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# Communicable Diseases Intelligence

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Contents:



VIRUS REPORTING SCHEME - Since the CDI was not published over the Christmas - New Year period, this issue contains a compilation of the virus reports for the two generations 6-19 December 1984 and 20 December - 2 January 1985. A total of 1813 reports were received for the two periods. Patterns suggested by the reports continue to indicate moderate rubella activity in New South Wales, South Australia and Western Australia. IgM antibody to rubella was detected in three patients aged 12, 16 and 20 years with encephalitis at the Institute of Clinical Pathology and Medical Research, Sydney, and in a neonate with congenital cataracts, hepatomegaly, jaundice, splenomegaly and hypoglycaemia at the State Health Laboratory Services, Perth.

. The majority of *C. trachomatis* diagnoses performed at the State Health Laboratory, Brisbane, are now done by the rapid direct immunofluorescent test (Syva Microtrack; Chlamydia Direct Specimen Test) rather than by culture. A smear of columnar epithelial cells from the patient is rolled onto an 8mm diameter clear area on a prepared slide. Following the transfer of the cellular debris, containing elementary bodies and a few intact cells with inclusion bodies, the slide is air dried and fixed with acetone. A single monoclonal antibody fluorescent conjugate preparation (containing Evans blue counterstain) against the outer wall of the elementary body is added. The slides are then washed, mounted and examined under buffered glycerol. This rapid procedure has demonstrated considerable application in the more remote regions, particularly because of the ease of specimen collection.

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(continued from page 5)

addition, the rate of AIDS for HB vaccine recipients in CDC vaccine trials among homosexually active men in Denver and San Franscisco does not differ from that for men screened for possible participation in the trials, but who received no HB vaccine because they were found immune to HB.

References


1. Science (1984) 225 : 69
2. Medical Sci. (1980) 77 : 7415
3. Science (1982) 218 : 571
4. MMWR (1984) 33 : 589
5. NEJM (1984) 311 : 1030


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## MULTIPLY-RESISTANT HAEMOPHILUS INFLUENZAE - SOUTH AUSTRALIA

(Contributed by D. Hansman and K. Forsyth, Microbiology Department, Adelaide Children's Hospital, Adelaide).

Cases of meningitis caused by multiply-resistant Haemophilus influenzae type b (Hib) have occurred recently in South Australia; the isolates being resistant to ampicillin and chloramphenicol and inactivating both drugs. Since such strains may be more widespread than is at present realised, and because prompt and effective therapy is mandatory, cases of bacterial meningitis should be treated from the outset with a regimen which includes a drug such as moxalactam which is known to be effective against multiply-resistant Hib.

Haemophilus meningitis, the commonest form of bacterial meningitis affecting children in Australia (and in many other regions[1]), is usually fatal if untreated. Currently, treatment is usually with either ampicillin or chloramphenicol. Some paediatricians prefer to give both drugs as initial therapy and, when the results of antimicrobial susceptibility tests become available, usually continue with a single drug. Chloramphenicol has the advantage that, until recently, strains of Hib have been uniformly sensitive. Although occasional irreversable idiosyncratic bone marrow aplasia may occur, the risk is low in relation to the death rate from haemophilus meningitis, which is currently about 5%. Ampicillin is non-toxic and equally effective (providing appropriate dosage is used), but in 1974 ampicillin-resistant Hib strains emerged, which destroyed the antibiotic by producing ß-lactamase. Such strains were first encountered in Australia in 1976, and in recent years have probably increased in prevalence. For the period 1 January 1983 to 14 December 1984, nine (22%) of the 41 strains of Hib isolated from children with meningitis treated at the Adelaide Children's Hospital were resistant to ampicillin.

Because of the rarity of chloramphenicol resistance in Hib, paediatricians have been able to rely on chloramphenicol as a drug of choice in the treatment of bacterial meningitis. Although strains of Hib resistant to chloramphenicol have been reported during the past five years from South East Asia, North America and Europe, these remain rare. However, recent events in South Australia have shown that current therapy should be reviewed.

In July 1984, an isolate of Hib from a child with bacteraemia in Adelaide was shown to be resistant to both ampicillin and chloramphenicol (Moore et al, in press). The child's illness was mild and may have resolved without treatment. However, in October, and again in November, strains of Hib with a similar pattern of resistance were isolated from children with meningitis. Neither child responded to conventional therapy. When the results of the antimicrobial susceptibility tests became available, moxalactam was substituted. Both children then responded.

All three isolates have shown similar properties; resistance to ampicillin with minimal inhibitory concentration (MIC) values = 6-12 µg/ml, with ß-lactamase production; resistance to chloramphenicol with MIC=12 µg/ml, accompanied by inactivation of chloramphenicol[2]. These drug levels are not readily attainable in cerebrospinal fluid, so the degree of resistance encountered is therefore significant clinically. Each strain was also resistant to tetracycline.

Now that multiply-resistant strains of Hib have appeared in Australia, the question of appropriate therapy arises. Moxalactam is (highly) active against Hib, including strains of Hib resistant to ampicillin and chloramphenicol, with MIC values circa $\leq$ 0.1 µg/moxalactam/ml. Moxalactam shows good penetration of inflamed meninges, attaining cerebrospinal fluid levels of about 7 µg/ml on day 2, and 4 µg/ml on day 10. A study in the USA has shown that survival rates in children with haemophilus meningitis treated with moxalactam are similar to those in children treated with a combination of ampicillin and chloramphenicol, and the prevalence of serious sequelae are similar with the two regimens[3]. The only side effect commonly encountered to date is diarrhoea, associated with suppression of the normal bacterial flora of the gut. Laboratory studies have revealed a mild thrombocytosis, elevation of SGOT values, and an occasional prolongation of the prothrombin time (which may be related to destruction of vitamin K-producing bacteria in the gut). No clinical evidence of bleeding has been observed in paediatric patients.

So that the child with bacterial meningitis will receive optimal therapy from the outset, a combination of moxalactam and ampicillin has been advocated[3]. One regimen which has been recommended is a combination of moxalactam 200 mg/kg/day and ampicillin 200-400 mg/kg/day in divided doses. This provides adequate "cover" for Hib and the two other major bacterial pathogens in postnatal meningitis - Neisseria meningitidis and Streptococcus pneumoniae. If Hib sensitive to ampicillin is isolated, which will usually be the case, ampicillin can be continued and moxalactam ceased. If the Hib strain is resistant to ampicillin, moxalactam can be continued and ampicillin ceased. If S. pneumoniae is isolated, either ampicillin alone can be continued or penicillin G substituted. In N. meningitidis, either moxalactam or ampicillin are appropriate.

References

1. CDR (1984) 84/19 : 3
2. Lancet (1977) 2 : 1366
3. J. Pediatr. (1984) 104 : 454

## BRUCELLA MELITENSIS SURVEILLANCE

(Contributed by W. Monaghan, Pathology Department, West Gippsland Hospital, Warragul, Victoria)

Brucella melitensis was isolated recently from a 47 year old male presenting with undulating fever three weeks on his return from Europe. Relatives in Sicily reported that the patient had consumed goat cheese on the island when they heard of his illness.

Small, non-motile, Gram-negative coccobacilli were isolated from horse blood agar cultures incubated under aerobic and 5-10% supplementary $CO_2$ conditions. No growth was detected under anaerobic conditions. Growth was also detected on plain nutrient and MacConkey agar. The organism was catalase, oxidase and urease positive, but $H_2S$ negative. The isolate was forwarded to the National Brucellosis Reference Centre, Canberra, where it was confirmed as B. melitensis, biotype 3 (penicillin-resistant).

The patient responded well to tetracycline therapy, and was discharged with an uneventful recovery.

Editorial Comment

Brucella-infected livestock excrete the organism in their milk sporadically throughout almost their entire period of lactation. Goat milk is an important factor in the development of human B. melitensis infection in countries where goats are widely kept and the disease is endemic (the countries of the Mediterranean basin, and some countries in South America, Asia and Africa). The soft cheese prepared in these countries from goat milk is a frequent source of infection, since the methods used to make the cheese do not kill the brucellae which may persist in the finished product for up to 1 1/2 months under conditions of low acidity and a temperature of 11-14°C.

Of the 3315 cases of brucellosis reported in the USA in the 14 year period 1965-78, 127 were associated with raw dairy products from Mexico, predominantly fresh cheese made from unpasteurized goat milk[1]. Outbreaks attributed to the consumption of Mexican goat cheese have been reported in Colorado and Texas[1,2]. Outbreaks have also occurred among members of tour groups to countries bordering the Mediterranean Sea[1]. All of the 13 B. melitensis infections reported in the UK during 1981-83 were contracted abroad, with the exception of two persons who became ill in the UK after eating cheese imported from Jordan and Sicily[3].

Because such cases frequently have no vocational or avocational exposure to Brucella species, other more common causes of fever are generally ruled out before a diagnosis of brucellosis is entertained, thereby delaying appropriate therapy. Other organisms that have caused cheese-associated diseases or have been shown to survive the cheese-making process include Group C streptococci, Escherichia coli, Mycobacterium tuberculosis and Campylobacter jejuni. Raw milk cheese contaminated with staphylococcal enterotoxin is also a common occurrence[4,5].

References

1. MMWR (1983) 32 : 548
2. JAMA (1975) 233 : 63
3. CDR (1984) 84/19 : 3
4. J. Dairy Res. (1971) 38 : 91
5. J. Food Protect. (1983) 46 : 637

## HEPATITIS B VACCINE: EVIDENCE CONFIRMING LACK OF AIDS TRANSMISSION

(Based on MMWR (1984) 33 : 685)

Hepatitis B (HB) vaccine acceptance has been seriously hindered by the fear of possible acquired immunodeficiency syndrome (AIDS) transmission from the vaccine. However, recent studies have provided important additional assurances concerning the safety of the currently licensed vaccine which is produced from pooled plasma of hepatitis B surface antigen-positive individuals, some of whom are also in high risk groups for AIDS. Concern was expressed that the aetiological agent of AIDS might be present in the vaccine and survive the inactivation steps used in the manufacturing procedure. The concern persisted, despite the fact that these steps were reportedly able to inactivate representative members of all known virus groups. However, the recent identification of a retrovirus as the aetiological agent of AIDS has allowed workers to:

- Directly test the inactivation of the AIDS virus by the inactivation steps used in the vaccine manufacturing procedure;
- Look for the AIDS virus nucleic acid sequences in the vaccine;
- Look for serological markers of infection from the AIDS virus in vaccine recipients.

Concurrently, monitoring of AIDS patients and high-risk groups has continued in order to look for any epidemiological evidence of an association between HB vaccine and AIDS.

The effect of the HB vaccine inactivation process on the AIDS virus and two other human retroviruses (HTLV-I and HTLV-II) was studied. Three separate inactivation steps are used in the manufacture of the US-licensed HB vaccine: (1) 1 µg/ml pepsin, pH 2, 37°C, 18 hours; (2) 8 molar urea, 37°C, 4 hours; and (3) 0.01% formaldehyde, 37°C, 72 hours. In separate studies conducted between the Centres for Disease Control (CDC) and the vaccine manufacturer Merck, Sharp & Dohme (MSD), and between State University of New York (SUNY) Upstate Medical Centre and MSD, cell culture supernatent fluid containing the AIDS virus and cultured cells containing HTLV-I, HTLV-II, and the AIDS virus were transported to MSD and individually exposed to the three inactivation steps. The materials were then returned to CDC and SUNY for detection of residual viral infectivity. Virus infectivity was assayed by adding the treated material to cultured lymphocytes and periodically monitoring these for signs of viral replication (reverse transcriptase activity and virus antigen expression)[1], and in the case of HTLV-I and HTLV-II, transformation[2,3]. No residual virus was detected in material treated with formalin or urea, while material treated with pepsin at pH 2 did have residual virus present. Heat, an inactivation step used in vaccines manufactured outside the US, has also been shown to inactivate the AIDS virus[4].

The second approach, which attempted to detect AIDS virus-related nucleic acid sequences using dot blot hybridisation analysis of the vaccine with an AIDS virus deoxyribonucleic acid (DNA) probe, was done at MSD using as a positive control infected cellular ribonucleic acid (RNA) preparations provided by CDC. The vaccine contained no detectable AIDS virus-related sequences at a sensitivity of less than one picogram of DNA per 20 µg dose of vaccine.

The third approach attempted to detect seroconversion to AIDS virus antibodies in paired sera of HB vaccine recipients. Paired sera were examined at CDC using a highly sensitive and specific ELISA assay for the AIDS virus. No seroconversions were detected in 19 individuals who had received vaccine manufactured from plasma pools that contained plasma of homosexual men. Previous workers have reported that sera of HB vaccine recipients did not show helper-T/suppressor-T ratio inversion, a finding common in AIDS patients[5].

Epidemiological approaches to detect an association between HB vaccine and AIDS have included analysis of data on AIDS cases reported to CDC concerning their receipt of HB vaccine and monitoring rates of AIDS in groups of homosexually active men who did or did not receive HB vaccine in the vaccine trials conducted by CDC in Denver, Colorado, and San Franscisco, California. To date, 68 AIDS cases have been reported among approximately 700,000 US HB vaccine recipients; 65 have occurred among persons with known AIDS risk factors, while risk factors for the remaining three are under investigation. In

(continued on page 1)

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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 | | | 8 | | 8 | 2 | 28 | | 46 |
| 0101 ADENOVIRUS TYPE 1 | 10 | 1 | | 2 | | 6 | | 1 | 20 |
| 0102 ADENOVIRUS TYPE 2 | 4 | | | | | | | 3 | 7 |
| 0103 ADENOVIRUS TYPE 3 | | | | 1 | 4 | 3 | | 5 | 13 |
| 0104 ADENOVIRUS TYPE 4 | | | | | 1 | | | | 1 |
| 0105 ADENOVIRUS TYPE 5 | | | | | 10 | 1 | | 2 | 13 |
| 0107 ADENOVIRUS TYPE 7 | | 2 | | | | | | | 2 |
| 0108 ADENOVIRUS TYPE 8 | | | 3 | | | | | | 3 |
| 0110 ADENOVIRUS TYPE 10 | | | | | | | | 1 | 1 |
| 0119 ADENOVIRUS TYPE 19 | 2 | | | | | | | | 2 |
| 0199 ADENOVIRUS TYPING PENDING | | 2 | | | 6 | 2 | | | 10 |
| 0201 INFLUENZA A VIRUS | 2 | | | 9 | | 7 | 5 | 1 | 24 |
| 0202 INFLUENZA A VIRUS SUBTYPE H3N2 | | | | | | 3 | | | 3 |
| 0203 INFLUENZA B VIRUS | 1 | | 4 | 2 | | 2 | 3 | 5 | 17 |
| 0301 PARAINFLUENZA VIRUS TYPE 1 | | | 1 | | 6 | 2 | | | 9 |
| 0302 PARAINFLUENZA VIRUS TYPE 2 | | | 1 | 1 | 1 | | | | 3 |
| 0303 PARAINFLUENZA VIRUS TYPE 3 | 2 | 2 | 2 | | 17 | 5 | 3 | 7 | 38 |
| 0304 PARAINFLUENZA VIRUS TYPE 4 | | | | | | | | 1 | 1 |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS) | | 1 | 5 | 1 | 8 | 11 | 3 | 1 | 30 |
| 0500 RHINOVIRUS (ALL TYPES) | 1 | | | 2 | 16 | 10 | 6 | 1 | 36 |
| 0600 MYCOPLASMA PNEUMONIAE | 1 | | 1 | 2 | | 11 | | 8 | 23 |
| 0700 ORNITHOSIS-PSITTACOSIS | | | | 1 | | | | | 1 |
| 0800 COXSACKIEVIRUSES GROUP A - NOT TYPED | | | | | | | | 1 | 1 |
| 0809 COXSACKIEVIRUS A9 | 3 | 1 | 1 | 1 | | | 2 | 1 | 9 |
| 0901 COXSACKIEVIRUS B1 | 1 | | | | | | | | 1 |
| 0903 COXSACKIEVIRUS B3 | 2 | | | | | 1 | | | 3 |
| 0904 COXSACKIEVIRUS B4 | | | | | | | | 2 | 2 |
| 0905 COXSACKIEVIRUS B5 | | 1 | | 2 | | 8 | 5 | | 16 |
| 1000 ECHOVIRUS NOT TYPED | | | | | | | 4 | | 4 |
| 1003 ECHOVIRUS TYPE 3 | | 1 | | | | | 1 | | 2 |
| 1006 ECHOVIRUS TYPE 6 | | | | | | 3 | | | 3 |
| 1007 ECHOVIRUS TYPE 7 | | | | | | | 3 | | 3 |
| 1011 ECHOVIRUS TYPE 11 | 1 | | | 1 | | | | | 2 |
| 1014 ECHOVIRUS TYPE 14 | | | | | | | 1 | | 1 |
| 1017 ECHOVIRUS TYPE 17 | 1 | | | | | | | | 1 |
| 1020 ECHOVIRUS TYPE 20 | | | | 2 | | | | | 2 |
| 1022 ECHOVIRUS TYPE 22 | | | | | | | 2 | | 2 |
| 1030 ECHOVIRUS TYPE 30 | 2 | | | 1 | | | | | 3 |
| 1100 POLIOVIRUS NOT TYPED | | | 2 | | | | 1 | | 3 |
| 1101 POLIOVIRUS TYPE 1 | | | | | | | | 2 | 2 |
| 1102 POLIOVIRUS TYPE 2 | | | | | | 1 | 1 | | 2 |
| 1104 POLIOVIRUS-VACCINAL STRAIN | | | | | | 1 | | | 1 |
| 1200 MUMPS VIRUS | 2 | | 1 | 6 | | | | | 9 |
| 1300 HERPES VIRUS GROUP-NOT TYPED | 33 | | 3 | 1 | | 2 | 1 | 1 | 41 |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | | 4 | | 6 | 1 | | | 1 | 12 |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS) | 3 | 1 | 1 | 3 | 1 | 3 | | 7 | 19 |
| 1303 VARICELLA-ZOSTER VIRUS | | 1 | 2 | 1 | | 2 | 1 | 1 | 8 |
| 1306 HERPES SIMPLEX TYPE 1 | 12 | | 17 | 36 | 16 | 34 | 66 | 17 | 198 |
| 1307 HERPES SIMPLEX TYPE 2 | 89 | | 17 | 44 | 2 | 46 | 150 | 79 | 427 |
| 1399 HERPES VIRUS TYPING PENDING | | | | 2 | 6 | 8 | | | 16 |
| 1401 COXIELLA BURNETI | 6 | | 2 | 2 | | 1 | 1 | 3 | 15 |
| 1502 PICORNA VIRUS-NOT TYPED | 4 | | 10 | | | | 3 | 3 | 20 |
| 1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS) | | | | | | | | 3 | 3 |
| 1521 MEASLES VIRUS | 5 | 5 | 3 | 1 | | 1 | | 2 | 17 |
| 1522 RUBELLA VIRUS | 24 | 3 | 9 | 6 | | 31 | 2 | 18 | 93 |
| 1532 HEPATITIS B ANTIGEN | 73 | 2 | 24 | 38 | 1 | 58 | 25 | 11 | 232 |
| 1535 HEPATITIS A ANTIBODY | 2 | | | 10 | | 3 | 1 | 4 | 20 |
| 1541 CHLAMYDIA A - C TRACHOMATIS | 28 | | 4 | 14 * | | | 49 | 52 | 147 |
| 1543 CHLAMYDIA A - LGV TYPE | | | | | | | | 1 | 1 |
| 1556 CMV - CYTOMEGALOVIRUS | 12 | 1 | 2 | 23 | 7 | 8 | 6 | 14 | 73 |
| 1562 REOVIRUS (ALL TYPES) | | | | 1 | | | | | 1 |
| 1564 ROTAVIRUS | | 5 | 18 | | 13 | 21 | 1 | 1 | 59 |
| 1599 ENTEROVIRUS TYPING PENDING | | 1 | 6 | | 11 | | | | 18 |
| 9902 POXVIRUS GROUP NOT TYPED | | | | 1 | | | | | 1 |
| 9992 ROSS RIVER VIRUS | | | | | | | 6 | 6 | 12 |
| 9993 ASTROVIRUS | | 1 | | | | | | | 1 |
| 9994 SMALL VIRUS (LIKE) PARTICLE | | 2 | | 1 | | | | | 3 |
| Total | 326 | 37 | 147 | 224 | 135 | 297 | 380 | 267 | 1,813 |

* Cultures performed at Microbiological Diagnostic Unit, Melbourne.

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PERIOD : 6/12/84 to 2/1/85 .... 85/1

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 | 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 | 1 | | | | 2 | | | | |
| 0101 ADENOVIRUS TYPE 1..... | | 9 | | 2 | | | 7 | | | | |
| 0102 ADENOVIRUS TYPE 2..... | 2 | 3 | | 1 | | 1 | | | | | |
| 0103 ADENOVIRUS TYPE 3..... | 1 | 6 | 1 | | | | 3 | | | | |
| 0104 ADENOVIRUS TYPE 4..... | | | | | | | 1 | | | | |
| 0105 ADENOVIRUS TYPE 5..... | | 10 | | | | | | | 1 | | |
| 0107 ADENOVIRUS TYPE 7..... | | 1 | | | | | 1 | | | | |
| 0201 INFLUENZA A VIRUS..... | 3 | 16 | | | | 1 | | 1 | | | |
| 0202 INFLUENZA A VIRUS SUBTYPE H3N2 | | 3 | | | | | | | | | |
| 0203 INFLUENZA B VIRUS..... | | 9 | | | | | | | 1 | | 1 |
| 0301 PARAINFLUENZA VIRUS TYPE 1.... | | 9 | | 1 | | | | | | | |
| 0302 PARAINFLUENZA VIRUS TYPE 2.... | | 3 | | | | | | | | | |
| 0303 PARAINFLUENZA VIRUS TYPE 3.... | | 36 | | | | 1 | | | | | |
| 0304 PARAINFLUENZA VIRUS TYPE 4.... | | 1 | | | | | | | | | |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS)..... | | 26 | | | | | | | | 1 | |
| 0500 RHINOVIRUS (ALL TYPES)..... | 1 | 29 | | | | | 1 | | 1 | | 1 |
| 0600 MYCOPLASMA PNEUMONIAE..... | 2 | 20 | | | | | | | | | 1 |
| 0700 ORNITHOSIS-PSITTACOSIS..... | | 1 | | | | | | | | | |
| 0809 COXSACKIEVIRUS A9..... | | 3 | | 2 | | 1 | 2 | | | | |
| 0901 COXSACKIEVIRUS B1..... | | | | 1 | | | | | | | |
| 0903 COXSACKIEVIRUS B3..... | 1 | | | 2 | | | | | | | |
| 0905 COXSACKIEVIRUS B5..... | | 3 | | 8 | | | 2 | | | | 2 |
| 1003 ECHOVIRUS TYPE 3..... | | | | | | | | | | | 1 |
| 1006 ECHOVIRUS TYPE 6..... | | | | 2 | | | 1 | | | | |
| 1007 ECHOVIRUS TYPE 7..... | | 1 | | 2 | | | | | | | |
| 1011 ECHOVIRUS TYPE 11..... | | 1 | | 1 | | | | | | | |
| 1014 ECHOVIRUS TYPE 14..... | | 1 | | | | | | | | | |
| 1020 ECHOVIRUS TYPE 20..... | | 1 | | | | | | | | | |
| 1022 ECHOVIRUS TYPE 22..... | | 1 | | | | | 1 | | | | |
| 1030 ECHOVIRUS TYPE 30..... | 1 | | | 1 | | | | | | | |
| 1100 POLIOVIRUS NOT TYPED..... | | | | | | | 1 | | | | |
| 1101 POLIOVIRUS TYPE 1..... | | | | | | | | 1 | | | |
| 1102 POLIOVIRUS TYPE 2..... | | | | | | | 2 | | | | |
| 1200 MUMPS VIRUS..... | 1 | | | 1 | | | | | | | |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | | 2 | | | | | | | | 1 | 8 |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS). | 5 | 1 | | | | | | | | | 1 |
| 1303 VARICELLA-ZOSTER VIRUS..... | | | 1 | | | | | | | | 3 |
| 1306 HERPES SIMPLEX TYPE 1..... | 6 | 7 | 1 | | | 1 | | | | 3 | 91 |
| 1307 HERPES SIMPLEX TYPE 2..... | 15 | | | | | | | 2 | | | 82 |
| 1401 COXIELLA BURNETI..... | 3 | | | | | | | | | | 1 |
| 1502 PICORNA VIRUS-NOT TYPED..... | | | | | | | 3 | | | | |
| 1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS)..... | | | | | | | | | | | 3 |
| 1521 MEASLES VIRUS..... | 5 | | 1 | | | 1 | | | | | 7 |
| 1522 RUBELLA VIRUS..... | 9 | 2 | 3 | | | | | | | | 63 |
| 1532 HEPATITIS B ANTIGEN..... | 105 | | | | | | | 84 | | | 1 |
| 1535 HEPATITIS A ANTIBODY..... | | | | | | | | 19 | | | |
| 1541 CHLAMYDIA A - C.TRACHOMATIS... | 1 | 1 | | | | | | | | | |
| 1556 CMV - CYTOMEGALOVIRUS..... | 3 | 13 | 1 | 1 | | 1 | | 1 | | 5 | 2 |
| 1562 REOVIRUS (ALL TYPES)..... | 1 | | | | | | | | | | |
| 1564 ROTAVIRUS..... | | 2 | | | | | 56 | | | | |
| 9902 POXVIRUS GROUP NOT TYPED..... | | | | | | | | | | | 1 |
| 9992 ROSS RIVER VIRUS..... | 2 | | | | | | | | | | 2 |
| 9993 ASTROVIRUS..... | | | | | | | 1 | | | | |
| 9994 SMALL VIRUS (LIKE) PARTICLE... | | | | | | | 3 | | | | |
| Total..... | 167 | 222 | 9 | 25 | | 7 | 87 | 109 | 4 | 10 | 271 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 6/12/84 to 2/1/85 ... 85/1
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 ...

| VIRUS OR VIRAL ANTIGEN | Eye | Genital | Endo/sal gland | RES | Muscle/joint | Congenital | PUO | Fever/malaise | Other | SIDS |
|---|---|---|---|---|---|---|---|---|---|---|
| 0100 ADENOVIRUS NOT TYPED..... | | | | | | | | 1 | | |
| 0101 ADENOVIRUS TYPE 1..... | 1 | 1 | | | | | | | | |
| 0103 ADENOVIRUS TYPE 3..... | 1 | | | | 1 | | 1 | 1 | | |
| 0105 ADENOVIRUS TYPE 5..... | | | | | | | | | 1 | 1 |
| 0108 ADENOVIRUS TYPE 8..... | 3 | | | | | | | | | |
| 0110 ADENOVIRUS TYPE 10..... | | | | | | | | 1 | | |
| 0119 ADENOVIRUS TYPE 19..... | 2 | | | | | | | | | |
| 0201 INFLUENZA A VIRUS..... | | | 1 | | 1 | | | 4 | | |
| 0203 INFLUENZA B VIRUS..... | | | 1 | | 1 | | 2 | 5 | 1 | |
| 0302 PARAINFLUENZA VIRUS TYPE 2.... | | | | | | | | 1 | | |
| 0303 PARAINFLUENZA VIRUS TYPE 3.... | | | | | | | | 1 | | 1 |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS)..... | | | | 1 | | | | 1 | | 1 |
| 0500 RHINOVIRUS (ALL TYPES)..... | 1 | | | | | | 1 | 1 | | 2 |
| 0600 MYCOPLASMA PNEUMONIAE..... | | | | | | | | | 1 | |
| 0809 COXSACKIEVIRUS A9..... | | | | | | | | 1 | | |
| 0904 COXSACKIEVIRUS B4..... | | | | | | | 1 | | 1 | |
| 0905 COXSACKIEVIRUS B5..... | | | | | | | | | 1 | |
| 1003 ECHOVIRUS TYPE 3..... | | | | | | | | 1 | | |
| 1014 ECHOVIRUS TYPE 14..... | | | | | | | | 1 | | |
| 1017 ECHOVIRUS TYPE 17..... | | | | | | | | 1 | | |
| 1020 ECHOVIRUS TYPE 20..... | | | | | | | | 2 | | |
| 1030 ECHOVIRUS TYPE 30..... | | | | | | | | | 1 | |
| 1101 POLIOVIRUS TYPE 1..... | | | | | | | | | | 1 |
| 1104 POLIOVIRUS-VACCINAL STRAIN.... | | | | | | | | | 1 | |
| 1200 MUMPS VIRUS..... | | | 6 | | | | | 1 | | |
| 1300 HERPES VIRUS GROUP-NOT TYPED.. | | | | 3 | | | | | | |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS). | | | 7 | | | | 2 | 3 | 3 | |
| 1303 VARICELLA-ZOSTER VIRUS..... | 1 | 1 | | | | | | 1 | | |
| 1306 HERPES SIMPLEX TYPE 1..... | 14 | 66 | 2 | | 1 | | 3 | 4 | 2 | |
| 1307 HERPES SIMPLEX TYPE 2..... | 1 | 329 | | | | | | 1 | | |
| 1399 HERPES VIRUS TYPING PENDING... | | 1 | | | | | | | 1 | |
| 1401 COXIELLA BURNETI..... | | 1 | 2 | | 1 | | 5 | 2 | 1 | |
| 1521 MEASLES VIRUS..... | | | | | | | | 1 | 3 | |
| 1522 RUBELLA VIRUS..... | 1 | | 4 | | 10 | 1 | | 2 | 7 | |
| 1532 HEPATITIS B ANTIGEN..... | | | | | 1 | | | 2 | 35 | |
| 1535 HEPATITIS A ANTIBODY..... | | | | | | | | 1 | | |
| 1541 CHLAMYDIA A - C.TRACHOMATIS... | | 144 | | | | | | | | |
| 1543 CHLAMYDIA A - LGV TYPE..... | | 1 | | | | | | | | |
| 1556 CMV - CYTOMEGALOVIRUS..... | 1 | 8 | | 1 | 1 | 4 | 2 | 4 | 24 | 1 |
| 9992 ROSS RIVER VIRUS..... | | | | | 10 | | | 3 | | |
| Total..... | 26 | 552 | 23 | 5 | 27 | 5 | 17 | 47 | 83 | 7 |