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# Communicable Diseases Intelligence

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Contents:



Editor: I F Cook

VIRUS REPORTING SCHEME: A total of 1 159 reports were processed for this period.

An increase in Enterovirus type 71 activity is apparent in the current reporting period with 18 reports of this virus associated with general malaise and/or mild fever (1), respiratory tract infection (9), encephalitis (1) and meningitis (7). Viral activity has been predominantly in children aged 1 year and below.

The demographics of these cases were:-

SEX: Male, 9 ; Female, 9
AGE: 0-1 year, 15; 2-5 years, 1; > 10 years, 1; not known,1.

Enterovirus type 71, prototype strain BrCr has been associated with and epidemic of viral (aseptic) meningitis among young patients at the Royal Children's Hospital, Melbourne, during the month of May. A full report of this epidemic will be published in the next edition of the CDI (CDI 86/16).

Enterovirus type 71 shares an antigen with coxsackievirus A16, the enterovirus isolated from cases of hand, foot and mouth disease reported in CDI 86/12, but there is no cross - neutralisation.

The activity of both viruses (enterovirus type 71 and coxsackievirus A16) has been monitored for the past ten reporting periods (Figure).

Regular monitoring of rotavirus (CDI 86/14) indicates a further increase in this virus activity with 120 cases of rotavirus associated diarrhoea reported for this period compared to 102 cases for the previous period. The demographic patterns of the cases remained unaltered.

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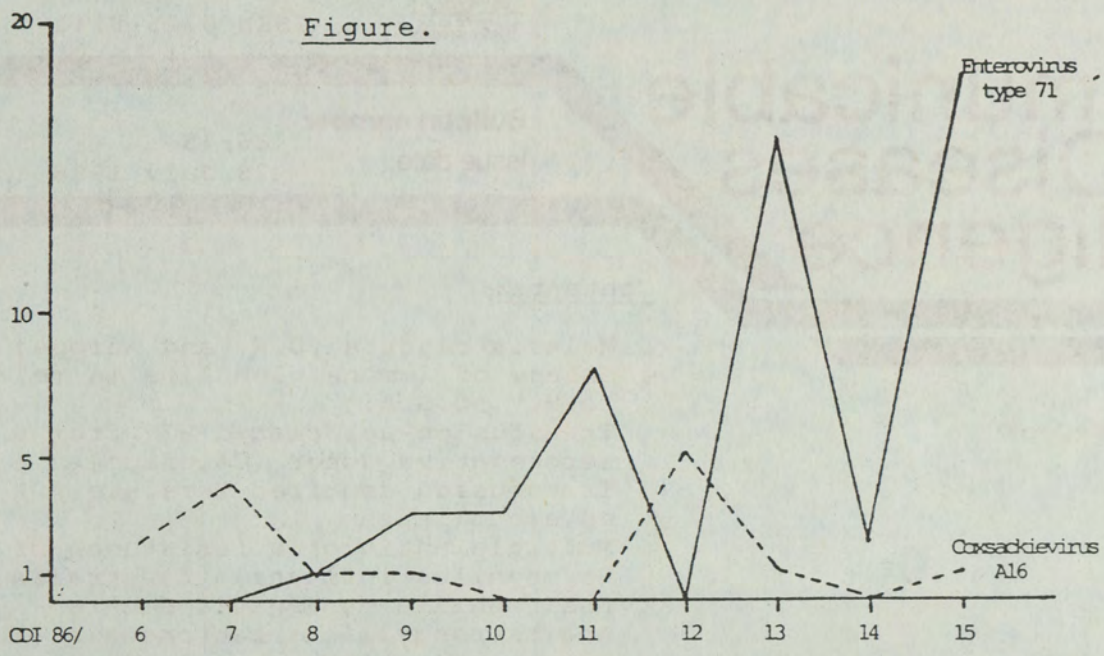
Figure.




MALARIA REPORTS
(based on CDR 86/25 and 86/26, June 1986)

The first malaria death in the United Kingdom for 1986 recorded a 57 year old British businessman who died with falciparum malaria in the South Western region. He had been taking maloprim prophylactically while working in Kenya. In 1985 a total of five malaria deaths were recorded; all the patients had been infected in Africa.

One of five workers who have recently developed falciparum malaria during their employment at the cargo terminal of Brussels airport, has died. These individuals have been handling cargo from Zaire.

In previous years, cases of falciparum malaria associated with European airports have occurred during periods of hot weather. There were two cases in the United Kingdom associated with Gatwick airport in 1983 and a British traveller with malaria, thought to have been infected on a home flight, came through Heathrow in the same year.

SAFETY OF IMMUNE GLOBULINS IN RELATION TO HIV (HUMAN IMMUNE DEFICIENCY VIRUS)
(Based on Food and Drug Administration Bulletin 1986; 16:3 and California Morbidity #21, 30 May 1986)

Laboratory tests by U.S. Food and Drug Administration (FDA) scientists, as well as epidemiologic data evaluated by the Centers for Disease Control (CDC), show that therapeutic immune globulin preparations carry no discernible risk of transmitting HIV infection and that their clinical use should not be changed because of concerns about the transmission of acquired immunodeficiency syndrome (AIDS).

In response to inquiries from health professionals regarding HIV infectivity, FDA scientists conducted laboratory tests to evaluate the basic fractionation processes used for production of immune globulin (IG), hepatitis B immune globulin (HBIG), and intravenous immune globulin (IVIG). After the products were spiked with HIV, the degree to which the virus was reduced by partitioning or inactivation at each of six individual steps ranged from $10^1$ to more than $10^4$ of in vitro infectious units (IVIU)/ml. The effectiveness of virus removal in the entire process was calculated to be greater than $1 \times 10^{15}$ IVIU/ml. Concentrations of infectious HIV in plasma of infected persons have been estimated at less than 100 IVIU/ml, and FDA scientists have shown that the geometric mean infectivity titre of plasma from 43 HIV infected persons was 0.02 IVIU/ml[1].

In addition, FDA and CDC scientists cultured 38 lots of HBIG, IVIG and IG, most of which contained HIV antibody. HIV was not recovered from any lot tested.

Epidemiologic data concur with the results of the laboratory tests. Several studies have shown that recipients of HBIG and IG, including recipients of lots known to be positive for antibody to HIV, did not show either serologic evidence or signs and symptoms of AIDS or other illnesses indicative of HIV infection[2]. These studies include data on 16 subjects who received HBIG that was strongly positive for HIV antibody. In this group, low levels of passively acquired HIV antibody were detected shortly after injection, but reactivity did not persist. Six months after the last HBIG injection, none of the 16 patients had antibody to HIV and thus were not infected with the virus[3].

Based on the estimated half-life of globulins in plasma, it can be calculated that passively acquired antibodies might be detected in sera of recipients for as long as six months after administration of immune globulins. This possibility should be recognized when attempting to determine the significance of HIV antibody in a patient who has recently received immune globulins, especially HBIG.

REFERENCES

1. Tranfusion (1986) 26:210-213
2. MMWR (1986) 35:231-232
3. Lancet (1985) 1:815.

## TRANSFUSION - ASSOCIATED HUMAN IMMUNODEFICIENCY VIRUS (HIV)* FROM A SERONEGATIVE DONOR - COLORADO

(Based on MMWR Vol. 35/No. 24 - 20 June 1986)

*The Human Retrovirus Subcommittee of the International Committee on the Taxonomy of Viruses has proposed the name human immunodeficiency virus (HIV) to replace HTLV-III (Science 1986; 232: 697).

In November 1985, a blood donor at a Colorado blood-collection centre was found to be seropositive for human immunodeficiency virus (HIV) antibody by both the enzyme-linked immunosorbent assay (ELISA) and Western blot methods. He had previously

donated at the centre in April and August 1985, when he had been seronegative by ELISA. Both recipients from the August donation, one of whom had no other risk factors for acquisition of HIV were subsequently found to be seropositive. Both recipients of the April donation were seronegative. The donor had probably been infected through sexual contact 12 weeks or less before the August donation. This is the first reported transmission of infection from a blood donor that has occurred despite routine screening for HIV antibody in blood banks and plasma centres.

Details of the donor and recipient investigation are as follows:

**Donor.** The donor was a 31-year-old man who had donated blood at the same centre in April, August, and November 1985. He was seronegative in April (optical densities of Abbott ELISA on sample/control = 0.052/0.160) and August (0.034/0.142), but seropositive by ELISA (0.926/0.173) and Western blot in November. His blood from the November donation was discarded, and physicians of the recipients from the August donation were notified by the blood centre of the possible transmission of HIV from these blood products.

When interviewed in April 1986, the donor stated that he had had sexual contact with one male partner, with the first exposure taking place on May 15, 1985. No condoms were used. His only other sexual partner was a man in 1974. He denied intravenous (IV) drug use or history of blood transfusion. He had no history of acute viral illnesses or symptoms of acquired immunodeficiency syndrome (AIDS) or AIDS-related complex (ARC) in 1985 or 1986. Physical examination in December 1985 was normal. Repeat ELISA testing in April 1986 revealed a high absorbency value ( 2.000/0.125), and Western blot was once again positive. Attempts at locating previous sera for antibody testing were unsuccessful.

**Donor's Partner.** The donor's sexual partner was a 22-year-old man who corroborated the donor's history of their initial sexual contact on may 15, 1985. He had been homosexually active since 18 years of age. He denied IV drug abuse or history of blood transfusion. After notification by the donor of his positive antibody status, the partner was tested for HIV in November 1985 and was seropositive by ELISA and Western blot; these findings were reconfirmed on a separate specimen in April 1986. He had not previously been tested for HIV antibody.

**Recipient 1.** Recipient 1 was a 60-year-old man who underwent surgery in August 1985. He received from 15 different donors six units of packed red blood cells, four units of fresh frozen plasma, and six units of platelets (incuding one unit from the previously described donor). He had been married for 30 years and denied extramarital sexual contact, either heterosexual or homosexual, or any previous blood transfusions or IV drug abuse. In February 1986, he had no symptoms of AIDS or ARC and had a normal physical examination. The HIV antibody test was positive by ELISA and Western blot and reconfirmed on a separate specimen in march 1986. His wife was seronegative for HIV antibody in April 1986.

Recipient 2. Recipent 2 was a 57-year-old man who underwent surgery in August 1985. He received two units of platelet-poor whole blood (including one unit from the previously described donor) and one unit of packed red blood cells. During the postoperative period, he had unexplained fever and diarrhoea that persisted for 6 weeks and was associated with a 9 kg weight loss. Stool specimens were negative for bacterial pathogens and ova and parasites, including cryptosporidia. In October 1985, he was tested for HIV antibody for reasons unrelated to the blood transfusion and was positive by ELISA and Western blot, which was confirmed on a separate specimen in April 1986. He had been divorced for 12 years and was strictly homosexual since that time, with multiple partners.

Other investigative findings. The blood donated in April 1985 was given to two recipients, and both were seronegative by ELISA when tested in May 1986.

One other person was a common donor to recipients 1 and 2 in August 1985. This person was retested in April 1986 and was negative by ELISA for HIV antibody. Of the 13 remaining donors to recipient 1, 11 were seronegative when retested 5 months or more after the August donations. Two donors reside outside Colorado and have not been retested. Of the two remaining donors to recipient 2, both were seronegative when retested 6 months or more after the August donations.

MMWR Editorial Note: This is the first report of HIV transmission from a person whose blood tested negative for HIV antibody at the time of blood donation. As with previous reports that have documented the presence of the virus in a small number of persons who have no detectable antibody, this donor appears to have had a recent infection (1,2). Most infected people develop antibody within 2-3 months of infection (2-6).

The current risk of transfusion-associated infection is small. The prevalence of positive Western blot tests among units screened by the American Red Cross in early 1985 suggests that 0.04% of all donated units may have been potentially infectious (7). Currently available screening tests detect HIV antibody in the great majority of infected persons. Since antibody may not be detectable in blood from donors with very recent infections, the safety of the blood supply also requires deferral of donation by persons at increased risk for HIV infection.

Donor-deferral programs, initially implemented in blood banks in the US in March 1983 and subsequently refined, provide all prospective donors with educational information on the practices associated with an increased risk of HIV infection. Evidence suggests that most persons at increased risk have stopped donating blood (8-10), but a few such individuals continue to donate. The donor described in this report said he felt he was not at risk for infection because he had only one sexual partner. Although a steady sexual relationship with a single partner is generally safer with regard to HIV infection than relationships with multiple sexual partners, the Centres for Disease Control (CDC) Atlanta Georgia recommend that men who have had sexual contact with another man since 1977 do not donate blood (11).

Efforts are continuing to assure maximum effectiveness of the US donor-deferral programs (12). As an example, blood collection agencies in that country have agreed to implement procedures in which prospective donors are asked to sign an expanded consent statement. The statement indicates that the prospective donor has reviewed and understands the informational material provided and that donors who are at increased risk for transmission of HIV or other infectious agents will not donate blood or plasma for transfusion to another person.

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3. Lancet (1984) ii: 1376-7
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6. Lancet (1985) ii: 1083-6
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8. NEJM (1984) 310: 1194
9. Transfusion (1985) 24: 434
10. Transfusion (1985) 25: 3-9
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12. NEJM (1986) 314: 1115-6.

## TRANSFUSION ACQUIRED YERSINIA ENTEROCOLITICA

Yersinia enterocolitica is a psychrophilic gram-negative bacterium which causes GI disturbances, notably acute self-limiting gastroenteritis or enterocolitis particularly in young children. It can also cause mesenteric lymphadenitis and terminal ileitis, the symptoms of which resemble appendicitis.

A report of a fatal case of transfusion acquired Y.enterocolitica septicaemia has recently been published(1). A decompression laminectomy was performed on a 61 year old man who had been diagnosed with an extradural tumour at the level of the body of the L1 vertebra. The histologic features of the tumour were those of poorly differentiated adenocarcinoma. After surgery he had persistent generalised muscle weakness and required assisted ventilation. On the 20th post-operative day a low haemoglobin level (104 g/L) necessitated transfusion with packed red cells. He developed rigors after less than one hour and transfusion was stopped after 100 mL had been administered. He subsequently became febrile, hypotensive (systolic blood pressure 70 mmHg) and oliguric, and treatment with cephalothin and gentamicin was commenced. He remained hypotensive, despite vasopressor therapy, and died 15 hours after the commencement of the transfusion.

Y.enterocolitica was subsquently isolated from blood cultures taken after the transfusion and from residual blood in the pack. Both isolates had the same biochemical profile, were sensitive to gentamicin and resistant to cephalothin, and belonged to serotype 03, biotype 4. There was no evidence of blood incompatibility.

In four previous reports of transfusion acquired Y.enterocolitica[2-4], the donors were all seropositive, suggesting donor asymptomatic bacteremia at the time of donation. Unfortunately the donor's serologic status was not determined in the present case, as further serum was not obtained, being psychrophilic, multiplies during storage of blood at 4°C. Y.entercolitica, and organism may thus be transmitted in large numbers to patients receiving contaminated blood transfusions.

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2. Scand J Infect Dis (1984) 16(4):411-2.
3. Transfusion (1982) 22:396-8.
4. Med Maladies Infect (1982) 13:197-9.

MULTIPLE ANTIBIOTIC RESISTANCE OF HAEMOPHILUS INFLUENZAE

(Contributed by B.E. Wild, J.W. Pearman and C.J.L. Richardson, Princess Margaret Hospital for Children, Perth, WA)

A multiply antibiotic-resistant strain of Haemophilus influenzae type b (MRHib) was isolated from the cerebrospinal fluid (CSF) of a child with meningitis. Following the initial diagnosis, the isolate had been reported as sensitive to ampicillin and treatment with ampicillin and chloramphenicol was initiated at a regional Western Australian hospital. The patient failed to respond to treatment and was subsequently transferred to Princess Margaret Hospital where a culture of CSF, obtained from a second lumbar puncture taken one week after initiating treatment, yielded a moderate growth of MRHib. The organism was resistant to chloramphenicol (as demonstrated by the method of Slack et al[1]), amoxycillin and tetracycline, but sensitive to cefotaxime, sulphonamide, trimethoprim and rifampicin. The organism produced a $\beta$-lactamase.

During the 1970's strains of H. influenzae were reported resistant to single antibiotics, including ampicillin, tetracycline and chloramphenicol. Resistance to ampicillin now occurs in 14% of H. influenzae isolates at Princess Margaret Hospital. Multiple antibiotic resistance of Hib was first reported from Thailand in 1980[2], then in the United States, the Caribbean and Europe, and subsequently in Australia in 1984 when 3 isolates were found in South Australia (Adelaide)[3]. Although no further reports of MRHib have come from South Australia, the occurrence of similar organisms in Victoria and Western Australia is significant for Australian clinicians treating invasive infections possibly caused by H. influenzae type b, such as epiglottitis, orbital cellulitis and meningitis in children.

For the past two decades, many paediatricians have used parenteral ampicillin and chloramphenicol combined until results of culture and antibiotic sensitivity testing permit the refinement of antibiotic treatment. It is now suggested that it would be more appropriate to use a combination of

either ampicillin and cefotaxime or cefotaxime plus chloramphenicol in the primary treatment of presumed Hib meningitis in children. Once antibiotic sensitivities have been obtained the regimen can be refined to that considered most appropriate for the clinical situation.

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2. Lancet (1980) ii: 1214-1217
3. MJA (1985) 142: 78

## EOSINOPHILIC MENINGITIS DUE TO ANGIOSTRONGYLIASIS CANTONENSIS IN TAIWAN

(Based on Epidem. Bull. (China)(1986) 2, 21-26)

Two outbreaks of eosinophilic meningitis occurred in Taiwan in the period June-September 1985.

In the first outbreak eight adults and one child from an extended family were hospitalised with headache, fever, diplopia, and myalgias. Findings on physical examination included nuchal rigidity (8), positive Kernig's sign (3), anisocoria (1), and papilloedema (1). Laboratory findings included eosinophilia ($\geq$ 5%) in the cerebral spinal fluid (CSF) (9) and peripheral blood (6). A worm of the species Angiostrongylus cantonensis was recovered from the CSF of one patient. Four of the nine died, including one who developed unilateral blindness. The remainder recovered with supportive therapy. All affected family members had eaten raw snails 1-3 weeks before onset of symptoms.

In the second outbreak four children under 6 years of age were admitted to hospital with fever, headache, vomiting, and lethargy. All four were playmates. Blood counts revealed eosinophilia ranging from 8-31% in three of the four children. Elevated eosinophil counts of 36% and 45% were found in the CSF of two children. A worm of the species A. cantonensis was recovered from the CSF of one child. All four children recovered with supportive therapy. Approximately 1-2 weeks before onset of symptoms, the children collected snails near their home. The children roasted the snails in their shells over an open fire and ate them. The number of snails consumed by each child was unknown. A fifth playmate who did not become ill, tasted, but did not swallow, the snail meat.

Epidem. Bull. Editorial Note:

Angiostrongyliasis results from ingestion of the third-stage larvae of the rat lung worm, A. cantonensis, The adult worms live in the pulmonary arteries of rodents and lay eggs which hatch in the lung, migrate to the bowel, and are passed as first-stage larvae in the faeces. The larvae enter natural molluscan intermediate hosts and develop into infective third-stage larvae. When a rodent eats infected molluscs, the larvae penetrate the gut, migrate to the brain, and finally to the pulmonary arteries where they develop into adult worms, completing the life-cycle. Man is an accidental dead-end host

and becomes infected by eating raw or under-cooked molluscs containing the third-stage larvae of A. cantonensis. The ingested larvae migrate to the brain, spinal cord, eyes, and lungs and die. The dead worms provoke a marked inflammatory response often accompanied by eosinophilia in the CSF. Clinical manifestations vary depending on the number and location of the worms. Extensive tissue damage can occur as the adult worms migrate through the brain or eyes. The case-fatality rate is low ($< 5\%$); blindness is one of the most common sequelae. The efficacy of anthelmintic therapy is still under investigation, although some authors recommend anthelmintics should not be given since the simultaneous death of many worms might provoke a severe inflammatory reaction.

CDI Editorial Comment

Mortality associated with Angiostrongylus cantonensis is of low order. However in the first epidemic reported above, 4 out of 9 patients died. This is atypical.

A. cantonensis causes sporadic cases of eosinophilic meningitis in Northern Australia, particularly Queensland.

Eosinophilic meningitis has also been reported from Thailand, Vietnam, Cambodia, Vanuatu, New Caledonia, Cook Island, Tahiti and Hawaii.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 7/7/86 - 21/7/86 BULLETIN NUMBER 86/15
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 | | | | 7 | | 14 |
| 0101 ADENOVIRUS TYPE 1 | | | | | | 2 | | 1 | 3 |
| 0102 ADENOVIRUS TYPE 2 | | | | 2 | | 4 | | | 6 |
| 0103 ADENOVIRUS TYPE 3 | | | | | | 2 | | | 2 |
| 0104 ADENOVIRUS TYPE 4 | | | | | | | | 1 | 1 |
| 0105 ADENOVIRUS TYPE 5 | | | | | | 1 | | 1 | 2 |
| 0106 ADENOVIRUS TYPE 6 | | | | | | 1 | | | 1 |
| 0108 ADENOVIRUS TYPE 8 | | | | 3 | | | | | 3 |
| 0137 ADENOVIRUS TYPE 37 | | | | | | | | 1 | 1 |
| 0199 ADENOVIRUS TYPING PENDING | | | 1 | | 4 | | | | 5 |
| 0201 INFLUENZA A VIRUS | | | | | | | | 1 | 1 |
| 0203 INFLUENZA B VIRUS | | | | | | | | 1 | 1 |
| 0301 PARAINFLUENZA VIRUS TYPE 1 | | 1 | | 1 | 6 | 1 | 3 | | 12 |
| 0302 PARAINFLUENZA VIRUS TYPE 2 | | | | 1 | 12 | 14 | | | 27 |
| 0303 PARAINFLUENZA VIRUS TYPE 3 | | | 1 | 2 | 1 | | 1 | 1 | 6 |
| 0399 PARAINFLUENZA VIRUS TYPING PENDING | | 1 | | | | | | | 1 |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS) | 2 | 23 | 6 | 7 | 33 | 4 | 19 | 4 | 98 |
| 0500 RHINOVIRUS (ALL TYPES) | | | | 7 | 27 | 4 | 1 | 1 | 40 |
| 0600 MYCOPLASMA PNEUMONIAE | | 1 | | | | | | | 1 |
| 0700 ORNITHOSIS-PSITTACOSIS | 2 | | | | | | | | 2 |
| 0800 COXSACKIEVIRUSES GROUP A - NOT TYPED | | | | | | 1 | | | 1 |
| 0816 COXSACKIEVIRUS A16 | | | | | | | | 1 | 1 |
| 0905 COXSACKIEVIRUS B5 | | | | | | 1 | | | 1 |
| 1003 ECHOVIRUS TYPE 3 | | | | 1 | | | | | 1 |
| 1007 ECHOVIRUS TYPE 7 | | 1 | | | | | | | 1 |
| 1011 ECHOVIRUS TYPE 11 | | | 1 | | | | | 9 | 10 |
| 1014 ECHOVIRUS TYPE 14 | | 1 | | | | | | | 1 |
| 1020 ECHOVIRUS TYPE 20 | | | | | | | | 1 | 1 |
| 1021 ECHOVIRUS TYPE 21 | | | | | 1 | | | | 1 |
| 1022 ECHOVIRUS TYPE 22 | | | | 1 | | | | | 1 |
| 1100 POLIOVIRUS NOT TYPED | | | 1 | | 3 | | | | 4 |
| 1101 POLIOVIRUS TYPE 1 | | | | | | 1 | | | 1 |
| 1102 POLIOVIRUS TYPE 2 | | | | | | | 1 | | 1 |
| 1200 MUMPS VIRUS | | | | | 1 | | | 2 | 3 |
| 1300 HERPES VIRUS GROUP-NOT TYPED | 1 | | 1 | 5 | | | 1 | 1 | 9 |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | | | | | | | | 1 | 1 |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS) | | 1 | 1 | | | | | 6 | 8 |
| 1303 VARICELLA-ZOSTER VIRUS | | 1 | 1 | 1 | | | | 5 | 8 |
| 1306 HERPES SIMPLEX TYPE 1 | | 4 | 11 | 23 | | 17 | 26 | 24 | 105 |
| 1307 HERPES SIMPLEX TYPE 2 | | | 22 | 54 | | 29 | 64 | 53 | 222 |
| 1399 HERPES VIRUS TYPING PENDING | 5 | | | 6 | 1 | | | 3 | 15 |
| 1401 COXIELLA BURNETI | | | | | | 1 | | | 1 |
| 1502 PICORNA VIRUS-NOT TYPED | | | 16 | | | | 5 | 2 | 23 |
| 1522 RUBELLA VIRUS | | | | 3 | | 2 | | 3 | 8 |
| 1532 HEPATITIS B ANTIGEN | 22 | 1 | 5 | 15 | 2 | 16 | 13 | 10 | 84 |
| 1535 HEPATITIS A ANTIBODY | 2 | | 2 | 9 | | 13 | 2 | 16 | 44 |
| 1541 CHLAMYDIA A - C TRACHOMATIS | | 1 | 8 | 14 | | 45 | 20 | 61 | 149 |
| 1543 CHLAMYDIA A - LGV TYPE | | | | | | | 2 | | 2 |
| 1556 CMV - CYTOMEGALOVIRUS | 1 | | 4 | 29 | 4 | 1 | 11 | 12 | 62 |
| 1563 CORONAVIRUS | | | | 1 | | | | | 1 |
| 1564 ROTAVIRUS | | 13 | 14 | 5 | 34 | 49 | 2 | | 117 |
| 1571 ENTEROVIRUS TYPE 71 (BRCR) | | | | | 18 | | | | 18 |
| 1599 ENTEROVIRUS TYPING PENDING | | 8 | 8 | | 7 | | | | 23 |
| 9992 ROSS RIVER VIRUS | | | | | | | | 3 | 3 |
| 9994 SMALL VIRUS (LIKE) PARTICLE | | 1 | | | | | | | 1 |
| Total | 35 | 59 | 109 | 190 | 154 | 209 | 178 | 225 | 1,159 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 7/7/86 - 21/7/86
Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

| VIRUS OR VIRAL ANTIGEN | No-ill or data | Respiratory | Encephalitis | Meningitis | Paralysis | CNS other unspec | GI | Hepatic | CVS | Urinary | Skin/mucs memb |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 0100 ADENOVIRUS NOT TYPED..... | | 1 | | | | | 1 | | | | |
| 0101 ADENOVIRUS TYPE 1..... | | 3 | | | | | | | | | |
| 0102 ADENOVIRUS TYPE 2..... | | 4 | | | | | 1 | | | | 1 |
| 0103 ADENOVIRUS TYPE 3..... | | | | | | | 1 | | | | |
| 0104 ADENOVIRUS TYPE 4..... | | 1 | | | | | | | | | |
| 0105 ADENOVIRUS TYPE 5..... | | | | | | 1 | 1 | | | | |
| 0106 ADENOVIRUS TYPE 6..... | | | | | | | 1 | | | | |
| 0201 INFLUENZA A VIRUS..... | | | | | | | 1 | | | | |
| 0203 INFLUENZA B VIRUS..... | | 1 | | | | | | | | | |
| 0301 PARAINFLUENZA VIRUS TYPE 1.... | | 12 | | | | | | | | | |
| 0302 PARAINFLUENZA VIRUS TYPE 2.... | | 28 | | | | | | | | | |
| 0303 PARAINFLUENZA VIRUS TYPE 3.... | | 6 | | | | | | | | | |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS)..... | 1 | 94 | | | | | | | | | 1 |
| 0500 RHINOVIRUS (ALL TYPES)..... | 1 | 10 | | | | | | | | | |
| 0600 MYCOPLASMA PNEUMONIAE..... | | 1 | | | | | | | | | |
| 0700 ORNITHOSIS-PSITTACOSIS..... | | 2 | | | | | | | | | |
| 0800 COXSACKIEVIRUSES GROUP A - NOT TYPED..... | | | | | | | | | | | 1 |
| 0816 COXSACKIEVIRUS A16..... | | | | | | | | | | | 1 |
| 0905 COXSACKIEVIRUS B5..... | | | | | | | | | 1 | | |
| 1003 ECHOVIRUS TYPE 3..... | | 1 | | | | | | | | | |
| 1007 ECHOVIRUS TYPE 7..... | | | | | | | | | | | 1 |
| 1011 ECHOVIRUS TYPE 11..... | | 2 | | 3 | | 2 | 1 | | | | |
| 1014 ECHOVIRUS TYPE 14..... | | | | | 1 | | | | | | |
| 1020 ECHOVIRUS TYPE 20..... | | | | | | 1 | | | | | |
| 1021 ECHOVIRUS TYPE 21..... | 1 | | | | | | | | | | |
| 1022 ECHOVIRUS TYPE 22..... | 1 | | | | | | | | | | |
| 1100 POLIOVIRUS NOT TYPED..... | | | | | | | 1 | | | | |
| 1101 POLIOVIRUS TYPE 1..... | | | | | | | 1 | | | | |
| 1200 MUMPS VIRUS..... | | | | 1 | | | | | | | |
| 1300 HERPES VIRUS GROUP-NOT TYPED.. | | | | | | 1 | | | | | 1 |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | | 1 | | | | | | | | | |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS). | 4 | 1 | | | | | | 1 | | | |
| 1303 VARICELLA-ZOSTER VIRUS..... | | 1 | | | | | | | | | 8 |
| 1306 HERPES SIMPLEX TYPE 1..... | 3 | 7 | | | | 1 | | | | | 60 |
| 1307 HERPES SIMPLEX TYPE 2..... | 14 | 1 | | | | | | | | | 55 |
| 1502 PICORNA VIRUS-NOT TYPED..... | | 4 | | | | 1 | 6 | | | | |
| 1522 RUBELLA VIRUS..... | 1 | | | | | | | | | | 4 |
| 1532 HEPATITIS B ANTIGEN..... | 19 | | | | | | | 49 | | | |
| 1535 HEPATITIS A ANTIBODY..... | 5 | | | | | | | 30 | | | |
| 1541 CHLAMYDIA A - C.TRACHOMATIS... | 4 | 1 | | | | | | | | | |
| 1543 CHLAMYDIA A - LGV TYPE..... | 1 | | | | | | | | | | |
| 1556 CMV - CYTOMEGALOVIRUS..... | 9 | 12 | | | | 2 | | 1 | | 9 | |
| 1563 CORONAVIRUS..... | | | | | | | 1 | | | | |
| 1564 ROTAVIRUS..... | | 3 | | | | | 113 | 1 | | | |
| 1571 ENTEROVIRUS TYPE 71 (BRCR).... | | 10 | 1 | 7 | | | | | | | |
| 9994 SMALL VIRUS (LIKE) PARTICLE... | | | | | | | | | | | 1 |
| Total..... | 64 | 207 | 1 | 11 | 1 | 9 | 129 | 82 | 1 | 9 | 134 |

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 7/7/86 - 21/7/86
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 |
|---|---|---|---|---|---|---|---|---|---|---|
| 0103 ADENOVIRUS TYPE 3..... | | | | | | | | | | 1 |
| 0108 ADENOVIRUS TYPE 8..... | 3 | | | | | | | | | |
| 0137 ADENOVIRUS TYPE 37..... | 1 | | | | | | | | | |
| 0201 INFLUENZA A VIRUS..... | | | | | | | 1 | | | |
| 0203 INFLUENZA B VIRUS..... | | | | | | | 1 | | | |
| 0400 RESPIRATORY SYNCYTIAL VIRUS (RS)..... | 1 | | | 1 | | | | 2 | | 1 |
| 0500 RHINOVIRUS (ALL TYPES)..... | | | | | | | | 1 | | 2 |
| 1011 ECHOVIRUS TYPE 11..... | | | | | | | 1 | | 1 | |
| 1102 POLIOVIRUS TYPE 2..... | 1 | | | | | | | | | |
| 1200 MUMPS VIRUS..... | | | 1 | | | | | | 1 | |
| 1301 HERPES SIMPLEX VIRUS NOT-TYPED | | | | | | | | 1 | | |
| 1302 EPSTEIN-BARR VIRUS (EB VIRUS). | 1 | | | 1 | | | | 1 | 1 | |
| 1306 HERPES SIMPLEX TYPE 1..... | 4 | 28 | | | | | | 1 | 1 | |
| 1307 HERPES SIMPLEX TYPE 2..... | | 150 | | | | 1 | | 1 | | |
| 1399 HERPES VIRUS TYPING PENDING... | | 1 | | | | | | | | |
| 1401 COXIELLA BURNETI..... | | | | | | | 1 | | | |
| 1502 PICORNA VIRUS-NOT TYPED..... | | | | | | | | | | 1 |
| 1522 RUBELLA VIRUS..... | | | | | | 1 | | | 2 | |
| 1532 HEPATITIS B ANTIGEN..... | | | | | | | | | 16 | |
| 1535 HEPATITIS A ANTIBODY..... | | | | | | | | | 9 | |
| 1541 CHLAMYDIA A - C.TRACHOMATIS... | 3 | 139 | | | | | | | 2 | |
| 1543 CHLAMYDIA A - LGV TYPE..... | | 1 | | | | | | | | |
| 1556 CMV - CYTOMEGALOVIRUS..... | | 7 | | | | 3 | 2 | 1 | 20 | 1 |
| 1564 ROTAVIRUS..... | | 1 | | | | | | | | |
| 1571 ENTEROVIRUS TYPE 71 (BRCR).... | | | | | | | | 1 | | |
| 9992 ROSS RIVER VIRUS..... | | | | | 3 | | 2 | | | |
| 9994 SMALL VIRUS (LIKE) PARTICLE... | | | | | | | 1 | | | |
| Total..... | 14 | 327 | 1 | 2 | 3 | 5 | 9 | 9 | 53 | 6 |

## NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 3 - 22 February 1986 to 21 March 1986

Bulletin .86/.15

| Disease | N.S.W. | VIC | QLD | S.A. | W.A. | TAS. | N.T. | A.C.T. | Total | CUMULATIVE TOTAL TO DATE FOR YEAR |
|---|---|---|---|---|---|---|---|---|---|---|
| Amoebiasis | | | 2 | | | | | | 2 | 9 |
| Ankylostomiasis | | | | | | | N.N. | | | 2 |
| Anthrax | | | | | | | | | | - |
| Arbovirus infection | 44 | 5 | 109 | 1 | 2 | | | | 161 | 306 |
| Brucellosis | | | 1 | | | | | | 1 | 6 |
| Campylobacter infections | 115 | N.N. | N.N. | 97 | 2 | N.N. | 9 | N.N. | 223 | * 656 |
| Chancroid | | | | N.N. | | N.N. | | | | - |
| Cholera | | | | | | | | | | - |
| Congenital rubella syndrome | | N.N. | N.N. | | | N.N. | | N.N. | | - |
| Diphtheria | | | | | | | 1 | | 1 | 6 |
| Donovanosis | | N.N. | 2 | N.N. | 2 | N.N. | 5 | | 9 | 16 |
| Giardiasis | 29 | N.N. | N.N. | 61 | 7 | N.N. | N.N. | N.N. | 97 | * 262 |
| Genital herpes | 76 | N.N. | 9 | 13 | N.N. | N.N. | 3 | | 101 | 313 |
| Gonococcal ophthalmia neonatorum | | N.N. | N.N. | | N.N. | N.N. | | N.N. | | - |
| Gonorrhoea | 110 | | 57 | 50 | 101 | 2 | 64 | 8 | 392 | 1126 |
| Hepatitis A (infectious) | 30 | 9 | 33 | 40 | 64 | | 2 | 1 | 179 | * 447 |
| Hepatitis B (serum) | 48 | 14 | 60 | 16 | 25 | 2 | 3 | 4 | 172 | * 438 |
| Hepatitis - unspecified | 6 | 1 | 3 | 2 | | N.N | | | 12 | 38 |
| Hydatid disease | | | | | | | | | | 1 |
| Lassa Fever | | N.N. | N.N. | | | N.N. | N.N. | N.N. | | - |
| Legionnaires' disease | 2 | | N.N. | | | N.N. | | N.N. | 2 | 29 |
| Leprosy | | 1 | | | | | | | 1 | 4 |
| Leptospirosis | 4 | 2 | 13 | | 1 | 2 | | | 22 | 46 |
| Lymphogranuloma venereum | 1 | N.N. | N.N. | N.N. | N.N. | N.N. | | | 1 | 1 |
| Malaria | 6 | 12 | 34 | 2 | 1 | | 2 | 2 | 59 | 180 |
| Marburg Disease | | N.N. | N.N. | | | N.N. | N.N. | N.N. | | - |
| Meningococcal infections | 1 | 1 | | | 1 | N.N. | | | 3 | 9 |
| Non-specific urethritis | 298 | N.N. | 1 | 104 | | N.N. | | 2 | 405 | 1148 |
| Ornithosis | | 2 | 1 | 1 | | | | | 4 | 10 |
| Pertussis (whooping cough) | 42 | 4 | N.N. | 36 | 4 | N.N. | 1 | N.N. | 87 | * 322 |
| Plague | | | | | | | | | | - |
| Poliomyelitis | | | | | | | | | | - |
| Q. fever | 5 | | 24 | 1 | | | N.N. | | 30 | 53 |
| Rabies | | N.N | N.N. | N.N | | N.N. | N N. | N.N. | | - |

2.

| DISEASE | N.S.W. | VIC | QLD | S.A. | W.A. | TAS. | N.T. | A.C.T. | Total | CUMULATIVE TOTAL TO DATE FOR YEAR |
|---|---|---|---|---|---|---|---|---|---|---|
| Salmonella infections | 72 | 9 | 84 | 46 | 20 | 6 | 30 | 2 | 269 | 728 |
| Shigella infections | 16 | 5 | 23 | 6 | 7 | | 19 | | 76 | 219 |
| Smallpox | | | | | | | | | | - |
| Syphilis | 32 | | 20 | 4 | 17 | | 77 | 1 | 151 | 413 |
| Tetanus | | 2 | | | | | | | 2 | 3 |
| Trachoma | | N.N. | 1 | | | N.N. | N.N. | | 1 | 5 |
| Tuberculosis (all forms) | 34 | 13 | 10 | 6 | 7 | 2 | 5 | 2 | 79 | 185 |
| Typhoid fever | 2 | 2 | | | | | | | 4 | 13 |
| Typhus (all forms) | | | 2 | | | | | | 2 | 2 |
| Vibrio parahaemolyticus infections | 1 | N.N. | N.N. | | | N.N. | | N.N. | 1 | 4 |
| Yellow Fever | | | | | | | | | | - |
| Yersinia enterocolitica infections | 10 | N.N. | N.N. | 1 | | N.N. | | N.N. | 11 | 22 |

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependant upon voluntary reporting by medical practitioners etc.)

N.N. Not Notifiable

\+ Adjustment to the Cumulative Total since last report:

| | | |
|---|---|---|
| Campylobacter infections | +1 | South Australia |
| Giardiasis | +1 | South Australia |
| Hepatitis A (infectious) | +1 | South Australia |
| Hepatitis B (serum) | +1 | South Australia |
| Pertussis (whooping cough) | +12 | South Australia |

IMPORTANT NOTICE

Our ref 86/4465

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