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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 1426 reports was processed this period. Influenza A activity appears to have declined (see Table 1). One hundred and fifty-one infections were reported (62 subtyped H_3N_2, 7 H_1N_1 and 82 untyped). The two H_3N_2 isolations reported from the Royal Alexandra Hospital for Children, Camperdown, were identified as A/Wellington/3/84. A/Vic/3/85 was isolated from a one month old male and an 83 year old male by the State Health Laboratory, Brisbane. There has been an increased number of influenza B virus reports for this period, compared with the similar time period in 1984 (see Table 2).

TABLE 1

	1984	1985
Influenza A reports, generation 14, 20 June-3 July	-	41
Influenza A reports, generation 15, 4 July-17 July	6	121
Influenza A reports, generation 16, 18 July-31 July	-	246
Influenza A reports, generation 17, 1 Aug-14 Aug	31	151

TABLE 2

	1984	1985
Influenza B reports, generation 14, 20 June-3 July	-	9
Influenza B reports, generation 15, 4 July-17 July	4	7
Influenza B reports, generation 16, 18 July-31 July	4	16
Influenza B reports, generation 17, 1 Aug-14 Aug	6	39

There have been fewer parainfluenza virus reports in 1985 compared with the similar time period in 1984 (Table 3).

TABLE 3

	1984	1985
Parainfluenza reports, generation 14, 20 June-3 July	54	16
Parainfluenza reports, generation 15, 4 July-17July	59	19
Parainfluenza reports, generation 16, 18 July-31 July	37	25
Parainfluenza reports, generation 17, 1 Aug-14 Aug	54	26

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GONOCOCCAL SURVEILLANCE - AUSTRALIA (JANUARY-MARCH 1985)

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

The AGSP records the sensitivity to antibiotics of gonococci isolated in all States and Territories of Australia as determined by agar plate dilution techniques. A full description of the program and the laboratory methods employed has been published[1].

This report provides details of the penicillin sensitivities of 1170 isolates of Neisseria gonorrhoeae for the period January-March 1985. Table 1 gives the percentages of gonococcal isolates classified as either (A) sensitive (minimal inhibitory concentration (MIC) value = 0.008 µg/ml) or (B) less sensitive (MIC = 0.12 µg/ml) to penicillin, by region. Comparison is made with the data obtained over the same period in 1984.

TABLE 1 Penicillin sensitivity of N. gonorrhoeae isolates (January-March 1985)

Centre	Percentage of isolates		
	Sensitive (A)*	Less Sensitive (B)*	PPNG**
Brisbane	26.5(34.5)***	63.3(53.0)	7.2(8.7)
Sydney	20.5(15.0)	59.3(55.0)	8.8(18.1)
Melbourne	18.2(12.0)	60.2(46.0)	10.2(5.1)
Adelaide	36.8(39.3)	45.0(48.3)	2.7(5.2)
Perth	25.8(27.0)	48.4(35.0)	5.7(6.0)

No. strains examined = 1170 (1363)
* The A and B categories represent modal distribution peaks
** Penicillinase-producing N. gonorrhoeae
*** Figures in parenthesis represent data for the corresponding period in 1984.

Increasing resistance to the penicillins may occur as the result of several separate chromosomal mutations which additively increase resistance (intrinsic resistance) either through alterations to penicillin-binding proteins or else through changes in the permeability of the outer membrane of the organism. When grouped according to their resistance, as determined by penicillin minimal inhibitory concentrations (MIC), the majority of gonococci are distributed in a bi-modal fashion into either sensitive or less sensitive categories. Infections with strains classified as sensitive or less sensitive to penicillin [(A) and (B) above], usually respond to standard treatment regimes.

In the current quarter, less sensitive strains predominated, confirming a trend evident for some time, especially in Brisbane, Sydney and Melbourne. In addition to the material shown in Table 1, information was also available from Hobart, Canberra and Darwin. Less sensitive strains predominated in Canberra whereas in Darwin and Hobart, the two groups were

equally represented, although numbers of isolates from these three centres were fewer than in other centres.

In addition to the sensitive and less sensitive grouping of intrinsically resistant gonococci, a third group of relatively-resistant gonococci (MIC $\geq$ 1.0 mg/L) is also recognised. In Australia the number of infections with strains in this MIC range has remained low, these strains representing less than 2% of all isolates in this quarter.

A second and distinct form of resistance occurs when gonococci acquire the capacity to destroy penicillin by elaborating a penicillinase. An outbreak of infections due to penicillinase-producing gonococci (PPNG) recently occurred in Sydney and currently there has been a sharp and now sustained increase in the number of such infections in Melbourne. A feature of such outbreaks is the isolation of PPNG from patients who have not travelled overseas.

In this quarter a total of 97 PPNG were isolated from all centres except Hobart. Of these, 64 infections were in overseas travellers (to Asia and the South Pacific) or their contacts. Thirteen were patients in Sydney and Melbourne who had acquired the infection locally, two had acquired PPNG infections in the Northern Territory without a history of travel and in 18 cases the source of infection was unknown. In the 83 male patients, 75 isolates were urethral and four pharyngeal. In 4 males, the site of infection was not specified. There were 13 endocervical infections in the 14 female patients and a single pharyngeal isolate.

REFERENCE

1. Br. J. Vener. Dis. (1984) 60 : 226-30

FATAL DEGENERATIVE NEUROLOGICAL DISEASE IN PATIENTS WHO RECEIVED PITUITARY-DERIVED HUMAN GROWTH HORMONE - USA

(Based on MMWR (1985) 34 : 359)

Reports of rapidly progressive and fatal degenerative neurological disorders in three recipients of human growth hormone (hGH) have been received by the US Food and Drug Administration (FDA) and the National Institutes of Health (NIH). In two cases, diagnoses of Creutzfeldt-Jakob disease (CJD) were made at autopsy.

All three patients had had growth failure secondary to growth hormone deficiency. They had been treated during childhood and adolescence with hGH extracted from pooled human cadaver pituitary glands. The hormone used to treat these patients was produced and distributed by the National Hormone and Pituitary Program (NHPP, formerly the National Pituitary Agency) under an investigational exemption for the use of a new drug (IND).

Case 1. A 20-year-old man with hypopituitarism and Type 1 diabetes mellitus developed dysarthria and a gait disturbance in May 1984. By September, his neurological status had deteriorated so that he was no longer able to walk, could not care for himself, and required bladder catheterisation. His mental status had deteriorated and he was unable to carry on a meaningful conversation. He died in November 1984. Examination of the brain revealed spongiform encephalopathy consistent with CJD.

This patient had grown poorly during the first year of life. Hypothyroidism was diagnosed when he was 15 months old. In September 1966, a diagnosis of growth hormone deficiency was made. The patient was treated with daily injections of hGH from September 1966 to July 1980.

Case 2. A 22-year-old man developed weakness and gait disturbance in autumn, 1983. During the next six months, he developed severe ataxia involving extremities, trunk and head. He also had speech impairment, difficulty swallowing and dementia. He died in April 1985. Histological examination of the brain at the Armed Forces Institute of Pathology revealed extensive changes of spongiform encephalopathy compatible with CJD.

This patient was evaluated for growth failure at seven years of age and was found to be growth hormone deficient. He was treated with hGH from June 1969 to October 1977.

Case 3. A 34-year-old man with hypopituitarism developed a gait disturbance in December 1983. He had received hGH from 1963 to 1969. Examination in June 1984 showed bilateral horizontal end gaze nystagmus, mild intention tremor and wide-based gait. The symptoms worsened over the next several months, with increasing somnolence, memory loss and urinary incontinence.

The patient's symptoms progressed to include swallowing difficulties, diplopia, and finally, dementia. He died in February 1985. No autopsy was done.

Comment

CJD occurs with a frequency of approximately one case per million population per year in the United States and Europe[1]. Most cases occur sporadically and involve patients over 50 years of age. Inoculation of chimpanzees with brain tissue from affected patients results in a similar neurodegenerative disease in the animals within 18-36 months[2]. Iatrogenic CJD has been reported in a patient who received a corneal transplant from an affected donor[3] and in two patients exposed to intracranial electrodes that had previously been used in a patient with CJD[4].

The CJD pathogen is resistant to chemical and physical methods commonly used for decontamination or sterilisation[5]. There is evidence suggesting that procedures used recently to extract and purify hGH from cadaver pituitary glands may eliminate experimental contamination by scrapie, an agent similar to the CJD pathogen. The methods used by the NHPP were changed in 1977, but there is no assurance that current procedures eliminate the risk of transmitting the CJD pathogen.

From 1963 to early 1985, approximately 10,000 US patients received hGH through the NHPP. The average duration of therapy was four years. Each patient received hormone from two or three batches per year. Each batch was derived from a pool of approximately 16,000 cadaver pituitary glands.

The three patients described here received hGH for 14, 8, and 6 years, respectively. Records of the NHPP indicate that patients 1 and 2 received several common lots. Patients 1 and 3 received one lot in common. No single lot was administered to all three patients; however, all three received hormone

during 1969. The occurrence of fatal neurodegenerative disorders consistent with CJD in three of 10,000 patients exposed to hGH between 1963 and 1985 strongly suggests that the hormone, a product of pooled human tissue, may have been the vehicle for transmission of the CJD pathogen. It is not yet known how many other members of this cohort may have developed similar neurodegenerative disorders. Epidemiological studies have been undertaken to determine the status of recipients of hGH.

Editorial Comment

The safety of natural growth hormone, following the reported deaths from CJD of three US patients who received the NIH-produced product several years ago, has been reviewed and natural hGH allowed to remain on the market in Austria, Denmark, France, Israel, Norway, Poland, Spain, Switzerland, Italy, Japan and Argentina.

Distribution of hGH has been suspended by regulatory authorities in Australia, Belgium, Finland, Greece, the Netherlands, Sweden, the UK, Canada, West Germany and the USA. Many of these countries, including Australia, are carrying out studies to determine whether the product should be allowed back on the market.

Retrospective studies in approximately 6,000 recipients of natural hGH have already been carried out in nine European countries, as well as in Japan, Argentina and Israel. There has been one report, from the UK, of a link between a death from CJD and natural hGH. However, there were no further cases of death from CJD found in the remaining patients in the retrospective studies.

Of the three reported deaths in the US, it now appears that one was not caused by CJD. The treating physician and the pathologist disagree over the diagnosis in the second case, and there may be an independent reading of the autopsy slides in the other death. The causal relationship was not established between use of natural hGH (in 1974) and the death this year of the UK patient from CJD.

The natural/recombinant issue. There are "unresolved safety concerns" regarding Genentech's rDNA hGH, according to an FDA Commissioner Dr Frank Young. Some patients have formed antibodies to the Genentech hormone, and while Genentech's New Drug Application (NDA) is being given high FDA priority and compassionate distribution has been expanded, approval of this product "may not be a panacea".

REFERENCES

1. Epidemiol. Rev. (1980) 2 : 113-35
2. Science (1968) 161 : 388-9
3. N. Engl. J. Med. (letter) (1974) 290 : 692-3
4. Lancet (letter) (1977) 1 : 478-9
5. N. Engl. J. Med. (1982) 306 : 1279-82

UPDATE: PUBLIC HEALTH SERVICE WORKSHOP ON HUMAN T-LYMPHOTROPIC VIRUS TYPE III ANTIBODY TESTING - USA

(Based on MMWR (1985) 34 : 477)

The enzyme immunoassay (EIA)* serological tests to detect antibody to human T-lymphotropic virus type 111 (HTLV-III) are highly sensitive and specific, according to reports presented at a US Public Health Service Workshop on HTLV-III Antibody Testing on 31 July, 1985. The tests are currently being used at blood banks, plasma collection centres, health departments, and selected clinical centres throughout the United States.

The US Food and Drug Administration reported cumulative HTLV-III antibody test data from more than 1.1 million units of blood collected at 155 centres to 16 June 1985. Of these, 2,831 (0.25%) were reported as positive based on a repeatedly reactive EIA test. The pattern of positive tests varied slightly in different regions of the country and by test kit used.

The Atlanta Region of the American Red Cross (ARC) and CDC reported data from testing more than 51,000 blood donors, of whom 0.23% were repeatedly reactive by the Abbott EIA method. Among the specimens from 106 blood donors with repeatedly reactive tests, 34 (32%) were strongly reactive (ratio of specimen absorbance to cutoff value 7.0 or greater). EIA tests categorised as strongly reactive correlated highly with both positive Western blot tests (94%) and culture for HTLV-III/lymphadenopathy-associated virus (LAV) (56%).

Of 220 donors whose tests were initially reactive and subsequently negative, as well as a random sample of 50 with an initially negative EIA test, none had either a positive Western blot test or positive culture. Among those donors notified and interviewed to date, 16 (89%) of 18 with strongly reactive EIA tests had identifiable risk factors for HTLV-III/LAV infection, while none of 20 with weakly reactive tests had identifiable risk factors.

To determine the sensitivity of the Abbott EIA test in high-risk persons, virus isolations were attempted from homosexual men attending a clinic for sexually transmitted diseases in San Francisco, California. None of 70 men with negative HTLV-III antibody tests had a positive culture, while 43 (60%) of 72 with repeatedly reactive tests were culture-positive. Among the 72 EIA-positive sera in this portion of the study, 70 (97%) were considered to be highly reactive. Ninety-seven percent of those EIA-positive specimens tested to date have had a positive Western blot test.

Data from other blood banking organisations paralleled the findings of the ARC/CDC study in suggesting that approximately one-third of EIA-positive sera from blood donors were strongly reactive, regardless of the test kit used. Donors with strongly reactive EIA tests were also highly likely to have positive Western blot tests and to have positive EIA tests by other test kits.

Weakly reactive EIA tests correlated poorly with positive Western blot tests and were judged to be nonspecific for HTLV-III/LAV infection. The reason for nonspecific test reactivity is unknown, but proposed refinements in the test may eliminate many of the low level reactions.

MMWR Editorial Note

Based on available data, only about 0.25% (1 in 400) blood donors have repeatedly reactive EIA tests to HTLV-III antibody. Approximately 0.08% (1 in 1,200) donors were found to have strongly reactive EIA tests, and these donors were likely to have other test results (Western blot, HTLV-III/LAV culture) that suggested they had been infected with HTLV-III/LAV.

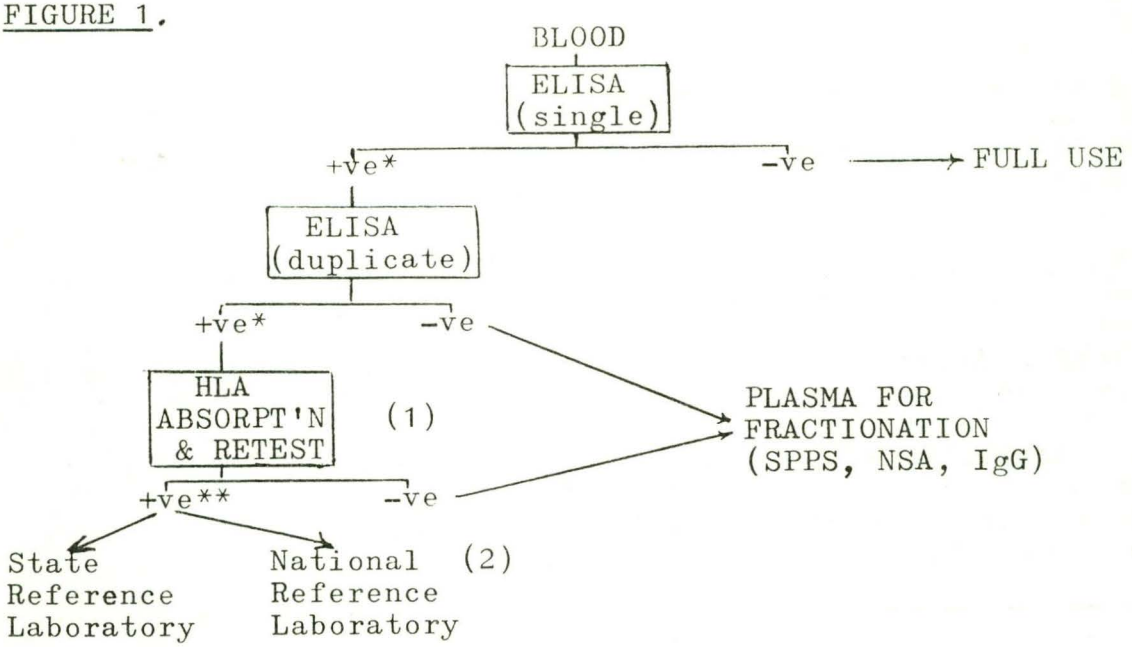
Thus, in less that five months, serological tests for HTLV-III antibody have been introduced and demonstrated to be highly useful in screening donated blood. Screening performed during this period may have removed as many as 1,000 potentially infectious units of blood from the US blood supply. Continued use of this highly sensitive test procedure for HTLV-III antibody, in combination with voluntary avoidance of donation by members of high-risk groups, will virtually eliminate the risk of acquired immune deficiency syndrome (AIDS) transmission by the USA's blood supply. Discussions and evaluations of other potentially appropriate and useful applications of this test are under way.

* EIA = ELISA

Editorial Comment

The National Reference Laboratory for AIDS convened a workshop at the Commonwealth Serum Laboratories on 12-13 August 1985 to review AIDS screening and confirmatory tests. The protocol agreed to by the Blood Transfusion Services was that shown diagramatically below in Figure 1. The workshop noted that 247,000 donations had been screened as of 31 July 1985 and seven confirmed positives had been detected.

FIGURE 1.



(1) As doubts are presently held concerning the reliability of the of the H9 confirmatory test, the HLA (DR4+, DR4-) absorption test is recommended, followed by ELISA testing of the absorbed sera.

(2) National Reference Laboratory is utilised where a State Reference Laboratory is not operative.

* Includes equivocal results.
** Plasma destroyed, second specimen collected.

ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS) - ITALY

(Based on WER (1985) 60: 260

Surveillance of AIDS was established in 1983 to study the geographical and temporal distribution of the disease and obtain data on risk factors and circumstances which may favour transmission of the infection. The surveillance system also provides documentation and bibliographical services for research workers and public health administrators.

As of 23 May 1985, 39 cases of AIDS had been notified. Seven cases of suspected AIDS are currently being investigated, but are not included in this figure as the diagnosis has not yet been verified by the surveillance team. Thirty-five of the 39 cases were in adults aged from 20 to 57 years. Most cases have been notified in the central and northern parts of the country. Twenty-two of the adult cases were homosexual men, 10 were intravenous drug users and one a haemophilac. Two cases did not belong to any known risk group: one was a heterosexual man who had lived a long time in the United States of America; the other a woman originating from a country in equatorial Africa.

Four cases were diagnosed in children between 7 and 27 months. All were children whose mothers were drug abusers. Antibody to LAV/HTLV-III was detected in three of the four children and in all four mothers, who were asymptomatic. A further case of lymphadenopathy syndrome has been notified in an infant whose mother is also a drug abuser.

(continued from page 1)

Other reports of interest include:-

- During July, the OIC WHO Influenza Reference Centre, Melbourne, isolated 24 influenza type A H_3 viruses and four type B. The type A viruses resembled A/Vic/3/85 and the type B, B/Vic/102/85.

- Increased influenza A activity has been observed in New Zealand this year. Virus isolates from March and April have been confirmed to be similar to A/Philippines/2/82 (H_3N_2) by the WHO Influenza Reference Centre, Melbourne, while isolates from June have been shown to have drifted antigenically and are more similar to A/Wellington/3/84(H_3N_2) and A/Vic/3/85(H_3N_2) viruses. Activity peaked about the first week of June in the Christchurch area. At its peak in the Dunedin area, up to 40% of children were reported absent from school and there was widespread absenteeism from the workplace.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 1/8/85 to 14/8/85 BULLETIN NUMBER 85/17
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	1		6		2		10	1	20
0101 ADENOVIRUS TYPE 1				1	1	2		3	7
0102 ADENOVIRUS TYPE 2				2	3	7		1	13
0103 ADENOVIRUS TYPE 3						2			2
0104 ADENOVIRUS TYPE 4								1	1
0105 ADENOVIRUS TYPE 5		1			2	1		2	6
0107 ADENOVIRUS TYPE 7	1					1			2
0108 ADENOVIRUS TYPE 8				1					1
0199 ADENOVIRUS TYPING PENDING		1	2		5	1			9
0201 INFLUENZA A VIRUS	22			13		14	14	19	82
0202 INFLUENZA A VIRUS SUBTYPE H3N2	3	2		20	20	13	4		62
0203 INFLUENZA B VIRUS				11		12	2	14	39
0206 INFLUENZA A VIRUS SUBTYPE H1N1				6		1			7
0299 INFLUENZA VIRUS		4							4
0301 PARAINFLUENZA VIRUS TYPE 1				2		9	2		13
0302 PARAINFLUENZA VIRUS TYPE 2				2				2	4
0303 PARAINFLUENZA VIRUS TYPE 3				1	2	1	1	4	9
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	10	10	2	27	27	91	34	24	225
0500 RHINOVIRUS (ALL TYPES)			1	4	7	4			16
0600 MYCOPLASMA PNEUMONIAE	2			4				1	7
0700 ORNITHOSIS-PSITTACOSIS				5		1	1		7
0816 COXSACKIEVIRUS A16				1					1
0904 COXSACKIEVIRUS B4				2	2	3			7
1003 ECHOVIRUS TYPE 3							1		1
1007 ECHOVIRUS TYPE 7				2					2
1017 ECHOVIRUS TYPE 17				1					1
1021 ECHOVIRUS TYPE 21		1		2					3
1022 ECHOVIRUS TYPE 22		1							1
1030 ECHOVIRUS TYPE 30					1				1
1100 POLIOVIRUS NOT TYPED			3		3				6
1101 POLIOVIRUS TYPE 1		1				3		1	5
1102 POLIOVIRUS TYPE 2				2					2
1104 POLIOVIRUS-VACCINAL STRAIN							3		3
1200 MUMPS VIRUS	1			2					3
1300 HERPES VIRUS GROUP-NOT TYPED	10			3				1	14
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1		1				1	3
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	2	1		1				2	6
1303 VARICELLA-ZOSTER VIRUS	7						2	1	10
1306 HERPES SIMPLEX TYPE 1	12			32		14	35	21	114
1307 HERPES SIMPLEX TYPE 2	53			41	1	16	46	31	188
1399 HERPES VIRUS TYPING PENDING				5	4				9
1401 COXIELLA BURNETI				2		3	3	1	9
1502 PICORNA VIRUS-NOT TYPED	7		10	2			3		22
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)						1			1
1521 MEASLES VIRUS	1						1		2
1522 RUBELLA VIRUS	1			1		1		9	12
1532 HEPATITIS B ANTIGEN	32		10	52		15	12	14	135
1535 HEPATITIS A ANTIBODY	3		1	11*		2		5	22
1541 CHLAMYDIA A - C TRACHOMATIS	32	1		27*		22	20	37	139
1556 CMV - CYTOMEGALOVIRUS	8		2	20	10	6	4	6	56
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	10	7	14	5	24	17	3	3	83
1565 CALICI VIRUS	1								1
1599 ENTEROVIRUS TYPING PENDING			7		5				12
9992 ROSS RIVER VIRUS				1			4	1	6
9993 ASTROVIRUS	6								6
9994 SMALL VIRUS (LIKE) PARTICLE	1	1							2
9995 DENGUE							1		1
Total	226	32	58	315	119	263	206	207	1,426

*** Cultures performed at Microbiological Diagnostic Unit, Melbourne.**

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 1/8/85 to 14/8/85 ....
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 me mb
0100 ADENOVIRUS NOT TYPED.....	1	8					10				
0101 ADENOVIRUS TYPE 1.....	1	4					2				
0102 ADENOVIRUS TYPE 2.....		10					1				
0103 ADENOVIRUS TYPE 3.....							1				
0105 ADENOVIRUS TYPE 5.....		4				1					
0107 ADENOVIRUS TYPE 7.....		1									
0199 ADENOVIRUS TYPING PENDING.....		2									
0201 INFLUENZA A VIRUS.....	7	59	1			1		1	2	1	
0202 INFLUENZA A VIRUS SUBTYPE H3N2	1	55									
0203 INFLUENZA B VIRUS.....	1	29							1		
0206 INFLUENZA A VIRUS SUBTYPE H1N1		7									
0301 PARAINFLUENZA VIRUS TYPE 1....		13									
0302 PARAINFLUENZA VIRUS TYPE 2....		4									
0303 PARAINFLUENZA VIRUS TYPE 3....		8									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	217									
0500 RHINOVIRUS (ALL TYPES).....		15									
0600 MYCOPLASMA PNEUMONIAE.....		7									
0700 ORNITHOSIS-PSITTACOSIS.....	1	6									
0904 COXSACKIEVIRUS B4.....	1	4									1
1003 ECHOVIRUS TYPE 3.....		1									
1007 ECHOVIRUS TYPE 7.....				1							
1021 ECHOVIRUS TYPE 21.....		1		1			1				
1030 ECHOVIRUS TYPE 30.....		1									
1100 POLIOVIRUS NOT TYPED.....							3				
1101 POLIOVIRUS TYPE 1.....		4					1				
1102 POLIOVIRUS TYPE 2.....		1		1							
1300 HERPES VIRUS GROUP-NOT TYPED..	5					1					6
1301 HERPES SIMPLEX VIRUS NOT-TYPED					1						1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	2							1			
1303 VARICELLA-ZOSTER VIRUS.....					1					1	9
1306 HERPES SIMPLEX TYPE 1.....		12				1				2	52
1307 HERPES SIMPLEX TYPE 2.....	7										
1401 COXIELLA BURNETI.....	1	3					1				
1502 PICORNA VIRUS-NOT TYPED.....	1	2				2	11		2	1	1
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											1
1521 MEASLES VIRUS.....		1					1				1
1522 RUBELLA VIRUS.....											3
1532 HEPATITIS B ANTIGEN.....	72							45			
1535 HEPATITIS A ANTIBODY.....	2							19			
1541 CHLAMYDIA A - C.TRACHOMATIS...	4	1									
1556 CMV - CYTOMEGALOVIRUS.....	1	19					3			7	1
1563 CORONAVIRUS.....		1									
1564 ROTAVIRUS.....	1						82				
1565 CALICI VIRUS.....							1				
9992 ROSS RIVER VIRUS.....	2	1									
9993 ASTROVIRUS.....							6				
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
Total.....	113	501	1	3	2	6	126	66	5	12	129

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 1/8/85 to 14/8/85 ...
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			
0102 ADENOVIRUS TYPE 2.....	1									1
0103 ADENOVIRUS TYPE 3.....	1									
0104 ADENOVIRUS TYPE 4.....		1								
0105 ADENOVIRUS TYPE 5.....										1
0107 ADENOVIRUS TYPE 7.....	1									
0108 ADENOVIRUS TYPE 8.....	1									
0201 INFLUENZA A VIRUS.....					1			14	2	
0202 INFLUENZA A VIRUS SUBTYPE H3N2					1		1	12	2	
0203 INFLUENZA B VIRUS.....					1		3	8	1	
0206 INFLUENZA A VIRUS SUBTYPE H1N1								3		
0301 PARAINFLUENZA VIRUS TYPE 1....								1		
0303 PARAINFLUENZA VIRUS TYPE 3....									1	
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1						2	5		2
0500 RHINOVIRUS (ALL TYPES).....									1	
0816 COXSACKIEVIRUS A16.....									1	
0904 COXSACKIEVIRUS B4.....							1	1		
1007 ECHOVIRUS TYPE 7.....								1		
1017 ECHOVIRUS TYPE 17.....									1	
1021 ECHOVIRUS TYPE 21.....								1		
1022 ECHOVIRUS TYPE 22.....								1		
1100 POLIOVIRUS NOT TYPED.....										3
1104 POLIOVIRUS-VACCINAL STRAIN....										3
1200 MUMPS VIRUS.....			3							
1300 HERPES VIRUS GROUP-NOT TYPED..	1	1					1			
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			1					1	1	
1303 VARICELLA-ZOSTER VIRUS.....					1					
1306 HERPES SIMPLEX TYPE 1.....	3	43						1	1	
1307 HERPES SIMPLEX TYPE 2.....		131								
1401 COXIELLA BURNETI.....							1	5		
1502 PICORNA VIRUS-NOT TYPED.....	2				1					
1521 MEASLES VIRUS.....								1		
1522 RUBELLA VIRUS.....				2	4				4	
1532 HEPATITIS B ANTIGEN.....					1				17	
1535 HEPATITIS A ANTIBODY.....									1	
1541 CHLAMYDIA A - C.TRACHOMATIS...		133							1	
1556 CMV - CYTOMEGALOVIRUS.....			4			4	2	3	12	2
9992 ROSS RIVER VIRUS.....					3			1		
9995 DENGUE.....								1		
Total.....	12	310	8	2	13	4	12	60	46	12