AUSTRALIA

Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 808 reports were received this period.

General patterns as suggested by the reports received include:

. an indication that the Ross River virus epidemic in Queensland is beginning to wane, (27 reports from that State compared with 38, 86 and 63 for the three previous periods). The only other case this period was one from Broome in Western Australia.

. Cases with 'genital' symptoms figured prominently. They amounted to over 20% of all reports (156, compared with 122 and 102 for the previous two periods). The majority were from patients with herpes type 2 infections, which also showed an increase over previous weeks.

. Localised outbreaks included the continuation of the hepatitis A outbreak in Western Australia with 13 reports this period and 24 in CDI 80/12; an outbreak of rubella in Meningie in South Australia, with 17 cases confirmed by serology over the past month; and a slight increase in the number of reports of Q. fever, with outbreaks in South Australia, Queensland and Victoria (Melbourne and Camperdown).

. Rotavirus - 58 reports compared with between 25 and 35 for the previous few periods. These were mainly from South Australia (32 reports), New South Wales (11) and Victoria (10 reports). The Institute of Medical and Veterinary Science, Adelaide, reported the detection of both rotavirus, by electronmicroscopy, and reovirus by primary cell culture and continuous cell line, in a 2-year old girl.

Other reports of interest:

. Influenza A (H_3N_2) - two further isolations, both from Melbourne, one of which was imported, in June, from Singapore. The other was from a one-year old boy whose specimen had been collected in March this year.

. A single titre level of 1:160 to adenovirus was detected in Western Australia in the blood of a three-year old boy suspected of having Guillain-Barré syndrome. However the condition was finally labelled as "polyneuropathy of unknown aetiology".

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β-LACTAMASE PRODUCING N. GONORRHOEAE

Figures for isolations of β-lactamase producing N. gonorrhoeae for this year have now been received from New South Wales, Victoria and Western Australia. They reported 17, 12 (including two reported previously) and 17 cases respectively. This brings the 1980 total to 76, a large increase on the numbers reported during the January to June periods of each of the previous three years: 1977 - 17; 1978 - 24; 1979 - 27. Of the 76 isolations in 1980, 13 were from females.

Although some of the increase may be due to better reporting and more vigorous contact tracing, it is probable that this year's figures do indicate a general increase in incidence of the infection in the Australian community, mainly in travellers returning from abroad. A similar but less marked increase for the first half of this year has also been noticed in the United States (MMWR 30/5/80).

The table below shows the number of reports of penicillinase producing N. gonorrhoeae (PPNG) isolations from the various States in Australia with the probably source of infection.

Reports of PPNG Cases by Probable Source of Infection
Australia - 1980 Jan-June

Probably Source	N.S.W.	VIC	QLD	S.A.	W.A.	N.T.	TOTAL
Philippines	8	3	3	~~10~~ (5)	2	1	~~27~~ 22
Thailand	2	3	-	1	4	-	10
Malaysia	-	-	3	-	1	-	4
Hong Kong	2	1	-	-	-	-	3
Singapore	-	2	-	-	1	-	3
S.E. Asia (unspec.)	-	1	-	2	5	-	8
U.S.A.	-	-	-	2 (13)	-	-	2
Australia	5	-	1	~~6~~ (10)	~~3~~ 4	-	~~15~~ 23
Unknown	-	~~2~~ 3	-	2	-	-	~~4~~ 5
INDONESIA		1					1
	17	~~12~~ 14	7	~~20~~ 25	~~16~~ 17	1	~~76~~ 81

Both the increasing number of imported cases and the fact that the source of infection in 25% of the cases was either local or unknown, serve as pertinent reminders of the potential for explosive local outbreaks if vigorous contact tracing is not undertaken in all cases.

The summary below of a report and editorial comments on an episode in Shreveport, Louisiana, U.S.A., based on MMWR (1980) 29 21:241 is therefore of relevance.

On January 21, an isolate of N. gonorrhoeae taken from a 34 year old man with gonococcal urethritis was confirmed as penicillinase producing. He had first been diagnosed on 27 November 1979, and had received repeated antibiotic therapy, including procaine penicillin with probenecid, tetracycline and ampicillin, for persistent gonococcal urethritis. Following reculture and identification of the organism, the patient was successfully treated with spectinomycin (2g IM). An intense follow-up program, involving identification of the sexual partners of the patient, and their contacts, was undertaken. In addition all gonococcal isolates were screened for penicillin resistance by means of disc tests with resistant isolates being

subsequently tested for β-lactamase production. This revealed a total of 28 PPNG cases which were distributed in eight (apparently unrelated) chains of infection. Although no direct evidence of importation was obtained, further studies indicated that the pattern of susceptibility to 12 different antimicrobial agents was virtually identical for each isolate, and very similar to that of PPNG isolates examined in the Far East.

The editor stressed that delays involved in identifying early cases of persistent infection would contribute to PPNG transmission, and post-treatment cultures, 3-7 days following therapy, must be regarded as mandatory for all gonorrhoea patients. Even patients who appear cured should be retested after seven days, as treatment failure is then still possible; all positive isolates should be tested for penicillinase production. Spectinomycin 2g IM should be administered to all patients who continue to be infected. The use of spectinomycin, however, should be restricted to patients who fail primary therapy (with penicillin, tetracycline or ampicillin); spectinomycin is not recommended as primary therapy for gonococcal infection. (This is also the recommended policy in Australia, as indiscriminate use of spectinomycin could result in the development of strains resistant to this drug. Ed.)

NOSOCOMIAL INFLUENZA INFECTIONS

(Based on Canada Diseases Weekly Report (1979) 5 (49) 225-227)

A number of nosocomial outbreaks of influenza involving adult and paediatric patients have been reviewed by Hoffmann and Dixon[1] with a view to the control of influenza in hospitals. Paisley et al[2] have described the wide spectrum of clinical features of influenza A virus infections in young children, including the occurrence of apnoeic spells similar to those reported in infants with respiratory syncytial virus[3] and pertussis. Nosocomial infections accounted for 10% of the influenza cases studied. In another report, influenza A virus caused a sudden outbreak of illness in a neonatal intensive care unit, characterised clinically by apnoea, lethargy, poor feeding and other non-specific signs indistinguishable from those seen in bacterial sepses[4]. Nosocomial influenza infection was an important cause of inter-current fevers in another group of infants aged two years or under who were hospitalized for a week or more. Infants continued to shed influenza A virus for 7 to 21 days. This is in contrast to adults who do not usually excrete virus for more than three or four days[5]. Thus, children with influenza A remain infectious for many days and, despite the short incubation period, relatively long intervals may occur before susceptible contacts develop illness[6]. During an epidemic of influenza, 20% or more of infected persons may have subclinical illness but are nevertheless capable of transmitting the virus to others[1].

These nosocomial infections are consequently a greater problem among paediatric patients than among adults because of their susceptibility to virus infections, the longer excretory period and quantity of virus shed. Infection may be particularly threatening to patients with underlying congenital heart disease, malformations of the nervous system, severe mental retardation and conditions such as cystic fibrosis[7]. Parents, visitors and hospital staff appear to be largely responsible for the introduction of much of the infection.

The following measures might be considered in an attempt to limit nosocomial respiratory infection 8,9,10.

(i) Whenever possible, elective hospital admissions of patients should be avoided if the patient has underlying diseases predisposing to more severe respiratory infection. This particularly applies during winter, or when there are recognised community outbreaks of RSV infection or influenza. If admission cannot be postponed, a single cot cubicle is preferable to an open ward.

(ii) All infants and young children with respiratory illness, even those developing mild 'colds' should be considered contagious and isolated whenever possible. Careful use of cubicles may reduce spread of some viruses. Every attempt should be made to establish the aetiological agent using whatever rapid viral diagnostic techniques may be available. Patients infected with the same agent can be nursed together.

(iii) Rigorous handwashing and the wearing of gowns (but not masks) by hospital personnel caring for paediatric patients are advisable, although the role these procedures play in preventing spread of respiratory viruses has not been established.

(iv) Hospital personnel with respiratory disease or influenza-like illness during recognised community outbreaks of RSV infections or influenza, should be considered to be infected until proven otherwise. They should be excluded from caring for patients, particularly young infants in high-risk categories. This should not necessitate prolonged absence from work, since virus-excretion in adults is usually of short duration.

Additional control measures may be applied during epidemics of influenza (1,11). One possibility would be the isolation of all influenza patients in one part of the hospital. Although restriction of visitors is unlikely to reduce nosocomial transmission, persons with obvious influenza-like illness should be strongly discouraged from entering the hospital. In addition, since exposure of patients to influenza virus is thought to be mainly through hospital employees because of their longer and closer contact with patients, the CDWR recommends that serious consideration be given to immunizing hospital employees against influenza, preferably several weeks in advance of the expected influenza season.

The editor of CDWR comments that during the 1978/79 influenza season, the circulating influenza viruses of the H1N1 and type B variety were typically seen in children and young adults (ie. those born since 1957 when the H1N1 strain was last in circulation). Since this is also the age group in which the communicable diseases of childhood are most common, influenza infection may accompany another illness. During the 1978 influenza season in Canada, influenza A and B viruses were isolated from young people with measles, atypical measles, rubella, mumps and rashes of unknown aetiology. Therefore, if children with a communicable disease are hospitalized and also exhibit influenza-like symptoms, the isolation and control measures outlined above are warranted. This should continue until the results of laboratory investigations are known in order to prevent a nosocomial outbreak.

The Editor further urges that the prophylactic use of the antiviral drug,

amantadine, be considered for unimmunized, high-risk patients and hospital personnel, or for those in whom influenza vaccine has not had sufficient time to induce a serological response.

Since amantadine does not appear to suppress antibody response, it can be used chemoprophylactically in conjuction with inactivated influenza A virus vaccine until protective antibody responses develop. It has little effect on influenza B or other respiratory tract infections.

Care should be taken in giving amantadine to patients with renal impairment, congestive heart failure, peripheral oedema or orthostatic hypotension, liver disease, recurrent eczematoid rash, or to psychotic or severely psychoneurotic patients not controlled by chemotherapeutic agents. Because of possible birth defects, amantadine should not be given to women of childbearing potential unless, in the opinion of the physician, the expected benefit to the patient outweighs the possible risk to the foetus. Since the drug is excreted in the milk, it should not be given to nursing mothers.

Editorial comment

A decision to use amantadine for both the prevention and therapy of all types of influenza A virus infections was recently made by the National Institute of Allergy and Infectious Diseases (NIAID) in the United States. The following comments are based on the report of this Committee published in Science (1979) 206:1058

The panel recommended that the drug be used prophylactically for periods of four to six weeks (the normal length of an influenza epidemic in a community) when there is epidemiological and virological evidence of influenza A infection in: children and adults at high risk of morbidity or mortality because of other diseases, adults whose activities are essential to community function and who have not been vaccinated for influenza A, and individuals in "semi-enclosed environments", especially older persons, who have not been vaccinated. The panel stressed that vaccination should remain the primary form of prophylaxis against influenza A, but concluded that amantadine should be used as an adjunct to the vaccine in the recommended groups when vaccination was not possible, and to provide supplementary protection during the 10 days between vaccination and the development of protective antibodies.

They also recommended that amantadine be used therapeutically in the same groups recommended for prophylaxis. Treatment could also be given to patients diagnosed as having life-threatening influenza pneumonia, infants with influenza-associated croup, and in individuals whose community function requires that they be returned to work as soon as possible.

Concern was expressed however in that influenza A virus only constitutes a small part of the total clinical influenza infection, even during epidemics; and complications could arise both with the possible emergence of amantadine-resistant strains of influenza A virus, and possible side effects if the recommended dose was exceeded.

The immunisation of hospital staff is not generally recommended in Australia. The Australian Drug Evaluation Committee has approved the use of amantadine for prophylactic purposes for the H_2N_2 and H_3N_2 types of influenza A virus. It is not available as a pharmaceutical benefit for this purpose however; only for Parkinson's disease which is not drug induced.

References:

1. Ann. Intern. Med. (1977) 87:725
2. Am. J.Dis. Child. (1978) 132:34
3. N.Engl. J.Med. (1979) 300:393
4. J. Pediatr. (1977) 91:974
5. Pediatrics. (1975) 55:673
6. BMJ. (1973) 2:571
7. BMJ. (1967) 4:316
8. MMWR. (1978) 27, No.30
9. N.Engl. J.Med. (1975) 293:1343
10. Pediatrics. (1978) 62:728
11. N.Engl. J.Med. (1978) 298:621

CLOSTRIDIUM DIFFICILE COLITIS

The Fairfield Hospital monthly report (11 June 1980) described the case of a 69-year old woman who was admitted with a two-day history of diarrhoea and nausea. Clostridium difficile was isolated from all three of her faecal cultures. She had not received antibiotics previously but she had been receiving treatment with prednisolone for asthma. Her diarrhoea ceased after treatment with vancomycin.

Editorial comment

Isolation of the toxin-producing Cl. difficile from the faeces of healthy adults is rare[1]. It is normally associated with the administration of antibiotics, particularly in elderly patients, and it is possible that intestinal ischaemia may be a contributory factor. The proliferation of Cl. difficile has been associated following treatment with ampicillin, tetracycline, chloramphenicol, and in particular lincomycin and clindamycin. Approximately 30% of Cl. difficile isolates are resistant to clindamycin[2]. The suggested treatment is oral vancomycin or metronidazole. In an editorial in the BMJ in 1979[3] the author recommended that antibiotic induced colitis patients be nursed togehter to reduce cross-infection, and that since Cl. difficile is a spore-forming organism, adequate disinfection procedures such as the use of gluteraldehyde should be adopted.

References

1. J. Inf. Dis. (1977) 136:822
2. Lancet. (1978) 1:1063
3. BMJ. (1979) 2:349

ERRATUM - CDI 80/12

The references in both the tables and page 1 to an isolation of Norwalk agent (virus code 1566) was incorrect. The entry should have read 1556, cytomegalovirus.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE /

REPORTING PERIOD - 12/6/80 - 25/6/80 BULLETIN NUMBER - 80/13

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED	4		4	1	1	4		2	16
0101 ADENOVIRUS TYPE 1				4	1	2			7
0102 ADENOVIRUS TYPE 2					2	2		2	6
0103 ADENOVIRUS TYPE 3				1	1				2
0104 ADENOVIRUS TYPE 4						1			1
0105 ADENOVIRUS TYPE 5					1	2			3
0106 ADENOVIRUS TYPE 6								1	1
0109 ADENOVIRUS TYPE 9								1	1
0119 ADENOVIRUS TYPE 19	1							3	4
0199 ADENOVIRUS TYPING PENDING			1		4				5
0201 INFLUENZA A VIRUS	1		1	1			3		6
0202 INFLUENZA A VIRUS SUBTYPE H3N2				1	1				2
0301 PARAINFLUENZA VIRUS TYPE 1						1		1	2
0302 PARAINFLUENZA VIRUS TYPE 2				3	5	2			10
0303 PARAINFLUENZA VIRUS TYPE 3	1	1			4	4		3	13
0399 PARAINFLUENZA VIRUS TYPING PENDING					1	1		10	12
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)		4		3	5	4	3	2	21
0500 RHINOVIRUS (ALL TYPES)	4	1		2	8	7	2		24
0600 MYCOPLASMA PNEUMONIAE		1	2				1	3	7
0700 ORNITHOSIS-PSITTACOSIS	1					1			2
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED							2	1	3
0816 COXSACKIEVIRUS A16	1								1
0902 COXSACKIEVIRUS B2						1			1
0903 COXSACKIEVIRUS B3		1							1
1007 ECHOVIRUS TYPE 7								1	1
1011 ECHOVIRUS TYPE 11						1			1
1022 ECHOVIRUS TYPE 22							1		1
1024 ECHOVIRUS TYPE 24							1		1
1026 ECHOVIRUS TYPE 26			1						1
1030 ECHOVIRUS TYPE 30				3					3
1101 POLIOVIRUS TYPE 1							2		2

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 12/6/80 - 25/6/80 BULLETIN NUMBER 2 - 80/13

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

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
1102 POLIOVIRUS TYPE 2								1	1
1103 POLIOVIRUS TYPE 3						1			1
1104 POLIOVIRUS-VACCINAL STRAIN					1	1			2
1200 MUMPS VIRUS	3	2	1	4	1		1	1	13
1300 HERPES VIRUS GROUP-NOT TYPED	6			1		3			10
1301 HERPES SIMPLEX VIRUS-NOT TYPED	6	2	2		1		23	51	85
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	1					3			4
1303 VARICELLA-ZOSTER VIRUS	1		2				1	1	5
1306 HERPES SIMPLEX TYPE 1	11		7	14		4			36
1307 HERPES SIMPLEX TYPE 2	88		5	23		8			124
1399 HERPES VIRUS TYPING PENDING			1	2		14			17
1401 COXIELLA BURNETI	2			14		13	18		47
1502 PICORNA VIRUS-NOT TYPED								13	13
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)			1			2			3
1521 MEASLES VIRUS			2	1				1	4
1522 RUBELLA VIRUS				2		9	1		12
1530 HEPATITIS A VIRUS								13	13
1532 HEPATITIS B ANTIGEN			10	24		1	8	5	48
1535 HEPATITIS A ANTIBODY						3			3
1541 CHLAMYDIA A - TRIC TYPE	10		1			1		33	45
1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART)	7								7
1556 CMV - CYTOMEGALOVIRUS	5	2	4	13	1	5	5	4	39
1562 REOVIRUS (ALL TYPES)						1			1
1564 ROTAVIRUS	4		11	10		32		1	58
1599 ENTEROVIRUS TYPING PENDING		1	8		12	4			25
ROSS RIVER VIRUS							27	1	28
SMALL VIRUS (LIKE) PARTICLE	2			1					3
Total	159	15	64	128	50	138	99	155	808

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 3

PERIOD : 12/6/80 to 25/6/80 .... 80/13
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/ mucs memb
0100 ADENOVIRUS NOT TYPED.....	1	2			1		6				1
0101 ADENOVIRUS TYPE 1.....		3					1				
0102 ADENOVIRUS TYPE 2.....		2					4				
0103 ADENOVIRUS TYPE 3.....							1				
0104 ADENOVIRUS TYPE 4.....			1								
0105 ADENOVIRUS TYPE 5.....		2									
0106 ADENOVIRUS TYPE 6.....							1				
0109 ADENOVIRUS TYPE 9.....	1										
0119 ADENOVIRUS TYPE 19.....	2										
0201 INFLUENZA A VIRUS.....	1	4									
0202 INFLUENZA A VIRUS SUBTYPE H3N2		2									
0301 PARAINFLUENZA VIRUS TYPE 1....		1					1				
0302 PARAINFLUENZA VIRUS TYPE 2....		10									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	11									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		20									
0500 RHINOVIRUS (ALL TYPES).....	1	14									
0600 MYCOPLASMA PNEUMONIAE.....		6									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....		1					2				
0816 COXSACKIEVIRUS A16.....											1
0903 COXSACKIEVIRUS B3.....				1							
1007 ECHOVIRUS TYPE 7.....											1
1011 ECHOVIRUS TYPE 11.....							1				
1022 ECHOVIRUS TYPE 22.....		1									
1024 ECHOVIRUS TYPE 24.....							1				
1026 ECHOVIRUS TYPE 26.....							1				

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 4

PERIOD : 12/6/80 to 25/6/80 .... 80/13

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; 83 -Enceph-alitis; 03 -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
1030 ECHOVIRUS TYPE 30				3							
1101 POLIOVIRUS TYPE 1		4									
1102 POLIOVIRUS TYPE 2						1					
1103 POLIOVIRUS TYPE 3							1				
1104 POLIOVIRUS-VACCINAL STRAIN							2				
1200 MUMPS VIRUS	2	1		5							
1300 HERPES VIRUS GROUP-NOT TYPED		1									2
1301 HERPES SIMPLEX VIRUS-NOT TYPED	24	4	2	1		1					39
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	2	1									
1303 VARICELLA-ZOSTER VIRUS											4
1306 HERPES SIMPLEX TYPE 1		1									23
1307 HERPES SIMPLEX TYPE 2											5
1401 COXIELLA BURNETI	1	1						1			
1502 PICORNA VIRUS-NOT TYPED	3	1				2	5				
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)	1										2
1521 MEASLES VIRUS		1		1			1				2
1522 ROBELLA VIRUS											10
1530 HEPATITIS A VIRUS	4							8			
1532 HEPATITIS B ANTIGEN	14							34			
1535 HEPATITIS A ANTIBODY								3			
1541 CHLAMYDIA A - TRIC TYPE	34										
1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART)											7
1556 CMV - CYTOMEGALOVIRUS	7	7				1	1	1		4	
1562 REOVIRUS (ALL TYPES)							1				
1564 ROTAVIRUS		1					57				
ROSS RIVER VIRUS	4										1
SMALL VIRUS (LIKE) PARTICLE							3				
Total	103	103	3	11	1	5	90	47		4	98

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 5

PERIOD : 12/6/80 to 25/6/80 ... 80/13
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		1	1		
0101 ADENOVIRUS TYPE 1	3									
0102 ADENOVIRUS TYPE 2								1		
0103 ADENOVIRUS TYPE 3	1									
0105 ADENOVIRUS TYPE 5							1			
0119 ADENOVIRUS TYPE 19	2									
0201 INFLUENZA A VIRUS								1		
0303 PARAINFLUENZA VIRUS TYPE 3							1			
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)								2		
0500 RHINOVIRUS (ALL TYPES)										1
0600 MYCOPLASMA PNEUMONIAE								1		
0700 ORNITHOSIS-PSITTACOSIS							1			
0902 COXSACKIEVIRUS B2					1					
1007 ECHOVIRUS TYPE 7							1			
1102 POLIOVIRUS TYPE 2								1		
1200 MUMPS VIRUS			1	2				2		
1300 HERPES VIRUS GROUP-NOT TYPED		4								
1301 HERPES SIMPLEX VIRUS-NOT TYPED	2	12						1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS)			1							
1303 VARICELLA-ZOSTER VIRUS								1		
1306 HERPES SIMPLEX TYPE 1	3	8						1		
1307 HERPES SIMPLEX TYPE 2		119								
1401 COXIELLA BURNETI							6	40		
1502 PICORNA VIRUS-NOT TYPED				1				1		
1522 RUBELLA VIRUS									2	
1530 HEPATITIS A VIRUS							1			
1541 CHLAMYDIA A - TRIC TYPE		11								
1556 CMV - CYTOMEGALOVIRUS		2		4		5	3	6	6	
ROSS RIVER VIRUS					24					
Total	11	156	2	7	26	5	15	59	8	1