



# Communicable Diseases Intelligence

Bulletin number 84/15

Issue date: 27 July 1984

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VIRUS REPORTING SCHEME A total of 1084 reports were received this period.

- . The two flavivirus reports included the diagnosis of dengue at Fairfield Hospital, Melbourne, in a 26 year old female who had recently returned from Manila; and the detection of cross-reacting IgM antibody to MVE, dengue type 4 and Alfuy antigens in sera referred to the State Health Laboratory, Brisbane, from Papua New Guinea from a patient with fever and lymphadenopathy.
- . Molluscum contagiosum-like particles were detected at Fairfield Hospital by electron microscopy of skin lesions from a 48 year old male who had been diagnosed as an AIDS patient in another Melbourne hospital in September 1983. The patient had a history of chronic perianal herpes and an active cytomegalovirus infection, and was admitted for treatment of subacute viral encephalitis. A 29 year old American male tourist, in whom a diagnosis of AIDS had been made during 1983 in the USA, was also admitted recently to Fairfield with repeated focal convulsions and a lung infection. A lung biopsy revealed a Pneumocystis carinii and cytomegalovirus infection, and a Candida species was isolated from bronchial washings. Peripheral atypical shingles lesions were also present on the palms and fingers. However following successful treatment, he has since returned to the USA. To date, 12 cases of AIDS with five deaths have been notified by the State health authorities throughout Australia.
- . Rubella specific IgM antibody was detected by Fairfield Hospital and the State Health Laboratory, Brisbane, in cord blood of two neonates. One neonate appeared healthy despite a history of maternal rubella late in the first trimester of pregnancy, but no information was provided for the other. Congenital rubella syndrome (CRS) represents the most serious outcome of rubella infection in terms of health burden. Costs for the lifetime care of an infant with CRS in the USA have been recently estimated to be in excess of \$200,000. The second consequence of infection in a pregnant woman is therapeutic abortion, which is thought to be more common than CRS. Strategies based on schoolgirl immunisation produce their results slowly and take about 20 years to achieve their full effectiveness, although the delineation and identification of women of childbearing age most likely not to be vaccinated would hasten the elimination of CRS.

## VIBRIO SURVEILLANCE - AUSTRALIA (1973-84)

(Contributed by P. Desmarchelier, Commonwealth Institute of Health, Sydney)

In the period January 1973 - March 1984, 104 cultures of *Vibrio* species isolated from human infections were referred to, or isolated at, the Commonwealth Institute of Health (CIH). The identification of the isolates and their source are listed in Table 1.

TABLE 1 Human isolates of *Vibrio* species, CIH (1973-1984)

<u>Species</u>	<u>Number</u>	<u>Source of isolate (Number)</u>
<u>V. cholerae</u> 01	13	faeces (12); blood (1)
<u>V. cholerae</u> non 01	34	faeces (30); blood (3); fallopian tubes (1).
<u>V. parahaemolyticus</u>	38	faeces (25); wound (7); ear infection (1); unknown (9)
<u>V. vulnificus</u>	6	wound (4); blood (1); leg ulcer (1)
<u>V. alginolyticus</u>	5	faeces (1); ear infection (1); wound (1); peritoneal fluid (1); unknown (1)
<u>V. mimicus</u>	4	respiratory tract (2); faeces (1); wound (1)
<u>V. harveyi</u>	1	leg ulcer - <u>S. aureus</u> also isolated
<u>V. damsella</u>	1	wound
<u>V. metschnikovii</u>	1	faeces
<u>V. fluvialis</u>	1	faeces

Five of the V. cholerae 01 strains (3 of serotype ogawa; 2 of serotype inaba) were known to have been acquired overseas, whereas eight cases (ogawa (1); inaba (7)) had no recent history of overseas travel. All the isolates were biotype eltor. All the imported strains were non-haemolytic with one strain producing both haemolytic and non-haemolytic colonies. By comparison, the indigenous strains were all strongly haemolytic, and seven of them have been shown to possess the genes encoding cholera toxin and to produce the toxin. The non-toxigenic local 01 strain was isolated together with a *Salmonella* species, and the significance of the isolation is therefore uncertain. Three patients had simultaneous infections; a *Salmonella* species was isolated from two cases and a V. cholerae non 01 strain was cultured subsequent to the isolation of V. cholerae 01 from one patient. Strains of V. cholerae non 01 were isolated predominantly from the faeces of patients suffering from diarrhoea. Of these infections, 23 were in travellers returning from overseas, three were acquired locally and four were supplied with insufficient information. Two of the three blood cultures were from patients known to be alcoholic, and the third was from a fatal case where the organism was also isolated from urine and a liver abscess. The fallopian tube infection was from a patient who practised aquatic sports. Two foodborne outbreaks due to V. cholerae non

01 occurred on international airline flights en route to Australia. The strains belonged to a variety of serotypes and showed no correlation between the clinical source and probable country of origin of the infection. None of the strains were shown to possess sequences homologous with cholera toxin genes or to produce the toxin.

V. parahaemolyticus was the most common marine *Vibrio* species isolated. Seventeen of the 25 faecal isolates were from travellers returning from South-East Asia. Five patients had no recent history of travel, of which two presented with chronic diarrhoea. One confirmed and one suspected foodborne outbreak occurred on an international airline flight to Australia. Where histories were provided, infections were associated with the consumption of sea-foods or marine activities such as swimming and fishing. Twenty of the faecal isolates were typable serologically, but all of the extra-intestinal isolates were nontypable.

V. mimicus was isolated from a variety of clinical sources. The faecal isolate was from an overseas traveller suffering from diarrhoea, while the other infections were acquired locally and the patients' histories suggested the probable sources were coastal waters. The strains belonged to different serological strains and did not possess cholera toxin genes.

Patients' histories also indicated that infections with the other marine *Vibrio* species were associated with either occupational or recreational activities in coastal or estuarine waters or the consumption of seafoods. The V. fluvialis infection was acquired locally, but no information was provided with the strain of V. metschnikovii.

With the exception of V. damsella, all of the *Vibrio* species from the human infections described have also been isolated at CIH from either local or imported food or water. V. damsella has been described more recently and therefore may not have been recognized if isolated in the past. In view of the low incidence of these species in human infections in Australia, and the ready availability of health care to most of the population, *Vibrio* infections may not be considered a major public health problem. However, the microbiologist needs to be aware of the possible presence of these species, particularly in the high risk groups described.

#### CHOLERA IN 1983

(Based on WER (1984) 59 : 141 and 150)

A total of 64061 cases of cholera in the world were reported to the World Health Organisation for the year 1983 compared with 54856 cases in 1982. Thirty-three countries reported infection during the year compared with 37 in 1982. No new country was reported infected during 1983.

Countries in Africa reported 36722 cases in 1983 (37427 in 1982); Ghana and Mozambique had an upsurge in cases while South Africa and Zaire had a marked reduction. The United Republic of Tanzania indicated that the reduction in the use of tetracycline for chemoprophylaxis resulted in a decline in the incidence of tetracycline-resistant strains in the country.

In Asia, the number of reported cases rose from 15191 in 1982 to 27005 in 1983, mainly as a result of important increases in India, Indonesia, Malaysia and Vietnam. Eleven countries reported cases in 1983 as compared with 12 in 1982. Classical

cholera strains are still being reported in Bangladesh following the reappearance in an outbreak there in late 1982, but as laboratory examination is not carried out for all cases, the exact proportion of classical, as compared with eltor, strains is not known.

There was a recrudescence of the cholera epidemic in the Trust Territory of the Pacific Islands in July 1983, after the Territory had been declared free of infection on 21 June: 314 cases were reported from the area in 1983 compared with 2214 in 1982.

#### ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS) SURVEILLANCE

##### AIDS - UNITED STATES OF AMERICA

(Based on MMWR (1984) 33 : 337)

As of 18 June 1984, 4918 patients meeting the surveillance definition for AIDS were reported to CDC, Atlanta. Although 2221 (45%) of all reported patients are known to have died, more than 76% of patients diagnosed before July 1982 are dead.

Of the 4861 adult AIDS patients, Pneumocystis carinii pneumonia continued to be the most common opportunistic disease (Table 1), and the groups at highest risk of acquiring AIDS remained homosexual or bisexual men (Table 2).

TABLE 1. Percentage distribution of AIDS cases by category of diagnosis - USA

<u>Diagnosis</u>	<u>% Adult cases</u>	<u>% Paediatric cases</u>
<u>P. carinii</u> pneumonia	53%	77%
Kaposi's sarcoma	24%	2%
Kaposi's sarcoma + <u>P. carinii</u> pneumonia	6%	4%
Other opportunistic infections	17%	18%
Number of cases	4861	57

TABLE 2. Percentage distribution of adult AIDS cases by patient group

<u>Patient group</u>	<u>% Distribution</u>
Homosexual/bisexual	71.9%
IV drug user	17.5%
Haitian born	3.8%
Haemophiliac	0.8%
Transfusion recipient	1.1%
Heterosexual sex partner	0.8%
Other/unknown	4.1%

P. carinii pneumonia was also the most common disease in paediatric AIDS cases (Table 1). Of the 57 patients, 23 came from families in which one or both parents had a history of intravenous drug abuse, 13 had one or both parents who were born in Haiti, and 12 had transfusions with blood or blood components before their onsets of illness.

To June 1984, 51 AIDS cases were reported to CDSC, London (Table 3). Forty-three cases were in homosexual/bisexual men, three in heterosexual men with known risk factors, two in haemophiliacs and three in female patients.

TABLE 3. Distribution of AIDS cases by category of diagnosis - UK

<u>Diagnosis</u>	<u>Cases</u>	<u>Deaths</u>
P. carinii pneumonia	17	11
Kaposi's sarcoma	20	6
Kaposi's sarcoma + P. carinii pneumonia	2	2
Other opportunistic infections	11	8
Cerebral lymphoma	1	1
TOTAL	51	28

AIDS - FRANCE, NETHERLANDS AND SWITZERLAND  
(Based on WER (1984) 59: 57, 138 and 178)

Notifications meeting the CDC criteria for case definition for AIDS in France, Netherlands and Switzerland are detailed in Table 4.

TABLE 4. Distribution of AIDS cases by category of diagnosis - France, Netherland and Switzerland  
Figures in parentheses relate to deaths

<u>Diagnosis</u>	<u>France</u> <sup>(1)</sup>	<u>Switzerland</u> <sup>(2)</sup>	<u>Cases</u> <u>Netherlands</u> <sup>(3)</sup>
Opportunistic infections	68(30)	10	
P. carinii pneumonia			6(4)
Suspected P. carinii pneumonia			1(1)
Kaposi's sarcoma	26(4)	2	5(2)
Kaposi's sarcoma + opportunistic infections	13(10)	6	
Kaposi's sarcoma + P. carinii pneumonia			2(2)
Other opportunistic infections			2(1)
TOTAL	107(44)	18(16)	16(10)

(1) Data to 1 January 1984

(2) Data to 1 March 1984

(3) Data to 1 May 1984

AIDS - THE AMERICAS, EXCLUDING THE USA  
(Based on WER (1984) 59 : 74)

Table 5 tabulates the notifications of AIDS cases in the Americas, excluding the USA, to 31 December 1983

TABLE 5. Notification of AIDS cases from the American continent

<u>Country</u>	<u>Number of cases as of 31 December 1983</u>
Argentina	8
Brazil	27
Canada	92(1)
Haiti	232

Jamaica	1
Mexico	4(2)
Surinam	1
Trinidad and Tobago	9
Uruguay	5

- (1) As of June 1984 (CDWR (1984) 10/25 : 98)  
 (2) As of 12 September 1983

#### AIDS - UPDATE

(Based on WER (1984) 59 : 128 and MMWR (1984) 33 : 377).

In May 1983, workers at the Harvard School of Public Health, the CDC, and the National Institutes of Health reported serological, virological and epidemiological evidence of an association of a retrovirus (human T-cell leukaemia virus-I; HTLV-I) with AIDS<sup>(1)</sup>. Certain animal retroviruses exhibit an affinity for lymphocytes and have been shown to cause diseases such as feline leukaemia, bovine leukaemia, and equine infectious anaemia, all of which share immunological and pathological characteristics with AIDS in man. HTLV-I had been previously identified as the cause of adult T-cell leukaemia, a neoplastic disease common in southern Japan but rare in the USA. It was believed that positive antibody finds with HTLV-I reflected cross-reactions with the possible causative agent.

At the same time workers at the Pasteur Institute, Paris, reported the isolation of a new retrovirus from cultured lymph node T lymphocytes of a homosexual man with lymphadenopathy, and therefore termed by them as lymphadenopathy-associated virus (LAV)<sup>(2)</sup>. Specific antibodies to LAV were detected in approximately 70% of patients with persistent lymphadenopathy and 40% of AIDS patients studied<sup>(3)</sup>. A viral isolate, possibly related to LAV, was also isolated from two siblings with haemophilia B, one of whom had an AIDS-like syndrome, indicating transmission of the retrovirus by plasma products<sup>(4)</sup>. Further reports have detailed the virus's strict tropism and cytopathic effect on lymphocytes of the OKT-4(helper) subset<sup>(5)</sup>, and its adaption to infect a lymphoblastoid cell line obtained by transformation with Epstein-Barr virus of B lymphocytes derived from a healthy donor<sup>(6)</sup>.

Concurrently, researchers from the National Cancer Institute, Bethesda, documented multiple (44) isolations from lymphocytes of patients with AIDS and conditions thought to be related to AIDS of a retrovirus termed by them as human T-lymphotropic retrovirus III (HTLV III)<sup>(7)</sup>. The key to these findings was the isolation of specific permissive clones from a human neoplastic aneuploid T-cell line derived from an adult with lymphoid leukaemia, which facilitates virus isolation from patients and permits high level production of virus<sup>(8)</sup>. Rates of antibodies to HTLV-III in AIDS patients and population groups at high risk of AIDS were very similar to those reported by the French workers<sup>(9)</sup>.

Although direct comparative results have not been published, HTLV-III and LAV are likely to be the same virus because:

- . They have the same appearance by electron microscopy.
- . They are both lymphotropic and cytopathic for OKT-4 cells.
- . Isolates from American AIDS patients (two from a blood donor-recipient pair and one from a homosexual male), when compared, were immunologically indistinguishable from LAV<sup>(10)</sup>.

- . Serological tests of a large number of specimens from patients with AIDS or related conditions show similar results when either of the prototype viruses is used as antigen.
- . Preliminary results suggest that LAV and HTLV-III are at least highly related based on competitive radioimmunoassay of their core proteins.

Traditionally, final proof that a particular agent causes a disease usually involves the attestation of Koch's postulates. However, present evidence does fulfil much of the modern equivalent i.e. an indicator (virus, viral protein or viral nucleic acid) of a specific viral infection must be found in all, or nearly all, patients with AIDS or with signs or symptoms that frequently precede AIDS; antibody to the same virus must be shown to develop in constant temporal association with the development of AIDS; and transmission of the same virus to a previously uninfected experimental animal or to a human must be demonstrated with subsequent development of the disease e.g. in a blood donor-recipient pair<sup>(10)</sup>.

Three basic serological procedures are currently described for detection of antibody to HTLV-III/LAV; an enzyme-linked immunosorbent assay (ELISA) to whole disrupted virus (9, 11, 12); a radioimmunoprecipitation assay (RIPA) to the presumed major core protein (called p25) of LAV<sup>(13)</sup>; and assay of antibody to major viral antigens by the Western blot technique<sup>(14)</sup>. Sera from several high-risk populations are being tested by these techniques by the National Cancer Institute, the Institut Pasteur, and CDC, with the support of numerous collaborators to determine the frequency of exposure to HTLV-III/LAV and to correlate seropositivity with current infection, clinical signs and symptoms, and prognosis.

Preliminary data suggest that serological evidence of exposure to HTLV-III/LAV may be common in certain populations at increased risk of AIDS. Antibody to HTLV-III was detected by ELISA in sera from six (35%) of 17 American homosexual men without symptoms of AIDS<sup>(11)</sup>. Sera from eight (18%) of 44 homosexual men without lymphadenopathy attending a venereal clinic in Paris had antibody detected by ELISA to LAV<sup>(9)</sup>. Antibody prevalence to LAV (RIPA) has increased from 1% (1/100) in 1978 to 25% (12/48) in 1980 and 65% (140/215) in 1984 among samples of sera from homosexual men attending a sexually transmitted diseases clinic in San Francisco. Antibody prevalence among the above men tested in 1984 who had no symptoms or clinical signs of AIDS or related conditions was 55% (69/126). In New York City, where the AIDS cases among intravenous (IV) drug users are concentrated, 87% (75/86) of recent heavy IV drug users without AIDS had antibody to LAV by ELISA, while over 58% (50/86) of the same group had antibody to LAV detected by RIPA. In contrast, fewer than 10% of 35 methadone patients from New York City had antibody to LAV by RIPA. All of these latter patients had been in treatment at least three years with greatly reduced IV drug usage. Eighteen of 25 (72%) asymptomatic persons with haemophilia A in a home-care treatment program demonstrated antibody to LAV antigens utilising the Western blot technique. All had used factor VIII concentrates from 1980 to 1982.

The high prevalence of antibody to HTLV-III/LAV among these groups demonstrate that exposure to the virus is much more common than AIDS itself among populations with increased incidences of the disease. If AIDS follows the pattern of many

other infectious diseases, host response to infection would be expected to range from subclinical to severe.

The serological tests are sufficiently sensitive and specific to be of value in estimating the frequency of infection with HTLV-III/LAV in certain populations, and for providing important information about the natural history of the disease in such groups. Less clear are the implications of a positive test result for an individual. For some, the result may be a false positive caused by infection with an antigenically related virus or nonspecific test factors. The determination of the frequency and cause of falsely positive tests is essential for proper interpretation of test results, but remains to be established, particularly in populations, such as blood donors who belong to no known AIDS risk groups, where the prevalence of true infection with HTLV-III/LAV is expected to be very low.

A positive test for most individuals in populations at greater risk of acquiring AIDS will probably mean that the individual has been infected at some time with HTLV-III/LAV. Whether the person is currently infected or immune is not known based on the serological test alone. HTLV-III/LAV has been isolated in both the presence and absence of antibody, but the frequency of virus in antibody-positive persons is yet to be determined. For seropositive individuals with mild or no signs of disease, including those in whom the virus can be demonstrated, the prognosis remains uncertain since the incubation period for the life-threatening manifestations of AIDS may range from one year to more than four years<sup>(15)</sup>.

#### References

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5. Science (1984) 225 : 59
6. Science (1984) 225 : 63
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8. Science (1984) 224 : 497
9. Science (1984) 224 : 506
10. Science (1984) 225 : 69
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15. NEJM (1984) 310 : 69

#### ERRATUM, LISTERIOSIS SURVEILLANCE - VICTORIA

In CDI (1984) 84/13, page 3, it was stated that "L. monocytogenes is a Gram-negative mobile rod." This should have read a Gram-positive mobile rod.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE  
 REPORTING PERIOD - 5/7/84 - 18/7/84 BULLETIN NUMBER 84/15  
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR		PHH/	FAIR-			STATE	STATE	Total
	(NSW)/ WVH (ACT)	RAHC (NSW)	POW (NSW)	FIELD (VIC)	RCH (VIC)	IMVS (SA)	LAB (QLD)	LAB (WA)	
0100 ADENOVIRUS NOT TYPED.....		2				5	1	4	12
0101 ADENOVIRUS TYPE 1.....				1			1		5
0102 ADENOVIRUS TYPE 2.....				1				3	1
0103 ADENOVIRUS TYPE 3.....			1				1	1	3
0104 ADENOVIRUS TYPE 4.....								1	1
0105 ADENOVIRUS TYPE 5.....						2	1		3
0106 ADENOVIRUS TYPE 6.....						7		1	8
0108 ADENOVIRUS TYPE 8.....								1	1
0114 ADENOVIRUS TYPE 14.....								1	1
0119 ADENOVIRUS TYPE 19.....								1	1
0131 ADENOVIRUS TYPE 31.....								1	1
0199 ADENOVIRUS TYPING PENDING.....						1	3		4
0201 INFLUENZA A VIRUS.....			6						6
0203 INFLUENZA B VIRUS.....			2				1		4
0301 PARAINFLUENZA VIRUS TYPE 1.....				3	7	2	1	3	16
0302 PARAINFLUENZA VIRUS TYPE 2.....		1		10	9	7		1	28
0303 PARAINFLUENZA VIRUS TYPE 3.....	1				3	3	7	1	15
0399 PARAINFLUENZA VIRUS TYPING PENDING.....						1			1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)....		13	4	10	21	21	17	18	104
0500 RHINOVIRUS (ALL TYPES).....				5	4	19	2	9	39
0600 MYCOPLASMA PNEUMONIAE.....	1	1	4	2			8	4	20
0700 ORNITHOSIS-PSITTACOSIS.....				2				2	4
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....								1	1
0809 COXSACKIEVIRUS A9.....								3	3
0816 COXSACKIEVIRUS A16.....							1		1
0902 COXSACKIEVIRUS B2.....								1	1
0904 COXSACKIEVIRUS B4.....		2							2
0905 COXSACKIEVIRUS B5.....				3					3
1005 ECHOVIRUS TYPE 5.....								2	2
1006 ECHOVIRUS TYPE 6.....				1	1				2
1009 ECHOVIRUS TYPE 9.....				3	1		1		5
1011 ECHOVIRUS TYPE 11.....					1				1
1014 ECHOVIRUS TYPE 14.....							1		1
1017 ECHOVIRUS TYPE 17.....				1					1
1021 ECHOVIRUS TYPE 21.....								1	1
1022 ECHOVIRUS TYPE 22.....						1	1		2
1024 ECHOVIRUS TYPE 24.....								1	1
1025 ECHOVIRUS TYPE 25.....								1	1
1026 ECHOVIRUS TYPE 26.....						1			1
1030 ECHOVIRUS TYPE 30.....		1							1
1101 POLIOVIRUS TYPE 1.....						2	2	1	5
1102 POLIOVIRUS TYPE 2.....								4	4
1104 POLIOVIRUS-VACCINAL STRAIN.....			2		6				8
1200 MUMPS VIRUS.....			2	9		2			13
1300 HERPES VIRUS GROUP-NOT TYPED.....	5			3		4	1	3	16
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....		1							1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....				2				5	7
1303 VARICELLA-ZOSTER VIRUS.....						1		1	2
1306 HERPES SIMPLEX TYPE 1.....				57		18	23	11	109
1307 HERPES SIMPLEX TYPE 2.....				84		20	45	19	168
1399 HERPES VIRUS TYPING PENDING.....			14		5	4			23
1401 COXIELLA BURNETI.....							4		4
1502 PICORNA VIRUS-NOT TYPED.....			2						2
1514 MOLLUSCUM CONTAGIOSUM.....					1	2	2	1	8
1521 MEASLES VIRUS.....			2						2
1522 RUBELLA VIRUS.....				3			1		4
1532 HEPATITIS B ANTIGEN.....	1		7	23	1	12	30	17	91
1535 HEPATITIS A ANTIBODY.....			2	4		4	12	2	24
1541 CHLAMYDIA A - C TRACHOMATIS.....				17			16	44	77
1556 CMV - CYTOMEGALOVIRUS.....	2		1	6	5	12	5	4	35
1564 ROTAVIRUS.....		21	28	4	16	36	1	31	137
1599 ENTEROVIRUS TYPING PENDING.....			6		8	4			18
9902 POXVIRUS GROUP NOT TYPED.....				2					2
9992 ROSS RIVER VIRUS.....							7	6	13
9994 SMALL VIRUS (LIKE) PARTICLE.....		2							2
9995 DENGUE.....				1					1
9998 ARBO. GROUP B. ....							1		1
Total.....	10	44	83	257	104	184	194	208	1,084

## AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 5, 7, 84 to 18, 7, 84 ....

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Viral Identifications by Clinical Information Table 1.

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/ mucous memb
0100 ADENOVIRUS NOT TYPED.....		3						1			
0101 ADENOVIRUS TYPE 1.....		4						1			
0104 ADENOVIRUS TYPE 4.....		1						1			
0105 ADENOVIRUS TYPE 5.....		2						1			
0106 ADENOVIRUS TYPE 6.....	1	3	1								
0114 ADENOVIRUS TYPE 14.....						1					
0131 ADENOVIRUS TYPE 31.....							1				
0201 INFLUENZA A VIRUS.....	1	4									
0203 INFLUENZA B VIRUS.....	1	2							1		
0301 PARAINFLUENZA VIRUS TYPE 1....		16									
0302 PARAINFLUENZA VIRUS TYPE 2....		26									1
0303 PARAINFLUENZA VIRUS TYPE 3....		13				1					1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	99									
0500 RHINOVIRUS (ALL TYPES).....		39									1
0600 MYCOPLASMA PNEUMONIAE.....	2	15				1			1		
0700 ORNITHOSIS-PSITTACOSIS.....	1	3									
0809 COXSACKIEVIRUS A9.....				3							
0816 COXSACKIEVIRUS A16.....											1
0904 COXSACKIEVIRUS B4.....							1				1
0905 COXSACKIEVIRUS B5.....		1		2							
1005 ECHOVIRUS TYPE 5.....		1									
1006 ECHOVIRUS TYPE 6.....		1									
1009 ECHOVIRUS TYPE 9.....		2					2				
1014 ECHOVIRUS TYPE 14.....							1				
1017 ECHOVIRUS TYPE 17.....				1							
1021 ECHOVIRUS TYPE 21.....				1							
1022 ECHOVIRUS TYPE 22.....							1				
1024 ECHOVIRUS TYPE 24.....							1				
1026 ECHOVIRUS TYPE 26.....							1				
1030 ECHOVIRUS TYPE 30.....				1							
1101 POLIOVIRUS TYPE 1.....		3					2				
1102 POLIOVIRUS TYPE 2.....							1				
1104 POLIOVIRUS-VACCINAL STRAIN....		2					3				
1200 MUMPS VIRUS.....	2			5							
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		1									
1303 VARICELLA-ZOSTER VIRUS.....											2
1306 HERPES SIMPLEX TYPE 1.....	5	6	1				1	1		1	56
1307 HERPES SIMPLEX TYPE 2.....	4										30
1514 MOLLUSCUM CONTAGIOSUM.....	1										
1521 MEASLES VIRUS.....		2	2			1					4
1522 RUBELLA VIRUS.....	2										1
1532 HEPATITIS B ANTIGEN.....	24							50	1		
1535 HEPATITIS A ANTIBODY.....	2							22			
1556 CMV - CYTOMEGALOVIRUS.....	5	12					1	2		6	
1564 ROTAVIRUS.....	1						136				
9992 ROSS RIVER VIRUS.....	4										1
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
Total.....	57	261	4	13		4	158	75	3	7	100

## AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 5/7/84 to 18/7/84 ...

84/15

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
0102 ADENOVIRUS TYPE 2.....								1		
0103 ADENOVIRUS TYPE 3.....	3									
0106 ADENOVIRUS TYPE 6.....							5			
0108 ADENOVIRUS TYPE 8.....		1								
0119 ADENOVIRUS TYPE 19.....	1									
0201 INFLUENZA A VIRUS.....									1	
0302 PARAINFLUENZA VIRUS TYPE 2....										1
0303 PARAINFLUENZA VIRUS TYPE 3....										1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....							1	4	1	
0500 RHINOVIRUS (ALL TYPES).....								1	1	
0600 MYCOPLASMA PNEUMONIAE.....							1	7		
0902 COXSACKIEVIRUS B2.....									1	
0904 COXSACKIEVIRUS B4.....								1		
1005 ECHOVIRUS TYPE 5.....								2		
1006 ECHOVIRUS TYPE 6.....							1			
1009 ECHOVIRUS TYPE 9.....						1	1	2		
1011 ECHOVIRUS TYPE 11.....									1	
1022 ECHOVIRUS TYPE 22.....							1			
1025 ECHOVIRUS TYPE 25.....				1						
1102 POLIOVIRUS TYPE 2.....					1			1		2
1104 POLIOVIRUS-VACCINAL STRAIN....						1				2
1200 MUMPS VIRUS.....			6							
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			5	2						
1306 HERPES SIMPLEX TYPE 1.....	4	35	1		1			2	1	
1307 HERPES SIMPLEX TYPE 2.....		133						1		
1401 COXIELLA BURNETI.....					3			4		
1521 MEASLES VIRUS.....					1					
1522 RUBELLA VIRUS.....						1				
1532 HEPATITIS B ANTIGEN.....				1					15	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	75				1				
1556 CMV - CYTOMEGALOVIRUS.....		1				2	1	3	2	1
9992 ROSS RIVER VIRUS.....					9			1		
9995 DENGUE.....										
9998 ARBO. GROUP B. ....			1					1		
Total.....	9	247	13	4	15	6	11	31	23	7