Communicable Diseases Intelligence

Bulletin number 81/20

Issue date: 9 October 1981

Contents:

CONTAMINATED OLIVE OIL - Further to the article on "Toxic Pneumonia - Spain" in CDI 81/19, all oils imported from Spain are being monitored for the brand names implicated in the outbreak, and samples assayed for aromatic amines by the Australian Government Analytical Laboratories, following liaison between the Department of Health and Australian Customs. Checks for the brand names are also being made at the retail level in all States.

VIRUS REPORTING SCHEME - A total of 868 reports were received this period. Reports of measles infections, mainly in young children, continue to rise (29 reports received compared with 22, 20 and 17 for the previous three periods). The reports from the Royal Alexandra Hospital for Children, Sydney, include a three year old boy with a neuroblastoma, a six year old girl with Wilms' tumour and a nine year old boy with Hodgkin's disease. It was stated that both the latter patients had been immunised with live measles vaccine at 12 months of age. Because of the possibility of persistent and aggravated infection, measles vaccine, like other live virus vaccines, is contra-indicated in subjects with diseases of the reticulo-endothelial system or those taking immuno-suppressive drugs. In addition, the virus tables of CDI 81/19 included a report of measles in a seven year old girl immunised at 12 months, the normal sibling of a leukaemia patient. Measles CF antibody was also detected by Fairfield Hospital, Melbourne, in two patients with clinical signs of SSPE.

In the eight week period, 27 July - 25 September 1981, 12 cases of coxsackievirus B4 infection were diagnosed by the Royal Children's Hospital, Melbourne. Of these, six patients presented with cough with fever, one with cough, fever and rash, one with cough and abdominal pain, two with diarrhoea and two were isolations from SIDS cases. Rotavirus was also detected in the faeces of one of the diarrhoea patients. The previous coxsackievirus B4 infections at this hospital were in December 1979.

NH AND MRC RECOMMENDATIONS ON IMMUNISATION PROCEDURES - Readers are asked to note that in the Medical Journal of Australia, 3 October 1981, the NH and MRC vaccine recommendations printed on page 364 are incorrect. The Editor has been notified, and a corrected version will be published in a forthcoming issue.

The Bulletin is compiled and distributed by the Environmental Health Branch, Department of Health, P.O. Box 100, Woden, A.C.T. 2606, Australia, and is available on request.

LEGIONNAIRES' DISEASE (LD) - SOUTH AUSTRALIA

(Contributed by D.J. Merry, J. Pitt and T.W. Steele, Institute of Medical and Veterinary Science, Adelaide).

The introduction in January 1979 of direct smear, culture and serological diagnostic techniques for LD at the Institute of Medical and Veterinary Science has assisted the identification of 20 cases of Legionella pneumophila serogroup 1 infection in South Australian patients to 31 July 1981 (see Table 1).

TABLE 1 LD investigation to 31 July 1981

Period	Direct smear, DFA, culture	Serology (IFA)	No. of cases
1979	17(40)	240 (327)	6
1980	24(42)	151 (237)	0
1981 (to 31 July 1981)	28(56)	213 (340)	14

The figures in brackets denote the total number of tests performed.

DFA - direct fluorescent antibody test.
IFA - indirect fluorescent antibody test.

All 20 cases of LD appeared to have been of a sporadic nature, as no epidemiological evidence for common sources of infection could be established. There is no apparent explanation for the absence of cases in 1980, or for the increased number in the first seven months of 1981. Suitable respiratory specimens were available for rapid laboratory diagnosis by direct fluorescent microscopy (DFA) in 12 of the 20 cases. Seven were positive by DFA, and of these five were positive by isolation on charcoal yeast extract (CYE) agar or passage in guinea pigs. Specimens were not available from the remaining eight patients. Table 2 tabulates in chronological order the laboratory data for the 20 cases. Early attempts to isolate L. pneumophila from patient respiratory secretions directly onto CYE agar were unsuccessful due to overgrowth by oral flora. However, positive in vitro culture was obtained in the last two cases (patients 19 and 20) with use of the selective antibiotics pimafucin, polymyxin and vancomycin.

The indirect fluorescent antibody technique (IFA) recommended by the Centers for Disease Control was used throughout the three year period, and changes in the batches of reagents were closely monitored. When serum samples were collected daily, it was possible to demonstrate seroconversion with serogroup 1 infections between days 9-12 from the onset of symptoms. These rises in diagnostic titres were usually abrupt, with three or four fold increases in a 24 hour period.

Antibody class studies are considered important early in investigations, and are useful in distinguishing between antibody elicited during infection and the range of titres in apparently healthy people[1]. In a survey conducted in March 1979, total antibody titres of 1/64 or higher were found in 31% and levels of 1/256 or higher in 8.4% of 208 healthy blood-donors and 126 LD case-related controls. However, only 4.2% had IgM antibody titres of 1/64 or higher. None of the case-related controls had pneumonia in the 12 months previous to the survey, and periodic monitoring of the blood-donors has shown no significant serological changes to date.

Table 2 Summary of Laboratory Findings in L.D. Serogroup 1 Cases

PATIENT			DFA	CULTURE		ANTIBODY TITRES (INCREASE)		OUTCOME
No.	Age	Sex		In vitro(1)	In vivo(2)	Total globulin change	IgM-specific change	
* 1	36	F	-	ND	ND	<64/8192	<64/2096	
* 2	34	M	+ (Spt, PM, lung)	+ (PM, lung)	+ (Spt)	32/1024	32/1024	Died
* 3	62	M	+ (Trach. suct.)	-	+ (Spt)	128/33 000	<32/4096	
4	57	M	-	-	-	64/1024	<32/1024	Died
* 5	53	M	NSR	NSR	NSR	128/8196	64/8196	
6	61	M	+ (Bronch. wash)	-	-	32/512	32/512	
7	75	M	+ (Spt)	-	-	16/128	<16/64	
8	63	M	NSR	NSR	NSR	32/4096	<64/2048	Died
9	57	M	NSR	NSR	NSR	64/512	64/512	
10	37	M	-	-	ND	<32/2048	<32/512	
11	52	M	NSR	NSR	NSR	<32/8192	<32/4096	
12	54	F	-	-	ND	<32/512	<32/512	
13	52	M	NSR	NSR	NSR	32/512	<32/64	
14	58	M	-	-	ND	16/1024	<16/256	
15	54	M	NSR	NSR	NSR	<16/256	<16/64	
16	58	F	NSR	NSR	NSR	16/128	<16/64	
17	64	M	+ (Spt, PM, lung)	+ (PM, lung)	+ (Spt)	128	32	Died
18	50	M	NSR	NSR	NSR	1024	1024	
19	68	M	+ (Trach. suct. Bronch. wash)	+	+	32/256	32/128	Died
20	74	F	+ (Bronch. wash)	+	ND	NSR	NSR	Died

* - Case reports detailed in MJA (1980) 1 : 368.
(1) - Charcoal yeast extract agar.
(2) - Guinea pigs.
NSR - No specimen received.
ND - Not done.
DFA - Direct fluorescent antibody.

Antibody titres to serogroups 2, 3 and 4 were occasionally seen, but only one patient (number 3 in Table 2) showed specific IgM titres to other serogroups. These titres were four fold lower than titres to serogroup 1.

Reference

1. MJA (1980) 1 : 365.

HEALTH HAZARDS OF PET EXCRETA
(Based on California Morbidity (1981) No. 23)

Excreta of pets create aesthetically unpleasant situations as well as providing sources for the transmission of various zoonoses; particularly toxoplasmosis and visceral larva migrans (VLM) caused by the larvae of the nematode Toxocara canis (dog) and possibly T. cati (cat) and T. leonina (cat and dog).

Infestation prevalences for the intestinal roundworm T. canis are 50-100% in young puppies and about 20% in mature dogs. Although there is a high prevalence of T. cati in cats (44-78%), their habit of burying faeces reduces the likelihood of transmission. The ascarid eggs are voided in the faeces, and 10-32% of soil samples in parks, playgrounds, sandboxes etc. may be infected. The eggs become infective in four weeks, and remain viable for months to years. Humans are infected by the ingestion of eggs from fingers, food or fomites contaminated with infected soil. The eggs then hatch in the small intestine, and the larvae penetrate the mucosa, migrating to the liver via the portal circulation, to the lungs and enter the systemic circulation. Although infection usually causes few or no symptoms, with up to 13% of apparently healthy persons possessing antibody, severe cases of VLM can occur from widespread larvae dissemination. The larvae may attack the retina causing blindness, as well as being found in the ear, brain, spinal cord, skin and other tissues.

Toxoplasmosis, caused by the protozoan Toxoplasma gondii, is transmitted by cat faeces. Up to 64% of cats are positive in serological surveys. Oocysts are excreted in the faeces 4-20 days after the cat becomes infected, and periodically thereafter. The eggs become infective within one to three days, and remain viable in soil for months. Transmission to humans is again from the ingestion of oocysts contaminating fingers, food or fomites. Human infections, while often asymptomatic, can result in fever and lymphadenopathy, chorioretinitis in chronic infections, and a fulminating form in immunologically incompetent individuals. Primary infections in pregnant women may lead to miscarriages and congenital malformations (see CDI 79/22).

Environmental control measures that might be adopted to reduce the risk of human infection include:

- Education of the public regarding the epidemiology of these diseases.

- The treatment of pets to eliminate Toxocara worms. Puppies, kittens and their mothers should be treated two, four, six and eight weeks following birth, and periodically thereafter to prevent massive liberation of eggs. No treatment is known to eliminate latent Toxoplasma infection.

- Leashing of pets, especially in playgounds and parks, and immediate removal and sanitary disposal of faeces wherever they are voided.

. Fencing of playground areas, although this precaution is unlikely to keep cats out.

. Advising pregnant women to avoid cats and contact with their faeces.

. Eliminating unwanted cats and dogs.

In the absence of effective animal restraint, it is particularly difficult to prevent contamination of sandboxes or children's sandpits which are favoured by children for digging and by cats for voiding. Repeated contamination precludes consideration of periodic sand replacement or sterilization. However, faecal contamination can be prevented by covering sandboxes with lids when not in use. The most suitable covering is a light, wood framed wire mesh, since heavy solid covers could result in entrapment and accidental trauma. Although the magnitude has not been measured, the risk posed by contaminated sandboxes is very real, and all efforts should be made to eliminate or reduce the exposure.

The health hazards associated with the use of dog and cat manure on home gardens are far greater than the potential benefit from its fertilizer value. Dog and cat faeces contain about 0.7% nitrogen, 0.25% phosphate and 0.02% potash. By comparison steer manure contains two and a half times as much nitrogen, the same amount of phosphate and about half as much potash. Dog and cat faeces should be disposed of by flushing down a toilet, burying at least six inches deep in soil, or by placement in tight plastic bags for garbage collection. Hands must be thoroughly washed after disposal of pet excreta.

ENTERIC ADENOVIRUSES
(Based on CDS 81/24)

Adenoviruses have long been regarded as agents which infect primarily the respiratory tract and/or conjunctivae. There are 36 established serotypes. The prototype strains were derived from respiratory specimens (8 serotypes), eye specimens (11), anal specimens (15), urine (1) and lung/kidney (1). However, only one third of these recognized serotypes have been firmly established as causative agents in human respiratory disorders, keratoconjunctivitis and acute haemorrhagic cystitis[1]. Adenoviruses have been implicated in cases of intussusception, meningitis, encephalitis, juvenile rheumatoid arthritis, orchitis, thyroiditis and appendicitis, as well as recent reports of symptomatic genital infection[2], but there is insufficient evidence to establish casual relationships with these presentations.

Adenoviruses, particularly types 1 and 2, are frequently shed in the stools of children[3], with excretion of one serotype often persisting for several months[4,5]. This persistent virus shedding could be the result of "passive" downflow from a prolonged respiratory infection, or of replication in the intestinal mucosa, since oral administration of infectious adenovirus in gelatin capsules has shown that common serotypes can replicate in the adult intestine. Although this replication probably follows a respiratory infection in some children, some of the higher numbered adenovirus serotypes are isolated almost exclusively from stool rather than respiratory specimens[6], possibly reflecting a predilection for replication in the gut rather than in the upper respiratory tract or conjunctivae[4].

There are few reports of any signs and symptoms of gastro-intestinal dysfunction during the faecal shedding of the 36 established adenovirus serotypes. Several outbreaks of diarrhoea, vomiting and abdominal pain in the late 1950's and early 1960's were attributed to some of the common serotypes, particularly types 3 and 7,[7] but prospective studies have now established little or no evidence that these serotypes are isolated more frequently from gastro-enteritis patients than from control subjects[8,9,10].

However, the advent of electron microscopy (EM) in virology brought the discovery of human rotaviruses and other small round virus-like particles in human stools[11]. Almost invariably these virus particles could not be cultured by conventional techniques, even though at least 10^6 particles/g. must be present to be detected by EM. This anomaly suggested some condition or factor present *in vivo* which allowed replication at the intestinal mucosa, but which was absent *in vitro*. Adenoviruses can be detected in the stools of 4-10% of children with gastro-enteritis. In one study, 216 of 392 adenovirus specimens produced no cytopathic effect (CPE) in tissue culture[12], and these "non-infectious" strains have been called "enteric", "fastidious", "uncultivable" or "non-cultivable" adenoviruses.

The fastidious enteric adenoviruses have been implicated as causative agents in two separate gastro-enteritis outbreaks. Six children and a nurse were involved in an incident in a hospital long-stay ward, with adenovirus particles detected in the stools of the nurse and four of the children[13]. In the second report, adenoviruses were detected in six of 17 children with gastro-enteritis on a RAF base in England[14].

DNA restriction enzyme analysis of the genomes of the enteric adenoviruses has indicated different and characteristic profiles from the established serotypes[15]. The strains also appear to agglutinate rat erythrocytes to moderately high titres[16], and two-directional haemagglutination-inhibition tests indicate a lack of identity with the established 36 serotypes. The development of an ELISA technique has also suggested unique antigenic determinants[17]. Enteric adenoviruses usually produce little or no CPE in tissue culture, but certain strains have been passaged in Chang cells, an established line of conjunctival cells[18]. Serial passage was effected at an incubation temperature of 33°C not 37°C, which could reflect an adaptation to growth at cool epithelial surfaces, or be a consequence of viral defectiveness exerted by incorrect *in vitro* conditions. Antiserum against one strain neutralised 24 other strains in culture, but neither the antiserum, nor the virus, cross-reacted with the 36 established serotypes. Another characteristic of these strains was a low infectivity, with isolates from different stools from the same patient even exhibiting degrees of infectivity.

The common strains of enteric adenoviruses will probably be given the designation "type 38". Related strains have been detected in the stools of children from Malaysia and the Transvaal, suggesting an ubiquitous epidemiology, although there may also be less common serotypes of enteric adenoviruses.

A list of references used for this article is available from the Editor.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 17/9/81 - 30/9/81 BULLETIN NUMBER . 81/20

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	2		2		1	2	9	1	17
0101	ADENOVIRUS TYPE 1	2				1	2			5
0102	ADENOVIRUS TYPE 2					3				3
0103	ADENOVIRUS TYPE 3					1	2			3
0107	ADENOVIRUS TYPE 7		1		1	2	1			5
0113	ADENOVIRUS TYPE 13								2	2
0114	ADENOVIRUS TYPE 14			1					1	2
0199	ADENOVIRUS TYPING PENDING					3	1			4
0201	INFLUENZA A VIRUS	3	1	4	1		1	9	1	20
0202	INFLUENZA A VIRUS SUBTYPE H3N2							1		1
0203	INFLUENZA B VIRUS	3	1					1		5
0206	INFLUENZA A VIRUS SUBTYPE H1N1							3		3
0299	INFLUENZA VIRUS								1	1
0301	PARAINFLUENZA VIRUS TYPE 1				1			1		2
0303	PARAINFLUENZA VIRUS TYPE 3		1	1	3	4	1	3		13
0400	RESPIRATORY SYNCYTIAL VIRUS (RS)	5	5	1	3	4	17	5	18	58
0500	RHINOVIRUS (ALL TYPES)	2	1		2	11		4	2	22
0600	MYCOPLASMA PNEUMONIAE	3		2	2		1	6	1	15
0700	ORNITHOSIS-PSITTACOSIS	1			2					3
0899	COXSACKIEVIRUS A9							1	1	2
0904	COXSACKIEVIRUS B4	2			1	4				7
0905	COXSACKIEVIRUS B5						3			3
1009	ECHOVIRUS TYPE 9						1		4	5
1011	ECHOVIRUS TYPE 11			2						2
1014	ECHOVIRUS TYPE 14						1			1
1017	ECHOVIRUS TYPE 17							1		1
1022	ECHOVIRUS TYPE 22	1		1	1					3
1025	ECHOVIRUS TYPE 25								1	1
1030	ECHOVIRUS TYPE 30						1			1
1099	ECHOVIRUS TYPING PENDING						3			3
1101	POLIOVIRUS TYPE 1				1			1		2
1102	POLIOVIRUS TYPE 2					1	1	1		3

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 17/9/81 - 30/9/81 BULLETIN NUMBER . 81/20

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	PAHC (NSW)	PIMR/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
1104 POLIOVIRUS-VACCINAL STRAIN	1				2				3
1200 MUMPS VIRUS	2			9	1	3		4	19
1300 HERPES VIRUS GROUP-NOT TYPED	12		1	3		9		3	28
1301 HERPES SIMPLEX VIRUS NOT-TYPED		3		3			1	48	55
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	5							2	7
1303 VARICELLA-ZOSTER VIRUS	2	1	1	1	1	1		1	8
1306 HERPES SIMPLEX TYPE 1	2		4	17		18	9		50
1307 HERPES SIMPLEX TYPE 2	34		12	26		9	17		98
1399 HERPES VIRUS TYPING PENDING			4		3	3			10
1401 COXIELLA BURNETI	5		1	1		2	9		18
1514 MOLLUSCUM CONTAGIOSUM						2			2
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)								1	1
1521 MEASLES VIRUS	5	6	6	4	6	2			29
1522 RUBELLA VIRUS	2		5	8		1	13		29
1532 HEPATITIS B ANTIGEN	11		4	28	1	9	6	12	71
1535 HEPATITIS A ANTIBODY	4					4		11	19
1541 CHLAMYDIA A - C TRACHOMATIS	21		3					5	29
1556 CMV - CYTOMEGALOVIRUS	12		4	8	5	1	9	9	48
1563 CORONAVIRUS				1					1
1564 ROTAVIRUS	12	31	8	3	15	23	4	4	100
1599 ENTEROVIRUS TYPING PENDING			7						7
POXVIRUS GROUP NOT TYPED				1					1
ROSS RIVER VIRUS							6		6
ASTROVIRUS	3							1	4
SMALL VIRUS (LIKE) PARTICLE	3					3			6
ARBO. GROUP B.							1		1
Total	156	51	74	131	69	128	121	134	868

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 3

PERIOD : 17/9/81 to 30/9/81 .... 81/20

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

	VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0100	ADENOVIRUS NOT TYPED							1				
0101	ADENOVIRUS TYPE 1	1	2					1				
0102	ADENOVIRUS TYPE 2	1	2									
0103	ADENOVIRUS TYPE 3		1									1
0107	ADENOVIRUS TYPE 7		2					1				
0201	INFLUENZA A VIRUS	3	14							1		1
0202	INFLUENZA A VIRUS SUBTYPE H3N2		2									
0203	INFLUENZA B VIRUS	2	3									
0206	INFLUENZA A VIRUS SUBTYPE H1N1	1	3									
0301	PARAINFLUENZA VIRUS TYPE 1		1									1
0303	PARAINFLUENZA VIRUS TYPE 3		14									
0400	RESPIRATORY SYNCYTIAL VIRUS (RS)	3	52				1					
0500	RHINOVIRUS (ALL TYPES)	1	20									
0600	MYCOPLASMA PNEUMONIAE	3	8									1
0700	ORNITHOSIS-PSITTACOSIS	1										
0809	COXSACKIEVIRUS A9		1				1					1
0904	COXSACKIEVIRUS B4	1	2	1	1			1				
0905	COXSACKIEVIRUS B5		1					2				
1009	ECHOVIRUS TYPE 9	3	1				1					
1011	ECHOVIRUS TYPE 11		1					1				
1014	ECHOVIRUS TYPE 14				1							
1017	ECHOVIRUS TYPE 17	1										
1022	ECHOVIRUS TYPE 22		1					2				
1025	ECHOVIRUS TYPE 25									1		
1030	ECHOVIRUS TYPE 30							1				
1101	POLIOVIRUS TYPE 1		1					1				
1102	POLIOVIRUS TYPE 2		1					2				

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

4

PERIOD : 17/9/81 to 30/9/81 .... 81/20

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 ali-tis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
1104 POLIOVIRUS-VACCINAL STRAIN....		1					1				
1200 MUMPS VIRUS.....		2		6							
1300 HERPES VIRUS GROUP-NOT TYPED..	1	1									
1301 HERPES SIMPLEX VIRUS NOT-TYPED	2	1									27
1302 EPSTEIN-BARR VIRUS (EB VIRUS).								3			
1303 VARICELLA-ZOSTER VIRUS.....	2	1		1							3
1306 HERPES SIMPLEX TYPE 1.....		6	3								24
1307 HERPES SIMPLEX TYPE 2.....											6
1401 COXIELLA BURNETI.....	4	2									1
1514 MOLLUSCUM CONTAGIOSUM.....											2
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....	1	5	3			2					16
1522 RUBELLA VIRUS.....	5	1									21
1532 HEPATITIS B ANTIGEN.....	39							30		1	1
1535 HEPATITIS A ANTIBODY.....	4							14			
1556 CMV - CYTOMEGALOVIRUS.....	14	12						3		4	1
1563 CORONAVIRUS.....							1				
1564 ROTAVIRUS.....	2						98				
POXVIRUS GROUP NOT TYPED											
[illegible] VIRUS											2
ASTROVIRUS							4				
SMALL VIRUS (LIKE) PARTICLE	1						5				
Total.....	97	165	7	9		5	122	50	2	5	111

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

81/20

PERIOD : 17/9/81 to 30/9/81 ...
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
C101 ADENOVIRUS TYPE 1.....										1
C103 ADENOVIRUS TYPE 3.....	1									
C107 ADENOVIRUS TYPE 7.....	1						1			
C113 ADENOVIRUS TYPE 13.....		2								
C114 ADENOVIRUS TYPE 14.....	1							1		
C201 INFLUENZA A VIRUS.....			1				1	5		
C400 RESPIRATORY SYNCYTIAL VIRUS (RS).....							1			1
C500 RHINOVIRUS (ALL TYPES).....								1		
C600 MYCOPLASMA PNEUMONIAE.....			1		1		1	3		
C700 ORNITHOSIS-PSITTACOSIS.....								2		
C904 COXSACKIEVIRUS B4.....										1
1011 ECHOVIRUS TYPE 11.....								1		
1022 ECHOVIRUS TYPE 22.....								1		
1104 POLIOVIRUS-VACCINAL STRAIN....										1
1200 MUMPS VIRUS.....			13							
1301 HERPES SIMPLEX VIRUS NOT-TYPED		25						1	1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			4						1	
1303 VARICELLA-ZOSTER VIRUS.....		1								
1306 HERPES SIMPLEX TYPE 1.....	3	10						2	1	
1307 HERPES SIMPLEX TYPE 2.....		92								
1401 COXIELLA BURNETI.....					2		1	7	1	
1521 MEASLES VIRUS.....	1								3	
1522 RUBELLA VIRUS.....	1				5					
1535 HEPATITIS A ANTIBODY.....								1		
1541 CHLAMYDIA A - C TRACHOMATIS...	1	28								
1606 CMV - CYTOMEGALOVIRUS.....		3		1		5		2	3	1
1654 REVET VIRUS					4					
ARBO. GROUP B.					1					
Total.....	9	161	19	1	13	5	5	27	10	5