



Communicable Diseases Intelligence

Bulletin number 80/19
Issue date: 26 September 1980

Contents:

Infectious gastroenteritis in Norfolk Island
Chlamydia trachomatis infections
South African Tick Typhus
Hand, Foot and Mouth Disease - U.K.

TYPHOID FEVER - QUEENSLAND - Salmonella typhi has been confirmed in a 27 year old man at the Royal Brisbane Hospital. The patient who had not travelled outside the Brisbane area, had an episode of diarrhoea in late August. He later became febrile and was hospitalised undiagnosed on 17 September. This still apparently isolated case is being investigated for possible sources of infection by the Queensland Department of Health.

VIRUS REPORTING SCHEME - A total of 1127 reports were received this period. General patterns as suggested by the reports received include an increase in the number of influenza B virus infections - 32 reports received compared to 13, 12 and 8 for the previous three periods, and the continuation of the rise in the number of reports of rotavirus infection - 154 reports received compared to 118, 116 and 103 for the previous three periods.

Reports of interest include:

- Influenza - The State Health Laboratory, Brisbane, reported four isolations of influenza type A virus and three isolations of influenza type B virus. Three of the type A isolates appeared to resemble bridging strains between A/Bangkok/1/79 and A/Texas/1/77, and have been referred to the WHO Influenza Centre for elucidation. The Institute of Clinical Pathology and Medical Research, Sydney, reported one isolation of influenza type A virus resembling A/Texas/1/77.

During August the WHO Influenza Centre, Commonwealth Serum Laboratories, Melbourne, identified six influenza type A isolates as resembling A/Bangkok/1/79, and one influenza type B isolate as resembling B/Singapore/222/79.

- Coxsackie A16 virus - Four cases of hand, foot and mouth disease were reported for this period, three from the State Health Laboratory, Brisbane, and one from Fairfield Hospital, Melbourne. (See also note on this disease on page 6.)
- Q fever - Fairfield Hospital, Melbourne, reported a serological response by CF to C. burneti in a 20 year old male employed in a laundry that services an abattoir.

INFECTIOUS GASTROENTERITIS IN NORFOLK ISLAND

(Contributed by M.F.H. Sexton, Government Medical Officer, Norfolk Island; and G.S. Grohmann and A.M. Murphy, Institute of Clinical Pathology and Medical Research, Sydney.)

During 1978, Norfolk Island (located in the South Pacific, 1500 kilometers from Sydney and 1000 kilometers from Auckland, resident population 1900, tourists in excess of 20000 annually) reported a relatively high incidence of presumed infectious diarrhoea.

<u>Diarrhoea</u>	<u>Rate/1000 population</u>
Infantile	225
Child/adult	384

Since the Pitcairn settlement on Norfolk Island, an infectious disease, known locally as "the sick and vomits", has occurred both endemically and in epidemics, but except for one case of giardiasis, no causative organism has ever been identified. The increasing incidence of the disease appears to parallel contamination of deep groundwater by effluents. There are some 18 water bores on the island and six effluent discharge points.

In a new attempt to identify the aetiological agent(s), faecal samples were forwarded to I.C.P.M.R., Sydney, for isolation and electron microscopy (E.M.). As with previous samples, no aetiological agents were isolated. However, E.M. studies provided 11 positive viral identifications from 13 specimens. The results are tabulated below:

	<u>EM Observations</u>					<u>Total</u>
	<u>Rota-virus</u>	<u>Astro-virus</u>	<u>Calici-virus</u>	<u>Small Round Virus (22-25nm)</u>	<u>No Viruses</u>	
Adults (21-75 years)	-	1	1	2	1	5
Children (2-8 years)	<u>1</u>	<u>4</u>	<u>2</u>	<u>-</u>	<u>1</u>	<u>8</u>
Totals	<u>1</u>	<u>5</u>	<u>3</u>	<u>2</u>	<u>2</u>	<u>13</u>

The association of these viruses with cases of gastroenteritis linked to contaminated groundwater is being investigated further by collecting more faecal specimens and groundwater samples, and by screening paired sera from gastroenteritis cases.

Editorial Comment

Electron microscopic examination of negatively stained faecal preparations from patients with gastroenteritis has resulted in the discovery of many viruses, some of which, for example, rotaviruses and the Norwalk agent

(parvovirus-like virus) undoubtedly cause of acute gastroenteritis. Other viruses, such as astroviruses, caliciviruses, adenoviruses and coronaviruses are implicated in gastrointestinal infections, although their role as causative agents is still tenuous⁽¹⁾. In general, in vitro cultivation of the viral agents is either not possible or extremely difficult.

The significance of gastroenteritis as a water-borne infection is also difficult to quantitate⁽²⁾, as little work has been done on the minimal effective doses of a virulent virus for a non-immune human. Even though, on average, large doses would be required, there is some evidence to suggest that for some viruses the minimum infective dose for a susceptible host may be as low as one infectious particle. This indicates that drinking water ideally should be free from viruses. Other workers have postulated that small amounts of virus in drinking water may provide an immunising dose, although such uncontrolled administration of a non-attenuated vaccine would never be advocated.

References

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CHLAMYDIA TRACHOMATIS INFECTIONS

The clinical spectrum of human infection with Chlamydia trachomatis is rapidly expanding. In 1979, the CDI received 782 reports of C. trachomatis infections. For the first six months of 1980, 722 reports were received, of these 235 recorded genital symptoms, 19 were from eye infections, and no clinical information was available for 462 reports.

However, these statistics are not indicative of the national figures, since only certain laboratories regularly receive and diagnose chlamydia infections (the State Health Laboratory, Perth, reported 77% of the chlamydia infections for 1979).

The genus Chlamydia is regarded as a group of related agents, and is placed between bacteria and viruses with respect to properties and characteristics. The organisms have been known by a variety of other names in the past; bedsonia, miyagawanella and "TRIC-agent" (an acronym for trachoma-inclusion conjunctivitis agent). There are two species; C. psittaci (subgroup B) which contains the agents of the zoonoses psittacosis and ornithosis; C. trachomatis (subgroup A) comprising the organisms responsible for trachoma, inclusion conjunctivitis, genital tract infections and lymphogranuloma venereum. With the exception of a few strains causing rodent pneumonitis, the C. trachomatis strains are human pathogens with man as the sole natural host. C. trachomatis species are differentiated from C. psittaci on the basis of the inclusion type. C. trachomatis inclusions stain with iodine, and C. psittaci inclusions do not. In addition, C. trachomatis is sensitive to sulphonamides, and C. psittaci is resistant. Both species have a complex intracellular replication cycle terminating in binary fission.

The strains of C. trachomatis are all related antigenically. There are

three serotypes of lymphogranuloma venereum agent (L-1, L-2, and L-3) and 12 of trachoma-inclusion conjunctivitis agent (A, B, Ba, C, D, E, F, G, H, I, J, and K).

C. trachomatis was first associated with human disease in 1907 when it was described as producing inclusion bodies in the conjunctivae of patients with trachoma⁽¹⁾. It was not until 1957 that the organism was cultured in embryonated eggs⁽²⁾, and not until 1965 that it was first isolated in tissue culture⁽³⁾. Since then C. trachomatis has been implicated in further diseases⁽⁴⁾, ocular and genital tract infections being the most widely recognized.

The serotypes D to K predominantly produce genitourinary disease, and are sexually transmitted. In the male, 20-50% of non-gonococcal urethritis is thought to be a result of C. trachomatis infection, and may present with dysuria with or without mucoid discharge, and may cause epididymitis and prostatitis⁽⁵⁾. Ureaplasma urealyticum appears to be responsible for the major proportion of the remaining C. trachomatis negative non-gonococcal urethritis infections⁽⁶⁾. Although C. trachomatis can cause cervicitis in females, a substantial proportion of chlamydial infections in the cervix are not only asymptomatic but may also be inapparent. However, there is still an unquantified risk of acute salpingitis⁽⁷⁾ and pelvic inflammatory disease, as well as a recent suggested aetiological role for C. trachomatis in peritonitis and perihepatitis in young women⁽⁸⁾. Babies born through a chlamydia-infected birth canal may become infected.

An acute inclusion conjunctivitis of the newborn (inclusion blennorrhoea) has been recognised for over seventy years. The incubation period is usually five to 14 days; earlier onset usually follows premature rupture of the membranes. The course of this acute mucopurulent conjunctivitis is generally benign, and it tends to resolve spontaneously several weeks to several months after onset. In severe cases, pseudomembranes may be formed in the conjunctivae, and scarring may result. Persistent infection can occur.

Estimates of incidence range from 1.4 to 4.4. cases per 1000 live births⁽⁹⁾. In a recent study from the Royal Maternity Hospital, Glasgow, 199 neonates with "sticky eyes" were examined. Seventy-six infections were found to be chlamydia positive, the remaining cases were probably caused by pyogenic bacteria⁽¹⁰⁾.

C. trachomatis can also be recovered from nasopharyngeal and tracheo-bronchial aspirates collected from infants with a distinct pneumonia syndrome⁽¹¹⁾. In a study in San Francisco, 20 infants born through chlamydia infected cervixes were investigated; seven developed conjunctivitis, four chlamydial pneumonia and a total of 14 exhibited a serological response⁽¹²⁾. Since C. trachomatis was isolated in 4% of pregnant women in that clinic, 2.8% of all newborns acquired chlamydial infection.

C. trachomatis respiratory tract involvement has also been reported in adults⁽¹³⁾, although immunosuppression appears to be a critical factor.

Whether these were primary infections or reactivation of latent infections has yet to be clarified.

C. trachomatis grows in the columnar epithelial cells of the conjunctiva, the cervix and the urethra, and respiratory tract. Since these cells are also present in the gastrointestinal tract and rectal mucosa, it could be considered that in appropriate epidemiological circumstances, C. trachomatis infection may present in any anatomic site containing such susceptible cells.

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SOUTH AFRICAN TICK TYPHUS

(Contributed by N.D. Stallman, State Health Laboratory, Brisbane.)

Sera was received from two patients who were members of a party of 25 tourists visiting the Eastern States of South Africa, Swaziland, Northern Botswana and Kenya in July 1980. Approximately one-third of the members became ill, the first patient showing symptoms in North Botswana. The main symptoms were severe headaches, stiff neck, fever and eschar. A rash was evident for some of the patients. None required hospitalisation and all were able to continue their trip.

The serum samples were forwarded to the Centre for Disease Control, Atlanta, for testing for rickettsioses. The results indicated an infection with a member of the spotted fever group, and were consistent with an infection with Rickettsia conori (South African Tick Typhus or Tick Bite Fever).

Editorial Comment

R. conori is responsible for a number of tick-borne rickettsial fevers, each having a different name depending on the geographic location - Boutonneuse fever, Marseilles fever, Kenya tick typhus, South African tick typhus and India tick typhus. As the names suggest, the disease is widely distributed throughout the African continent, in India and in those parts of Europe and the Middle East adjacent to the Mediterranean, Black and Caspian Seas. In more temperate areas, the highest incidence is during

warmer months when ticks are numerous; in tropical areas the disease occurs throughout the year. Outbreaks may occur when groups of susceptibles are brought into an endemic area.

In South Africa, the presumed vectors include the ectoparasites Haemaphysalis leachi, Amblyomma hebraeum, Rhipicephalus sanguineus, Boophilus decloratus and Hyalomma aegyptium. Infection in nature is maintained by transovarian and transstadial passage in the ticks. The zoonosis presents with a mild to moderately severe febrile illness of a few days to two weeks, characterised by a primary lesion at the site of the tick bite. This lesion progresses to a small ulcer (2-5mm in diameter) with a black centre and red areola. Regional lymph nodes become enlarged, and a maculopapular erythematous rash appears on day four or five, usually involving palms and soles, and persists to day six or seven. The fatality rate is less than 3% even without antibiotic therapy.

In Australia, Rickettsia australis is the aetiological agent for Queensland tick typhus. The vector is Ixodes holocyclus, an ectoparasite of wild rodents and marsupials. Although this is a mild disease without complications, personal prophylactic measures such as wearing protective clothing impregnated with a tick repellent (dimethylphthalate, dibutylphthalate or diethyltoluamide) should be considered when entering tick infected areas.

HAND, FOOT AND MOUTH DISEASE - U.K.

(Based on CDR 80/34 and CDS 80/25)

An epidemic of hand, foot and mouth disease is current in Britain. A total of 93 laboratory reports were collated for the first 33 weeks of 1980, although with such a benign disease, cases are often not investigated, and the number of laboratory reports is a poor measure of the extent of the disease.

The syndrome, mainly of children, is easily recognized and characterised by an ulcerative stomatitis, with a maculopapular rash on the dorsal aspects of the hands and sides of feet which becomes vesicular and lasts about ten days. In young children there may be a peri-anal rash and pyrexia is a common feature, and there may be a reluctance to feed because of the painful mouth ulcers. The illness is benign and usually uncomplicated. The commonest cause is Coxsackie A16 virus, but A5 and A10 have also been responsible for sporadic cases, and more recently Coxsackie B viruses have been associated with the syndrome.

A recent study at the Ruckhill Hospital, Glasgow, has suggested a rural preponderance for some of these cases, and when cases occurred in towns they were on the rural edges. For four of the eight outbreaks studied, the father's occupation was associated with animals. Since the infection spreads rapidly within family units, but often minimally in institutions, the possibility of an animal reservoir has been suggested. Although cattle may exhibit a low level of "antibody" to human enteroviruses, it has not been determined whether the reaction is due to non-specific inhibitors or cross reaction with other animal viruses.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

2

REPORTING PERIOD - 4-9-80 - 17-9-80 BULLETIN NUMBER - 80/19
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW) / WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	PAIR- FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
1030 ECHOVIRUS TYPE 30.....				3					3
1099 ECHOVIRUS TYPING PENDING.....			5						5
1101 POLIOVIRUS TYPE 1.....	2							1	3
1102 POLIOVIRUS TYPE 2.....						1	1		2
1103 POLIOVIRUS TYPE 3.....	1		2			2	1	1	7
1104 POLIOVIRUS-VACCINAL STRAIN.....					1				1
1200 MUMPS VIRUS.....	4			5	1	3			13
1300 HERPES VIRUS GROUP-NOT TYPED.....	9			3	1	2	1		16
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....	2			2			39	48	91
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	6					4			10
1303 VARICELLA-ZOSTER VIRUS.....	3		3			2	1		9
1306 HERPES SIMPLEX TYPE 1.....	2			25		9			36
1307 HERPES SIMPLEX TYPE 2.....	24			27		11			62
1399 HERPES VIRUS TYPING PENDING.....			8		3	3			14
1401 COXIELLA BURNETI.....	5			9		1	12		27
1502 PICORNA VIRUS-NOT TYPED.....								1	1
1514 MOLLUSCUM CONTAGIOSUM.....				1					1
1521 MEASLES VIRUS.....	2	3		1	1		1		8
1522 RUBELLA VIRUS.....	10		2	1			2	5	20
1530 HEPATITIS A VIRUS.....				1		16		15	32
1531 HEPATITIS B VIRUS.....				30		10	9	5	54
1532 HEPATITIS B ANTIGEN.....	19		4				1		24
1535 HEPATITIS A ANTIBODY.....	6			1					7
1541 CHLAMYDIA A - TRIC TYPE.....	5					1		35	41
1556 CMV - CYTOMEGALOVIRUS.....	5		10	10	3	4	7	4	43
1563 CORONAVIRUS.....				4					4
1564 ROTAVIRUS.....	55		32	21		22		28	158
1565 CALICI VIRUS.....	1								1
1599 ENTEROVIRUS TYPING PENDING.....					4	5			9
ROSS RIVER VIRUS							4		4
ASTROVIRUS	6								6
SMALL VIRUS (LIKE) PARTICLE	2			1		1			4
Total.....	227	28	88	179	89	194	118	204	1,127

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

3

PERIOD : 4/9/80 to 17/9/80 80/19
 Viral Identifications by Clinical Information Table 1.
 Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Enceph-
 alitis; 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.....	2	10				1	6				
0101 ADENOVIRUS TYPE 1.....		2				1					
0102 ADENOVIRUS TYPE 2.....	1	1									
0103 ADENOVIRUS TYPE 3.....		2							1		1
0105 ADENOVIRUS TYPE 5.....							1				
0107 ADENOVIRUS TYPE 7.....		1									
0201 INFLUENZA A VIRUS.....	1	40				2		1	1		
0202 INFLUENZA A VIRUS SUBTYPE H3N2		10				1					
0203 INFLUENZA B VIRUS.....	3	25									
0301 PARAINFLUENZA VIRUS TYPE 1.....		1									
0302 PARAINFLUENZA VIRUS TYPE 2.....		1			1						
0303 PARAINFLUENZA VIRUS TYPE 3.....		20									
0399 PARAINFLUENZA VIRUS TYPING PENDING.....		1									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	160				1	1				1
0500 RHINOVIRUS (ALL TYPES).....		14					1				1
0600 MYCOPLASMA PNEUMONIAE.....	2	11									
0700 ORNITHOSIS-PSITTACOSIS.....								1			
0809 COXSACKIEVIRUS A9.....		1				1					
0816 COXSACKIEVIRUS A16.....		1									4
0902 COXSACKIEVIRUS B2.....		2				1					
1002 ECHOVIRUS TYPE 2.....		1									
1006 ECHOVIRUS TYPE 6.....							1				
1007 ECHOVIRUS TYPE 7.....		2				1					
1009 ECHOVIRUS TYPE 9.....					1						
1011 ECHOVIRUS TYPE 11.....							1				
1022 ECHOVIRUS TYPE 22.....						1	1		1		

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

4

PERIOD : 4/9/80 to 17/9/80 80/19

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.-CONTINUED

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
1023 ECHOVIRUS TYPE 23.....							1				
1030 ECHOVIRUS TYPE 30.....		1			1		1				
1101 POLIOVIRUS TYPE 1.....		2									
1102 POLIOVIRUS TYPE 2.....		1									
1103 POLIOVIRUS TYPE 3.....							5		1		
1104 POLIOVIRUS-VACCINAL STRAIN....	1										
1200 MUMPS VIRUS.....					6						
1300 HERPES VIRUS GROUP-NOT TYPED..	1		2		1						10
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1	2				1					46
1302 EPSTEIN-BARR VIRUS (EB VIRUS) .	3										
1303 VARICELLA-ZOSTER VIRUS.....	2										5
1306 HERPES SIMPLEX TYPE 1.....		2			1					4	16
1307 HERPES SIMPLEX TYPE 2.....		2									12
1401 COXIELLA BURNETI.....	4	2									
1514 MOLLUSCUM CONTAGIOSUM.....											1
1521 MEASLES VIRUS.....	2	2				1					4
1522 RUBELLA VIRUS.....	2										17
1530 HEPATITIS A VIRUS.....	5	1						27			
1531 HEPATITIS B VIRUS.....	22	1						31			
1532 HEPATITIS B ANTIGEN.....	15						1	7			
1535 HEPATITIS A ANTIBODY.....								7			
1556 CMV - CYTOMEGALOVIRUS.....	9	4			1	1		2		5	
1563 CORONAVIRUS.....							4				
1564 ROTAVIRUS.....		1					154				
1565 CALICI VIRUS.....							1				
ROSS RIVER VIRUS	1										
ASTROVIRUS							6				
SMALL VIRUS (LIKE) PARTICLE							4				
Total.....	78	327	2	12		13	189	76	4	9	118

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

5

PERIOD : 4/9/80 to 17/9/80 ... 80/19
 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/malaise	Other	SIDS
0100 ADENOVIRUS NOT TYPED.....			3				3			
0101 ADENOVIRUS TYPE 1.....										1
0102 ADENOVIRUS TYPE 2.....	1							1		
0103 ADENOVIRUS TYPE 3.....								1		
0106 ADENOVIRUS TYPE 6.....						1				
0110 ADENOVIRUS TYPE 10.....		1								
0119 ADENOVIRUS TYPE 19.....	1	5								
0201 INFLUENZA A VIRUS.....					1		2	2		
0202 INFLUENZA A VIRUS SUBTYPE H3N2							1	5		
0203 INFLUENZA B VIRUS.....							2	4		1
0303 PARAINFLUENZA VIRUS TYPE 3....						1		1	1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)							1	3		1
0700 ORNITHOSIS-PSITTACOSIS.....				1						
0809 COXSACKIEVIRUS A9.....								1		
0816 COXSACKIEVIRUS A16.....								1		
0902 COXSACKIEVIRUS B2.....								1		
1006 ECHOVIRUS TYPE 6.....	1									1
1007 ECHOVIRUS TYPE 7.....								2		
1025 ECHOVIRUS TYPE 25.....								1		
1101 POLIOVIRUS TYPE 1.....										1
1102 POLIOVIRUS TYPE 2.....										1
1103 POLIOVIRUS TYPE 3.....										1
1200 MUMPS VIRUS.....			8	1						
1300 HERPES VIRUS GROUP-NOT TYPED..		1	1				1			
1301 HERPES SIMPLEX VIRUS NOT-TYPED		64								
1302 EPSTEIN-BARR VIRUS (EB VIRUS) .			6						1	
1303 VARICELLA-ZOSTER VIRUS.....			1	1						

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

6

PERIOD : 4/9/80 to 17/9/80 ... 80/19

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 ...

-CONTINUED

VIRUS OR VIRAL ANTIGEN	Eye	Gen-ital	Endo/sal gland	RES	Muscle/joint	Con-genital	PUO	Fever/mal-aise	Other	SIDS
1306 HERPES SIMPLEX TYPE 1.....	8	4						1	2	
1307 HERPES SIMPLEX TYPE 2.....		45							3	
1401 COXIELLA BURNETI.....					1		1	20		
1522 RUBELLA VIRUS.....			1		2					
1532 HEPATITIS B ANTIGEN.....					1					
1541 CHLAMYDIA A - TRIC TYPE.....		41								
1556 CMV - CYTOMEGALOVIRUS.....	1	2	1		1	2	4	9	4	1
1564 ROTAVIRUS.....			1				1	2		
ROSS RIVER VIRUS					3					
Total.....	12	163	22	3	9	4	16	55	11	8