG strains were detected, but using a conservative transmission rate of 22%(6), it can be seen that infected women in this environment are major factors in the hyperendemicity of gonorrhoea. It has been suggested that working in cooperation with sympathetic management at the place of work, rather than attempting to police individual itinerant women, may be a more effective way of minimizing the impact of STD's on prostitutes, their clients and the general population(4,5).

Spectinomycin is highly effective and widely used against PPNG, although strains of spectinomycin-resistant PPNG and spectinomycin-resistant non-PPNG have been reported(7,8). Since spectinomycin resistance may develop even during one single dose therapy, health personnel should be aware that not all patients treated with spectinomycin will be cured of gonorrhoea, and that post-treatment cultures should be an integral part of patient management. The recommended 2 gm dose of spectinomycin IM should be adhered to, since there is no additional benefit prescribing higher dosage.

References

1. Br. J. Vener. Dis. (1984) 60 : 226
2. CDI (1982) 82/3 : 2
3. NEJM (1982) 307 : 438
4. MJA (1984) 140 : 272
5. MJA (1984) 140 : 268
6. Am. J. Epidemiol (1970) 91 : 170
7. WER (1983) 58 : 136
8. BMJ (1983) 287 : 1827

CONCURRENT VIRAL AND BACTERIAL MENINGITIS - UNITED KINGDOM

(Based on CDR (1984) 84/40 : 3).

A three year old girl was referred by her general practitioner with a 24 hour history of vomiting, fever and irritability, followed by increasing drowsiness and a grand mal convulsion lasting ten minutes. On arrival in hospital she was semi-conscious, poorly perfused, febrile (37.7°C) with marked neck stiffness and opisthotonic posture, but no focal neurological signs. Examination of the cerebral spinal fluid (CSF) showed 3,812 x 10^6 leucocytes/L (95% polymorphs), 916 x 10^6 red cells/L, protein of 0.95 g/L, glucose of 0.2 mmol/L and lactate of 8.35 mmol/L (upper limit of normal 2.87 mmol/L). A stained smear revealed numerous pleomorphic Gram-negative rods. The patient was treated with intravenous chloramphenicol 100 mg/kg/day 6 hourly intravenously. Haemophilus influenzae type b, β-lactamase negative, was isolated from blood cultures and CSF within 24 hours.

The fever settled within 13 hours of admission, but the patient remained irritable, with persistent opisthotonus, neck stiffness and intermittent drowsiness for over 48 hours. In view of the poor clinical response to treatment a repeat lumbar puncture was performed 48 hours after admission. Examination of this second CSF showed 10,600 x 10^6 leucocytes/L (90% polymorphs), 1,200 x 10^6 red cells/L, protein of 0.98 g/L and glucose of 2.1 mmol/L. A Gram-stained smear showed no organisms and culture for bacteria was negative. However, this specimen yielded echovirus type 25 in tissue culture. There was a recurrence of fever to 38.4°C on day 3 and this persisted for 48 hours. Following this the fever settled and the patient improved clinically. Chloramphenicol was continued for a total of 12 days and the patient made an uneventful recovery.

The first publication of concurrent viral and bacterial meningitis involved H. influenzae type b and echovirus type 9(1). Two cases of simultaneous meningococcal and enteroviral infection of the CSF were subsequently reported(2,3). Although viral culture of the first CSF from this patient was not attempted, isolation from a second specimen taken just 48 hours later strongly implies that this was a further case of simultaneous bacterial and viral meningitis. Prospective studies of the aetiology of meningitis suggest that such dual infections are extremely rare(1,3). Nevertheless, the occurrence of secondary fever during the course of treated bacterial meningitis is common and its aetiology often remains uncertain(4). In this child, persisting drowsiness prompted re-examination of the CSF, an investigation which provided an unexpected, but rewarding explanation for the patient's clinical course.

References

1. NEJM (1962) 267 : 142
2. Lancet (1976) 2 : 1412
3. Lancet (1977) 1 : 371
4. J. Pediatr. (1979) 77 : 957

FORMATION OF THE ASIAN GROUP FOR RAPID VIRAL DIAGNOSIS CDI 84/22

(Contributed by S.K. Lam, Department of Medical Microbiology, University of Malaya, Kuala Lumpur, Malaysia).

During the Sixth International Congress of Virology held in Sendai, Japan, 1-7 September 1984, 44 participants met to form the Asian Group for Rapid Viral Diagnosis (ASGRVD). The aims of the Group are to:

- Promote the use of rapid and simple diagnostic procedures for clinical purposes, especially in developing countries in the South East Asia and the Western Pacific
- Encourage the development of new techniques, especially for viral diseases of importance to these two Regions
- Organise and promote training programs
- Hold scientific meetings and symposia on this subject
- Promote the production of good quality reagents and the distribution of these to participating laboratories
- Disseminate information on this subject to members
- Collaborate with other organisations such as the World Health Organisation, and the European and Pan-American Groups for Rapid Viral Diagnosis in this and related fields.

Membership at US $10 per annum is open to any scientist who is interested and/or actively engaged in rapid viral diagnosis in South-East Asia and the Western Pacific. Meetings of the membership will occur at least once every two years and will be timed to coincide with scientific meetings organised by the Committee or of other related organisations. All enquiries, as well as application for membership, should be addressed to:-

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Department of Medical Microbiology
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Editorial Comment

Methods of aetiological diagnosis of viral infections now fall into two categories, conventional virological methods such as cell culture, and the newly evolved rapid laboratory methods. The latter are based on the detection of antigen without the cultivation of the viruses and/or the detection of IgM antibodies without using the time-consuming classical examination of paired sera. However, such techniques must offer tangible advantages to influence the management of a patient or a community as well as being easy to perform by junior staff without elaborate quality assessment and sophisticated equipment.

Following the rationalisation of these methods, WHO has been able to recommend specific techniques for particular infections(1). These include immunofluorescence for respiratory tract infections, electronmicroscopy and immunofluorescence for skin infections and infections of the CNS, and enzyme-linked immunosorbent assay (ELISA) for the investigation of rotaviruses in faeces. The ELISA technique using an anti-mu (IgG antibody raised in rabbits or swine to human mu chains) coated carrier for trapping the IgM also appears to be the current method of choice for detecting specific IgM. However, the production of monoclonal antibodies may further improve both the duration and specificity of these techniques. Future diagnostic microbiology may also be revolutionised by the development of nucleic acid hybridisation techniques using specific recombinant DNA probes(2). Consequently, such proposed liaison between fellow scientists practising methods of rapid viral diagnosis is essential for the use, promotion and quality control of these novel techniques.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 11/10/84 - 24/10/84 BULLETIN NUMBER 84/22

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			6		8		26		40
0101 ADENOVIRUS TYPE 1	4	1			1	3		4	13
0102 ADENOVIRUS TYPE 2	3	1		1	8			4	17
0103 ADENOVIRUS TYPE 3				1	2		1	3	7
0104 ADENOVIRUS TYPE 4		1							1
0105 ADENOVIRUS TYPE 5			1						1
0106 ADENOVIRUS TYPE 6					2				2
0107 ADENOVIRUS TYPE 7	1	1			2				4
0108 ADENOVIRUS TYPE 8	1							2	3
0111 ADENOVIRUS TYPE 11				1					1
0119 ADENOVIRUS TYPE 19	1				1				2
0137 ADENOVIRUS TYPE 37								1	1
0199 ADENOVIRUS TYPING PENDING		1	2		8	1			12
0201 INFLUENZA A VIRUS			3	1		19	1	6	30
0202 INFLUENZA A VIRUS SUBTYPE H3N2		1		4	3				8
0203 INFLUENZA B VIRUS	19		7	1		2	12	2	43
0301 PARAINFLUENZA VIRUS TYPE 1						4	1		5
0302 PARAINFLUENZA VIRUS TYPE 2						4	1		5
0303 PARAINFLUENZA VIRUS TYPE 3	1				6	10	4	7	28
0304 PARAINFLUENZA VIRUS TYPE 4								3	3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	8	6		5	6	13	17	2	57
0500 RHINOVIRUS (ALL TYPES)			1	8	13	7	6	6	41
0600 MYCOPLASMA PNEUMONIAE	3		3			2	2	3	13
0700 ORNITHOSIS-PSITTACOSIS	1							2	3
0809 COXSACKIEVIRUS A9				1	1				2
0904 COXSACKIEVIRUS B4								1	1
0905 COXSACKIEVIRUS B5						3	1		4
1000 ECHOVIRUS NOT TYPED							1		1
1001 ECHOVIRUS TYPE 1	1								1
1006 ECHOVIRUS TYPE 6				1	1			1	3
1009 ECHOVIRUS TYPE 9	2								2
1011 ECHOVIRUS TYPE 11					1				1
1012 ECHOVIRUS TYPE 12	1								1
1022 ECHOVIRUS TYPE 22		1							1
1024 ECHOVIRUS TYPE 24					3			1	4
1029 ECHOVIRUS TYPE 29			1						1
1100 POLIOVIRUS NOT TYPED			2		2				4
1101 POLIOVIRUS TYPE 1			1				2	1	4
1102 POLIOVIRUS TYPE 2	2						1	2	5
1103 POLIOVIRUS TYPE 3								2	2
1200 MUMPS VIRUS	3						2	1	6
1300 HERPES VIRUS GROUP-NOT TYPED	23		5	6			3	1	38
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1		1				1	3
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5	1				3		11	20
1303 VARICELLA-ZOSTER VIRUS	5		2	1	1		1	2	12
1306 HERPES SIMPLEX TYPE 1	17			25	25	10	57	23	157
1307 HERPES SIMPLEX TYPE 2	141			69		21	109	53	393
1399 HERPES VIRUS TYPING PENDING					3	1			4
1401 COXIELLA BURNETI	1					1	10	3	15
1502 PICORNA VIRUS-NOT TYPED			4					1	5
1521 MEASLES VIRUS	1	1	1			1		6	10
1522 RUBELLA VIRUS	22		9			8	3	15	57
1532 HEPATITIS B ANTIGEN	61		4	18	1	17	16	10	127
1535 HEPATITIS A ANTIBODY	4		1	9		4	2	6	26
1541 CHLAMYDIA A - C TRACHOMATIS	33		7				45	41	126
1556 CMV - CYTOMEGALOVIRUS	9	2	4	32	7	2	15	12	83
1563 CORONAVIRUS								2	2
1564 ROTAVIRUS		4	10	1	2	7	2		26
1599 ENTEROVIRUS TYPING PENDING		1	3		5				9
9902 POXVIRUS GROUP NOT TYPED						1			1
9992 ROSS RIVER VIRUS							7		7
9993 ASTROVIRUS		1					1		2
9994 SMALL VIRUS (LIKE) PARTICLE				1					1
Total	373	24	77	187	112	144	350	241	1,508

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 11/10/84 to 24/10/84 .... 84/22

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/ mucs memb
0100 ADENOVIRUS NOT TYPED		1									
0101 ADENOVIRUS TYPE 1	1	9					2				1
0102 ADENOVIRUS TYPE 2		10					4				
0103 ADENOVIRUS TYPE 3		5									1
0104 ADENOVIRUS TYPE 4							1				
0105 ADENOVIRUS TYPE 5							1				
0106 ADENOVIRUS TYPE 6		1					1				
0107 ADENOVIRUS TYPE 7		2					3				
0119 ADENOVIRUS TYPE 19		1					1				
0201 INFLUENZA A VIRUS	5	17		1					1		1
0202 INFLUENZA A VIRUS SUBTYPE H3N2		7									
0203 INFLUENZA B VIRUS	4	26				1			1		1
0301 PARAINFLUENZA VIRUS TYPE 1	1	4									
0302 PARAINFLUENZA VIRUS TYPE 2	1	4									
0303 PARAINFLUENZA VIRUS TYPE 3	1	22					1			1	
0304 PARAINFLUENZA VIRUS TYPE 4		2									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1	53									4
0500 RHINOVIRUS (ALL TYPES)		41					3				
0600 MYCOPLASMA PNEUMONIAE		12									
0700 ORNITHOSIS-PSITTACOSIS		2									
0809 COXSACKIEVIRUS A9		1									1
0904 COXSACKIEVIRUS B4							1				
0905 COXSACKIEVIRUS B5		1					2				
1001 ECHOVIRUS TYPE 1		1									
1006 ECHOVIRUS TYPE 6		3		1							
1009 ECHOVIRUS TYPE 9	1	1									
1011 ECHOVIRUS TYPE 11		1									
1022 ECHOVIRUS TYPE 22							1				
1024 ECHOVIRUS TYPE 24		1		2							
1100 POLIOVIRUS NOT TYPED							2				
1101 POLIOVIRUS TYPE 1		3					1				
1102 POLIOVIRUS TYPE 2							3				
1200 MUMPS VIRUS	1			1							1
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1						2			1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	3	1	1	1				1			
1303 VARICELLA-ZOSTER VIRUS	1	1					1				8
1306 HERPES SIMPLEX TYPE 1	6	15	1	2				1		1	71
1307 HERPES SIMPLEX TYPE 2	10										58
1401 COXIELLA BURNETI	5	2		1							
1521 MEASLES VIRUS		4	1	1							6
1522 RUBELLA VIRUS	15										28
1532 HEPATITIS B ANTIGEN	69							41		1	
1535 HEPATITIS A ANTIBODY	3						1	21			
1541 CHLAMYDIA A - C.TRACHOMATIS	2	3									
1556 CMV - CYTOMEGALOVIRUS	11	17	1		2			2		11	1
1563 CORONAVIRUS		1	1	1							
1564 ROTAVIRUS							26				
9992 ROSS RIVER VIRUS			1								3
9993 ASTROVIRUS							1				1
9994 SMALL VIRUS (LIKE) PARTICLE							1				
Total	141	276	6	11	2	1	57	68	2	14	188

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 11/10/84 to 24/10/84 ... 84/22

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
0100 ADENOVIRUS NOT TYPED.....	1									
0101 ADENOVIRUS TYPE 1.....								1	2	
0102 ADENOVIRUS TYPE 2.....							1		1	1
0103 ADENOVIRUS TYPE 3.....	1								1	
0108 ADENOVIRUS TYPE 8.....	2	1								
0111 ADENOVIRUS TYPE 11.....									1	
0119 ADENOVIRUS TYPE 19.....	1									
0137 ADENOVIRUS TYPE 37.....		1								
0201 INFLUENZA A VIRUS.....					1		1	5	1	1
0202 INFLUENZA A VIRUS SUBTYPE H3N2							1	3		
0203 INFLUENZA B VIRUS.....				1			4	7	2	
0303 PARAINFLUENZA VIRUS TYPE 3....					1		3			
0304 PARAINFLUENZA VIRUS TYPE 4....								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....							1	1		
0500 RHINOVIRUS (ALL TYPES).....									1	
0600 MYCOPLASMA PNEUMONIAE.....					1					
0700 ORNITHOSIS-PSITTACOSIS.....								1		
0809 COXSACKIEVIRUS A9.....							1	1		
0905 COXSACKIEVIRUS B5.....										1
1001 ECHOVIRUS TYPE 1.....	1									
1012 ECHOVIRUS TYPE 12.....										1
1024 ECHOVIRUS TYPE 24.....							2			
1029 ECHOVIRUS TYPE 29.....								1		
1102 POLIOVIRUS TYPE 2.....								2		
1103 POLIOVIRUS TYPE 3.....										2
1200 MUMPS VIRUS.....				2				2		
1301 HERPES SIMPLEX VIRUS NOT-TYPED					1					
1302 EPSTEIN-BARR VIRUS (EB VIRUS).				9				2	5	
1303 VARICELLA-ZOSTER VIRUS.....	1	1							1	
1306 HERPES SIMPLEX TYPE 1.....	6	51		1			1	3	3	
1307 HERPES SIMPLEX TYPE 2.....	1	322				1			1	
1401 COXIELLA BURNETI.....					2		2	7		
1521 MEASLES VIRUS.....				1						
1522 RUBELLA VIRUS.....				4	1	8		4	7	
1532 HEPATITIS B ANTIGEN.....									16	
1535 HEPATITIS A ANTIBODY.....								1		
1541 CHLAMYDIA A - C.TRACHOMATIS...		122								
1556 CMV - CYTOMEGALOVIRUS.....		9		2	2		3	2	8	17	2
9992 ROSS RIVER VIRUS.....						5			2		
9993 ASTROVIRUS.....						1				
Total.....	14	507		20	5	18	4	19	53	59	8