AUSTRALIA

# Communicable Diseases Intelligence

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Contents:



VIRUS REPORTING SCHEME - Since there was no publication of the CDI over Christmas-New Year, this issue contains a compilation of the virus reports for the two periods, 11 December to 24 December 1980, and 25 December to 7 January 1981 (i.e. four weeks).

Complete computer tabulation of the 1980 data reported to the CDI is being prepared, and will be sent to contributing laboratories. Other readers requiring copies should write to the Editor.

A total of 1187 reports were received for the two periods.

Reports of interest include:

- Two arbovirus group B infections, both clinically dengue, were reported by Fairfield Hospital, Melbourne, in a 25 year old man and a 20 year old woman who had travelled together in Sri Lanka, India, Burma and Thailand. Both had been referred as outpatients for the investigation of diarrhoea. Giardia lamblia and Campylobacter fetus subspecies jejuni were detected in faecal specimens of both patients. In addition, Shigella sonnei was isolated from the man, and Salmonella hvittingfos from the woman.
- A fatal case of Legionnaires' disease was diagnosed in an eight year old child at the Princess Margaret Hospital, Perth. Laboratory confirmation was made at the State Health Laboratory, by immunofluoresence on a lung biopsy.
- Kawasaki disease was diagnosed in a two year old girl at the Royal Alexander Hospital for Children, Sydney. The patient gave a serological response by CF to parainfluenza virus type 3.
- E.coli bacteremia was diagnosed at Fairfield Hospital, Melbourne, in a 66 year old man presenting with a four week history of intermittent fever. The organism was isolated from a bone-marrow aspirate and blood culture.
- Salmonellosis was diagnosed by the Enteric Diseases Laboratory, Perth, in sixty health surveyors and workers from the Health Education Department. Undercooked pork was the suspected source. S. give was isolated from eight patients, S. ball from five patients, and two patients excreted both serotypes.

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## ADVERSE REACTIONS TO HUMAN DIPLOID CELL RABIES VACCINE

(Based on MMWR (1980) 29:609)

The Merieux human diploid cell rabies vaccine (M-HDCV) has been available in Australia for rabies post-exposure prophylaxis for the past two years. The vaccine has largely replaced duck embryo vaccine because of the higher levels of antibody stimulated by fewer doses of vaccine, and fewer adverse reactions. This article on the safety and efficacy of M-HDCV is an appendix to the recommendations for vaccine use detailed in CDI 80/16. In a 12 week period from 23 June 1980, approximately 25 200 doses of Merieux human diploid cell rabies vaccine (M-HDCV) were distributed in the United States. About 2 500 people who had received prophylaxis with HDCV during this time were surveyed by the CDC for adverse reactions.

Four patients (one per 625 treated) had systemic allergic reactions ranging from hives to anaphylactic shock. Although two of the patients reported allergies to other drugs in the past, the other two gave no such history. Two of the cases were complicated by simultaneous administration of human rabies immune globulin or tetanus toxoid. In two of the cases however, repeated administration of the vaccine alone resulted in the appearance of the adverse reaction.

Four cases of fever and severe headache (one per 625 treated) were seen. The febrile headaches were not associated with stiff neck or other signs of meningitis or encephalitis. The symptoms characteristically resolved within 24 hours, and occasionally, but not invariably, recurred following additional injections of M-HDCV.

Other systemic reactions occasionally reported were chills, diarrhoea, malaise, headache without fever, and fever without headache. Local reactions, affecting fewer than 25% of persons treated, consisted of redness, swelling or pain at the site of injection.

Although two of the persons were admitted to hospital or observed by a physician because of severe allergic reactions during administration of successive doses of vaccine, in no instance was it necessary to discontinue the post-exposure prophylaxis regimen.

The adverse reactions noted in the above survey have been similar to those described in European trials[1,2]. However, a single case of Guillain-Barré syndrome was observed two weeks after the second prophylactic dose of M-HDCV in a 14 year old Norwegian boy living in Zambia[3]. The patient recovered. A cause-effect relationship between Guillain-Barré syndrome and M-HDCV administration was not established.

No deaths, or cases of rabies or encephalopathy have been reported in the United States following vaccination with M-HDCV. In addition, there have been no documented cases of failure to develop protective antibody when the five dose post-exposure prophylaxis regimen has been adhered to. (In Australia, a sixth 'booster' dose is recommended 90 days after initiation of vaccination[4]).

References

1. Lancet (1975) 1:660
2. Dev. Biol. Stand.(1978)40:101
3. Scand. J. Infect. Dis. (1980) 12:231
4. CDI (1980) 80/16

## LEPTOSPIROSIS - VICTORIA

(Based on Fairfield Hospital monthly report, November 1980.)

Three leptospiral infections were confirmed at Fairfield Hospital, Melbourne, during November:

- A 64 year old man was admitted with a four day history of fever, rigors, severe headache and myalgia. On examination he was febrile, and had marked conjunctival suffusion. Cerebrospinal fluid examination was normal. Leptospirosis was considered the likely diagnosis on epidemiological grounds, since the patient had been working on the dairy farm of his son who was recovering from a similar illness. Initial blood and cerebrospinal fluid samples were inoculated into leptospiral EMJH culture medium, and after one week the blood cultures were positive for leptospirae under dark ground microscopy. This is only the second isolation of the organisms from blood cultures at Fairfield Hospital, since isolation requires specimens taken during the first few days of illness (normally prior to hospital admission) and definitive culture conditions.

- A 15 year old boy developed leptospirosis as a result of contact with cattle on his father's weekend farm. He was admitted with fever, headache, enlarged glands and a low white cell count. No antibiotics were given, and his fever took 12 days to resolve. Diagnosis was confirmed serologically, but the serotype has not yet been determined.

- A 47 year old dairy farmer presented with a five day history of fever with rigors, severe headache and myalgia. Cerebrospinal fluid examination on the third day of illness was normal. On admission he was noted to be afebrile and mildly jaundiced, hepatosplenomegaly and skin petechiae were present, and he complained of severe headache. Repeated cerebrospinal fluid examination showed an aseptic meningitis with xanthochromia, 650 leucocytes/cmm (93% polymorphs), 580 red cells/cmm, a total protein reading of 1405 mg/l and a normal sugar level. His renal function deteriorated with a serum creatinine of 0.63 m mol/l and a blood urea of 43.2 m mol/l, but this, together with his liver dysfunction, later returned to normal with clinical recovery. Serum samples showed seroconversion to Leptospira hardjo to a titre of 1:8192.

Most cases of leptospirosis present without jaundice, either as a self-limiting febrile illness or with an aseptic meningitis. The last case indicates that leptospirosis can occasionally produce severe disease in man.

## GASTRO-ENTERITIS OR LACTOSE INTOLERANCE

(Based on CDWR (1980) 6:238)

Gastro-enteritis is caused not only by a variety of infectious agents, but may be the result of an enzyme deficiency which prevents proper digestion of the disaccharide sugar, lactose. This lactose intolerance is classified as primary, secondary or congenital. If there is no underlying intestinal disease, the syndrome is defined as primary, and if there are underlying problems, as secondary. Congenital lactose intolerance (alactasia) is rare[1]. Since the gastrointestinal upsets resulting from lactose intolerance can mimic food poisoning, not only doctors, but also persons involved in the

follow-up of food complaints, should be aware of the syndrome.

Most children and many adults consume milk and milk products which contain lactose, e.g. cow's milk with 4-5% lactose and whey powder with 65% lactose. Utilisation of the sugar is normally through the action of the enzyme lactase. However, after weaning many persons gradually decrease its production. The lack of lactase (hypolactasia) subsequently prevents them from digesting lastose, and gastrointestinal symptoms such as gas, bloating, abdominal pain, indigestion, excessive flatulence and diarrhoea often result. The condition is caused by acids and other biproducts made by the gu flora fermenting the unutilised lactose. Symptoms usually develop one to three hours after ingestion, although the incubation period may be as short as 30 minutes or as long as 12 hours.

It is estimated that 33 million persons have low lactase levels in the United States alone. Racial or ethnic background seems to be an important factor. Those whose ancestry stems from northern Europe suffer the least from the condition (10-15% of the population), while Orientals, Indians, Arabs, Jews, Negroes, Eskimos, American Indians and Australian Aboriginals are much more prone to hypolactasia (44-100%). The apparent genetic variation in lactose tolerance probably results from the consumption of milk over many centuries, as has occurred in Northern Europe, so that a large proportion of the population continues to produce lactase after weaning(2).

Patients with hypolactasia who know that drinking milk can create problems usually reduce their consumption. Symptoms may be dose related, so that one person can consume one litre of milk with no symptoms, while another cannot drink any.

However, many individuals suffering from chronic gastro-enteritis are unawar of their enzyme deficiency, and may consult a physician who does not recognize the condition. Lactose intolerance may be suspected if:-

- There is a family history of milk-related problems.
- The patient belongs to a high prevalence racial or ethnic group.
- The patient has been drinking more milk recently, e.g. a milk diet prescribed for a peptic ulcer condition, or the consumption of a large quantity of lactose at once, such as more milk or ice cream on a hot day.
- The case is an individual attack of gastro-enteritis, and not the result of a number of people becoming ill after a foodborne out-break.
- The short period of time between the consumption of milk and the appearance of symptoms.
- The existence of an unrelated intestinal problem which may be aggrevated by hypolactasia.

If lactose intolerance is suspected, the easiest test is to have the patient avoid milk or milk products for two weeks. In addition to cottage cheese, yogurt, ice cream and other dairy products, certain non-dairy products, such as baked goods, may contain lactose in the form of whey powder or skim milk powder. If free of intestinal problems during that time

confirmation of hypolactasia can be achieved by the onset of symptoms within a few hours after drinking two large glasses of milk, and further specific biochemical tests. The protein component of milk may cause allergies unrelated to hypolactasia, but these can be distinguished by giving the patient pure lactose.

For most people suffering from hypolactasia the treatment is avoidance of lactose-containing products. For manufactured foods, labels listing ingredients should be carefully checked for lactose or lactose-containing components. Individuals with some tolerance should be encouraged to experiment with various types and amounts of food to determine their normal acceptable limits. However, travelling often presents problems, since a lactose-free diet can rarely be obtained on aircraft or in restaurants, and brands of food will probably be different from those available at home. Microbial lactase enzyme can be added to milk which is incubated for about 24 hours at refrigerated temperatures and then consumed, or taken as tablets during the eating of lactose-containing products. Further information on lactose intolerance can be obtained from the listed references[3-6].

Editorial Comment

The incidence of lactose intolerance in Australians of European descent appears to be low[7], while in Aboriginal children the incidence appears to be as high as 80%[8]. However, Aboriginal children have a high incidence of malnutrition and gastrointestinal infections and infestations[9] which are known to predispose to secondary lactose intolerance. Studies indicate that the condition is usually temporary, and the true incidence of lactase deficiency in older Aboriginal children and adults is not known. In addition, the wider use of pre-hydrolysed low lactose milk[10] and preparations of dairy based carbohydrate-free formulas designed to provide similar fat and protein content to milk[11], have facilitated the planning of nutritional rehabilitation programs. Further cases of this syndrome may be seen by doctors and health workers because of the increasing numbers of Vietnamese and immigrants from other high prevalence areas settling in Australia.

References


1. MJA (1972) ii:431
2. Scientific American (1972) 227:71
3. BMJ (1975) 2:351
4. NEJM (1975) 292:1156
5. J. Milk Food Tech. (1972) 35:32
6. Patient Care (1976) 10:116
7. Aust. Ann. Med. (1968) 17:300
8. MJA (1967) i:46
9. Aust. N.Z. J. Med. (1973) 3:576
10. MJA (1977) ii (Supplement):10
11. Aust. Paed. J. (1974) 10:164


## HEPATITIS A OUTBREAK IN A DAY-CARE CENTRE - U.S.A.

(Based on MMWR (1980) 29:565)

From 11 January to 22 May 1980 an outbreak of hepatitis A, involving one suspected and 17 confirmed cases, occurred in a child-care centre in Texas. The index case was a five year old child, followed by infection in the

director of the centre two weeks later. Over the next 14 weeks, two other children, an employee of the centre, and 13 household contacts developed hepatitis. The centre was kept open, but took no new admissions until the outbreak had terminated. The centre employees and parents of children were recommended to receive human normal immunoglobulin (NIGH) prophylaxis. There was no evidence of spread of the outbreak, even though some parents transferred their children to other day-care centres in that period.

In the USA, between 9-12% of hepatitis A infections, or hepatitis, type unspecified, are estimated to be day-care related[1].

Characteristics of such outbreaks are:

- Hepatitis in children of day-care age is usually asymptomatic.
- Household contacts are often infected as a consequence of spread within a centre, and generally constitute the majority of recognized cases.
- Children in nappies age two years or less are most likely to transmit infection to household contacts; children age four and older rarely spread infection beyond the centre.

Detection of outbreaks is therefore dependent upon centre directors, parents and health authorities recognizing that cases in families of children attending a day-care centre may indicate asymptomatic transmission among children at that centre.

The presence of children in nappies is a primary factor in facilitating transmission, and prevention and control of infection should focus on this group. Appropriate hygienic standards should be endorsed, particularly in the washing of hands of staff and of young children who cannot wash themselves adequately. Changing surfaces should be impermeable and should be regularly cleaned and disinfected. A fresh solution of a 1:32 dilution (½ cup per gallon) of household bleach in tap water is a suitable disinfectant. Accessory items (e.g. cans of baby powder, jars of vaseline) should also be disinfected daily, as they can be inadvertently soiled during changing.

Overall control measures include the suspension of new admissions, or they should receive prophylactic NIGH before entering. Centres need not be closed. Parents should be discouraged from transferring children to other centres.

The efficacy of administering NIGH in such outbreaks is being evaluated, but it would appear that even the presence of one child or employee of a centre that has children under two years, necessitates the recommendation of NIGH prophylaxis to all centre children and employees. In addition, any new children admitted to the centre within six weeks of the last case should receive NIGH. Where cases are recognized simultaneously in multiple families at a single centre, administration of NIGH to household contacts of children age three or less might be considered. In those centres having only children age two and older, spread is less likely after an index infection, and NIGH should be considered only for centre staff and for age-group contacts of the infected child.

Reference

1. CDC Hepatitis surveillance report no.45. May 1980

# AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

1

REPORTING PERIOD - 11-12-80 - 7-1-81 BULLETIN NUMBER - 81/1

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 | 5 | 1 | 5 | | | 5 | 7 | | 23 |
| 0101 ADENOVIRUS TYPE 1 | | 1 | 1 | 1 | 2 | 6 | | 1 | 12 |
| 0102 ADENOVIRUS TYPE 2 | | | | 2 | 2 | 6 | | 4 | 14 |
| 0103 ADENOVIRUS TYPE 3 | | | | | | | | 1 | 1 |
| 0105 ADENOVIRUS TYPE 5 | 1 | | | | | | | 2 | 3 |
| 0106 ADENOVIRUS TYPE 6 | | | | | | 4 | | | 4 |
| 0107 ADENOVIRUS TYPE 7 | | | | | | 1 | | | 1 |
| 0119 ADENOVIRUS TYPE 19 | | | | | | 3 | | 14 | 17 |
| 0199 ADENOVIRUS TYPING PENDING | | 1 | | | 8 | 3 | | | 12 |
| 0201 INFLUENZA A VIRUS | 2 | | 6 | 2 | | | 1 | | 11 |
| 0203 INFLUENZA B VIRUS | | | 2 | 3 | 1 | | | | 6 |
| 0303 PARAINFLUENZA VIRUS TYPE 3 | 1 | 5 | | | 11 | | 3 | 2 | 22 |
| 0399 PARAINFLUENZA VIRUS TYPING PENDING | | | | | | 2 | | 1 | 3 |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS) | 1 | 2 | | 2 | 7 | 5 | 3 | 2 | 22 |
| 0500 RHINOVIRUS (ALL TYPES) | | | | 4 | 4 | 7 | 11 | | 26 |
| 0600 MYCOPLASMA PNEUMONIAE | 6 | | 17 | 4 | | | 7 | 3 | 37 |
| 0700 ORNITHOSIS-PSITTACOSIS | 1 | | | 1 | | | | | 2 |
| 0800 COXSACKIEVIRUSES GROUP A - NOT TYPED | | | | | | | | 2 | 2 |
| 0809 COXSACKIEVIRUS A9 | | | | 2 | | | 3 | 1 | 6 |
| 0816 COXSACKIEVIRUS A16 | 1 | | | 2 | | | 3 | | 6 |
| 0901 COXSACKIEVIRUS B1 | | | | | 1 | | | | 1 |
| 0902 COXSACKIEVIRUS B2 | | | | 2 | 1 | 2 | | 1 | 6 |
| 0904 COXSACKIEVIRUS B4 | | 2 | | | | | | | 2 |
| 0905 COXSACKIEVIRUS B5 | | | | | | | | 1 | 1 |
| 1002 ECHOVIRUS TYPE 2 | | | | | | | | 1 | 1 |
| 1009 ECHOVIRUS TYPE 9 | 6 | | | 2 | | 4 | | | 12 |
| 1014 ECHOVIRUS TYPE 14 | 1 | | 2 | | 1 | | | | 4 |
| 1019 ECHOVIRUS TYPE 19 | | | | | | 1 | | | 1 |
| 1022 ECHOVIRUS TYPE 22 | | | | | 5 | | | | 5 |
| 1027 ECHOVIRUS TYPE 27 | 3 | | | | | | | | 3 |
| 1030 ECHOVIRUS TYPE 30 | 2 | | 1 | 3 | | | | | 6 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 11 - 12 - 80 - 7 - 1 - 81 BULLETIN NUMBER - 81/1

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 |
|---|---|---|---|---|---|---|---|---|---|
| 1033 ECHOVIRUS TYPE 33 | 1 | | | | | | | | 1 |
| 1101 POLIOVIRUS TYPE 1 | | | | | | | | 1 | 1 |
| 1102 POLIOVIRUS TYPE 2 | | | | | | | 1 | 1 | 2 |
| 1103 POLIOVIRUS TYPE 3 | | | | | | | 1 | | 1 |
| 1200 MUMPS VIRUS | | 1 | 1 | 7 | 1 | 5 | 1 | | 16 |
| 1300 HERPES VIRUS GROUP-NOT TYPED | 10 | | 5 | 3 | | 7 | | | 25 |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | 13 | 2 | | | 1 | 1 | | 84 | 101 |
| 1303 VARICELLA-ZOSTER VIRUS | 2 | | 5 | 3 | | | 2 | | 12 |
| 1306 HERPES SIMPLEX TYPE 1 | 3 | | 6 | 60 | | 16 | 15 | | 100 |
| 1307 HERPES SIMPLEX TYPE 2 | 59 | | 1 | 41 | | 15 | 26 | | 142 |
| 1399 HERPES VIRUS TYPING PENDING | | | 4 | | 2 | 4 | | | 10 |
| 1401 COXIELLA BURNETI | 12 | | 2 | 2 | | | 19 | | 35 |
| 1502 PICORNA VIRUS-NOT TYPED | 2 | | | | | | | 2 | 4 |
| 1514 MOLLUSCUM CONTAGIOSUM | | | | | | 2 | | | 2 |
| 1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS) | | | | | | | | 1 | 1 |
| 1521 MEASLES VIRUS | 3 | 3 | 4 | | | | 1 | 5 | 16 |
| 1522 RUBELLA VIRUS | 11 | | 1 | 14 | | | 6 | 11 | 43 |
| 1531 HEPATITIS B VIRUS | | | | 1 | | | | | 1 |
| 1532 HEPATITIS B ANTIGEN | 18 | | 16 | 46 | | | 10 | 6 | 96 |
| 1535 HEPATITIS A ANTIBODY | 3 | | 3 | 18 | | | | 12 | 36 |
| 1541 CHLAMYDIA A - TRIC TYPE | 14 | | 6 | | | | | 123 | 143 |
| 1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART) | 1 | | | | | | | | 1 |
| 1556 CMV - CYTOMEGALOVIRUS | 6 | | 8 | 35 | 5 | | 3 | 3 | 60 |
| 1564 ROTAVIRUS | 6 | | 3 | | | 12 | | | 21 |
| 1599 ENTEROVIRUS TYPING PENDING | | 4 | 5 | | 16 | 4 | 4 | | 33 |
| ROSS RIVER VIRUS | | | | | | | 3 | | 3 |
| ASTROVIRUS | 2 | | | | | | | | 2 |
| SMALL VIRUS (LIKE) PARTICLE | 3 | | | | | | | | 3 |
| ARBO. GROUP B. | | | | 3 | | | | | 3 |
| Total | 200 | 22 | 104 | 263 | 68 | 115 | 130 | 285 | 1,187 |

# AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 3.

**PERIOD :** 11/12/80 **to** 7/1/81 .... 81/1

**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 | 5 | | | | | 5 | | | | |
| 0101 ADENOVIRUS TYPE 1..... | | 2 | | | | | 7 | | | | 1 |
| 0102 ADENOVIRUS TYPE 2..... | 1 | 1 | | | | 3 | 4 | | | | 1 |
| 0103 ADENOVIRUS TYPE 3..... | | | | | | | 1 | | | | |
| 0105 ADENOVIRUS TYPE 5..... | | | | | | 1 | 1 | | | | |
| 0106 ADENOVIRUS TYPE 6..... | | 1 | | | | | 2 | | | | |
| 0107 ADENOVIRUS TYPE 7..... | | | | | | | 1 | | | | |
| 0119 ADENOVIRUS TYPE 19..... | 13 | | | | | | | | | | |
| 0201 INFLUENZA A VIRUS..... | 1 | 2 | | | | | | | | | |
| 0203 INFLUENZA B VIRUS..... | 1 | 5 | | | | | | | | | |
| 0303 PARAINFLUENZA VIRUS TYPE 3.... | 1 | 17 | | | | | | | | | 1 |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS)..... | | 20 | | | | 1 | | | 1 | | |
| 0500 RHINOVIRUS (ALL TYPES)..... | 2 | 24 | | 1 | | | | | | | |
| 0600 MYCOPLASMA PNEUMONIAE..... | 3 | 26 | | | | | | | 1 | | |
| 0700 ORNITHOSIS-PSITTACOSIS..... | | 2 | | | | | | | | | |
| 0800 COXSACKIEVIRUSES GROUP A - NOT TYPED..... | | | | | | 1 | | | | | 1 |
| 0809 COXSACKIEVIRUS A9..... | | 2 | | 3 | | | | | | | 2 |
| 0816 COXSACKIEVIRUS A16..... | | 1 | | | | | | | | | 6 |
| 0901 COXSACKIEVIRUS B1..... | | | | | | | 1 | | | | |
| 0902 COXSACKIEVIRUS B2..... | | | | 1 | | | 2 | | 1 | | |
| 0904 COXSACKIEVIRUS B4..... | | | | 2 | | | | | | | |
| 0905 COXSACKIEVIRUS B5..... | | | | | | | | | 1 | | |
| 1009 ECHOVIRUS TYPE 9..... | | 2 | 1 | 3 | | | 4 | | | | |
| 1014 ECHOVIRUS TYPE 14..... | 1 | | | | | | 2 | | 1 | | |
| 1019 ECHOVIRUS TYPE 19..... | | | | | | | | | | | 1 |
| 1022 ECHOVIRUS TYPE 22..... | 5 | | | | | | | | | | |

# AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

4.

PERIOD : 11/12/80 to 7/1/81 .... 81/1

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; 83 -Encephalitis; 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 | Respiratory | Encephalitis | Meningitis | Paralysis | CNS other unspec | GI | Hepatic | CVS | Urinary | Skin/mucs memb |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1027 ECHOVIRUS TYPE 27..... | 1 | | | 2 | | | | | | | |
| 1030 ECHOVIRUS TYPE 30..... | | | 1 | 4 | | | 1 | | | | |
| 1033 ECHOVIRUS TYPE 33..... | | | | | | | 1 | | | | |
| 1101 POLIOVIRUS TYPE 1..... | | | | | | 1 | | | | | |
| 1102 POLIOVIRUS TYPE 2..... | 1 | 2 | | | | | | | | | |
| 1103 POLIOVIRUS TYPE 3..... | 1 | | | | | | | | | | |
| 1200 MUMPS VIRUS..... | 2 | | 3 | 4 | | | | | | | |
| 1300 HERPES VIRUS GROUP-NOT TYPED.. | 4 | | | | 1 | | | 1 | | | 5 |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | 26 | 2 | 1 | | | | | | 1 | | 69 |
| 1303 VARICELLA-ZOSTER VIRUS..... | 2 | | | | | | | | | | 9 |
| 1306 HERPES SIMPLEX TYPE 1..... | | 9 | 2 | 1 | | | | 1 | | 2 | 43 |
| 1307 HERPES SIMPLEX TYPE 2..... | | | | | | | | | | | 13 |
| 1401 COXIELLA BURNETI..... | 11 | 3 | | | | | | 1 | | | |
| 1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)..... | | | | | | | | | | | 1 |
| 1521 MEASLES VIRUS..... | 1 | | | 1 | | | | | | | 12 |
| 1522 RUBELLA VIRUS..... | 5 | | | | | | | | | | 34 |
| 1532 HEPATITIS B ANTIGEN..... | 38 | | | | | | 2 | 51 | | | |
| 1535 HEPATITIS A ANTIBODY..... | | | | | | | | 35 | | | |
| 1541 CHLAMYDIA A - TRIC TYPE..... | 120 | | | | | | | | 1 | | |
| 1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART)..... | | | | | | | | | | | 1 |
| 1556 CMV - CYTOMEGALOVIRUS..... | 10 | 7 | | | | 2 | | 2 | 2 | 14 | 1 |
| 1564 ROTAVIRUS..... | 1 | | | | | | 19 | | | | |
| ROSS RIVER VIRUS .... | 1 | | | | | | | | | | |
| ASTROVIRUS .... | | | | | | | 2 | | | | |
| SMALL VIRUS (LIKE) PARTICLE .... | | | | | | 1 | 1 | | | | |
| Total..... | 253 | 133 | 8 | 22 | 1 | 10 | 56 | 91 | 9 | 16 | 202 |

5.

# AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

81/1

PERIOD : 11/12/80 to 7/1/81 ...
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 |
|---|---|---|---|---|---|---|---|---|---|---|
| 0100 ADENOVIRUS NOT TYPED..... | 2 | | 1 | | | | 1 | 2 | 1 | |
| 0101 ADENOVIRUS TYPE 1..... | | | | | | | | 1 | | 1 |
| 0102 ADENOVIRUS TYPE 2..... | | | | | | 1 | 2 | 2 | | 1 |
| 0103 ADENOVIRUS TYPE 3..... | | | | | | | | 1 | | |
| 0105 ADENOVIRUS TYPE 5..... | | | | | | | | 2 | | |
| 0106 ADENOVIRUS TYPE 6..... | 1 | | | | | | | | | |
| 0119 ADENOVIRUS TYPE 19..... | 3 | | | | | | | | | |
| 0201 INFLUENZA A VIRUS..... | 1 | | | | 1 | | 2 | 2 | 1 | |
| 0203 INFLUENZA B VIRUS..... | | | | | | | | 2 | | |
| 0303 PARAINFLUENZA VIRUS TYPE 3.... | | | | | | | 1 | 1 | | |
| 0500 RHINOVIRUS (ALL TYPES) ..... | | | | | | | | 1 | | |
| 0600 MYCOPLASMA PNEUMONIAE..... | | | 1 | | | | 1 | 4 | 1 | |
| 0800 COXSACKIEVIRUSES GROUP A - NOT TYPED..... | | | | | | | | 1 | | |
| 0809 COXSACKIEVIRUS A9..... | | | | | | | | 2 | | |
| 0902 COXSACKIEVIRUS B2..... | | | | | | | | 2 | 1 | |
| 1002 ECHOVIRUS TYPE 2..... | | | | | | | | | | 1 |
| 1009 ECHOVIRUS TYPE 9..... | | | | | | | 2 | 1 | | |
| 1200 MUMPS VIRUS..... | | | 7 | | | | | | 1 | |
| 1300 HERPES VIRUS GROUP-NOT TYPED.. | | 4 | | | | | | 1 | | |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | 1 | 1 | | | | | | | | |
| 1306 HERPES SIMPLEX TYPE 1..... | 11 | 24 | | | | | 3 | 5 | 2 | |
| 1307 HERPES SIMPLEX TYPE 2..... | | 12o | | | | | | | | |
| 1401 COXIELLA BURNETI..... | | | | | 1 | | 5 | 15 | 1 | |
| 1514 MOLLUSCUM CONTAGIOSUM..... | | 2 | | | | | | | | |
| 1521 MEASLES VIRUS..... | | | | | | | 1 | 2 | | |
| 1522 RUBELLA VIRUS..... | | | 3 | 2 | 1 | | | 4 | 2 | |
| 1532 HEPATITIS B ANTIGEN..... | | | | | 1 | | | 1 | | |

**AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE** 6.

**PERIOD : 11/12/80 to 7/1/81 ...** 81/1

**Viral Identifications by Clinical Information Table 2.**
**Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;**
**38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;**
**G8 -Fever/malaise; 09 -Other; A1 -SIDS ...** **-CONTINUED**

| VIRUS OR VIRAL ANTIGEN | Eye | Genital | Endo/sal gland | RES | Muscle/joint | Congenital | PUO | Fever/malaise | Other | SIDS |
|---|---|---|---|---|---|---|---|---|---|---|
| 1535 HEPATITIS A ANTIBODY..... | | | | | | | | 1 | | |
| 1541 CHLAMYDIA A - TRIC TYPE..... | 3 | 18 | | | | | | | 1 | |
| 1556 CMV - CYTOMEGALOVIRUS..... | | 1 | 1 | 5 | | 2 | 2 | 5 | 10 | 1 |
| 1564 ROTAVIRUS..... | | 1 | | | | | | | | |
| ROSS RIVER VIRUS .... | | | | | 2 | | | | | |
| SMALL VIRUS (LIKE) PARTICLE .... | | | | | | | | 1 | | 1 |
| ARBO. GROUP B. ... ..... | | | | | | | | 3 | | |
| Total..... | 22 | 177 | 13 | 7 | 6 | 3 | 20 | 62 | 21 | 5 |