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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME: A total of 1 448 reports were processed for this period.

Eight cases of Q fever were reported for this period (4 from New South Wales, 2 from South Australia and 2 from Queensland). Occupational exposure data were only available for the two South Australian cases; male meatworkers aged 21 and 24 respectively. None of these eight patients were involved in the Q fever vaccine field trial conducted in South Australia.

Cytomegalovirus was isolated from the leucocytes of an adult male AIDS patient who showed symptoms of progressive dementia.

Herpes Simplex Virus type 1 was isolated from the faeces of a 24 year old male HIV positive patient who presented with persistent diarrhoea.

Adenovirus type 2 was isolated from the faeces of 2 male infants, aged 1 and 3 months respectively, who had died of Sudden Infant Death Syndrome. Adenoviruses infect epithelial cells of mucous membrane, the cornea and other organ systems, and intussusception of infancy has also been ascribed to adenoviruses types 1,2,3 and 5.

Monitoring of viral activities for this period indicates an on-going increase in activity of :-

- RSV, 281 cases compared with 241, 200 and 98 reported for the three previous reporting periods.
- Enterovirus type 71(BRCR), 30 cases compared with 23, 6 and 18 reported for the three previous reporting periods.

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Figures given may be subject to revision.

CHOLERA OUTBREAK: INDONESIA

The Indonesian Press of 7 August 1986 had reported that at least 74 people have died and almost 2 000 have been hospitalised following cholera outbreaks in seven Indonesian provinces.

The Health Ministry had indicated that the deaths had occurred between June and the third week of July following cholera outbreaks in North and West Sumatra, Aceh, East Nusatenggara, Central Sulawesi and Central and East Java.

The worst affected areas were three districts in North Sumatra where 34 people died and 906 were under medical care. The Health Ministry added that none of the areas affected were tourist destinations.

CHOLERA - HEALTH ADVICE FOR THE AUSTRALIAN INTERNATIONAL TRAVELLER

Since a number of countries have revised their cholera vaccination requirements, effective 1 August 1986, the chapter on Cholera appearing in the 1986 revised edition of the booklet 'Health Information for International Travel' published by the Commonwealth Department of Health, is now amended to read:

CHOLERA

Cholera is an acute disease due to Vibrio cholerae which is characterised by profuse; painless diarrhoea which results in muscular cramps due to hypocalcaemia and severe hypotension due to fluid loss. The disease is transmitted from man to man through the ingestion of faecally contaminated food and water.

Cholera vaccine

Properly designed clinical trials with a bivalent vaccine containing Inaba and Ogawa serotypes have been conducted in Bangladesh and the Philippines. These trials demonstrated that in populations with high levels of natural immunity in whom vaccine-induced immunity was further reinforced by abundant current transmission, the efficacy of this vaccine was 50-70% and the duration of protection varied between three to six months. There are no data on the efficacy and duration of protection provided by this cholera vaccine in populations who have no cholera immunity, however reduced efficacy and duration of protection would be expected.

In addition, this cholera vaccine does not prevent the transmission of the disease or eliminate the carrier state.

The vaccine available in Australia is a heat-killed suspension of the Inaba and Ogawa serotypes of the classical biotype of V.cholerae 01, Phenol 0.5% w/v is added as a preservative and each millilitre of vaccine contains 8 000 million organisms of Inaba and Ogawa serotypes.

Recommendation for use

Despite the fact that cholera is endemic in Asia, Africa, the Middle East and parts of Oceania, use of the currently available cholera vaccine is not recommended due to its minimal

efficacy and short duration of action. Emphasis should be placed on the careful selection of food and water by international travellers and temporary residents in countries where V. cholerae infections can be acquired.

Cholera immunisation as a travel requirement

Although the World Health Organization advises that cholera vaccination should no longer be required as a condition for entry into any country in the world, health officials in some countries may not be aware of this recommendation and travellers to cholera endemic countries should receive a single vaccination with cholera vaccine to satisfy entry requirements demanded by these officials.

International Certificates of Vaccination may be required for entry into Albania, Cape Verde, India, Lesotho, Madagascar, Malta, Pakistan, Pitcairn, Somalia and Sudan. A single dose of cholera vaccine will satisfy this requirement.

The dose of vaccine required in this situation is listed below.

Age	dose
10 year and above	0.5 mL
5-9 years	0.3 mL
less than 5 years	0.1 mL

Cholera injections are NOT required for entry into Australia.

Side effects and adverse reactions

Adverse reactions to cholera vaccination usually consists of tenderness and induration at the site of the injection, sometimes associated with systemic symptoms of fever, headache and malaise. Severe adverse reactions involving the central nervous and cardiovascular systems, the skin and kidneys have been reported, although usually as isolated case studies.

Precautions and contraindications

There are no data on the safety of cholera vaccination during pregnancy. Its use in pregnancy should therefore be carefully individualised through risk-benefit considerations.

MALARIA ACQUIRED WITH DOUBLE CHEMICAL PROPHYLAXIS: CHLOROQUINE PLUS FANSIDAR(R) - A CASE REPORT

(based on CDWR Vol 12-24, 14 June 1986)

In January this year, a 21 year old Canadian woman was admitted to hospital for investigation of a febrile syndrome. She had returned 4 days earlier from Zaire where she had spent the previous three months. During her entire stay in Africa she had apparently taken the recommended prophylactic doses of chloroquine (2 tablets once per week) plus Fansidar(R) (1 tablet once per week). She took her last prophylactic dose on the day she left the malarial zone. That evening she experienced fever and headache which spontaneously subsided and returned 48 hours later on the day of her arrival in Canada. The next morning she experienced periods of fever followed by chills and an intense headache in the evening.

At admission the patient complained of dizziness with clouded vision, difficulty in concentrating, pain at the nape of her neck, headache, myalgia, asthenia, and soft stools. Clinical examination revealed a 39.7°C fever, conjunctival hyperaemia, fullness in the left hypochondrial region with the liver palpable 2.5 cm below the rib cage.

On the following day the patient felt better but the fever recurred the next day. A blood smear taken during the febrile episode revealed young undifferentiated trophozoites of Plasmodium, with a 1% degree of parasitaemia. The patient was treated with 1.5 g (base) of chloroquine phosphate, divided in 4 doses over 3 days, followed by an oral dose of Fansidar(R) (3 tablets). The fever gradually subsided within 48 hours of treatment and the blood smear no longer showed Plasmodium 24 hours after the Fansidar(R) was taken. The patient was discharged with a prescription for primaquine (15 mg base once daily) for 14 days.

Two days following the end of the primaquine treatment, the patient returned to hospital with recurrent headaches accompanied by hot spells. She was readmitted with a 38.8°C fever and petechiae which were visible from her ankles to her knees. The spleen was palpable 3.5 cm below the rib cage. Platelet count was normal but haemoglobin had decreased from 106 to 95 g/L in 16 days. Lymphocytosis of 35% and monocytosis of 12% were detected. Blood smears this time revealed trophozoites suggestive of P. vivax and gametocytes (banana-shaped) typical of P. falciparum. Treatment for chloroquine-resistant P. falciparum (CRPF) was initiated with quinine sulfate (600 mg orally, 3 times a day for 3 days) and pyrimethamine (25 mg orally, twice a day for 3 days). The patient responded favourably to treatment, became afebrile 48 hours after treatment and the clinical and haematological parameters returned to acceptable levels. The patient was discharged 3 days later with a prescription for tetracycline (250 mg orally, 4 times a day for 7 days) since sulfadiazine was not available. Subsequent serial weekly blood smears for the following 5 weeks were parasite-free. One month after the end of the treatment, the patient had fully recovered and was ready to resume normal activities.

CDI Editorial Comment

The management of malaria involves accurate diagnosis, positive identification of the malaria parasite species and appropriate use of therapeutic medications. These principles do not appear to have been adhered to in the management of the above case:-

- the malarial species were not initially identified. However as chloroquine-resistant P. vivax malaria has not been adequately documented anywhere in the world, the likelihood is that the parasite was P. falciparum.

- during the first hospitalisation, no rationale was given to justify treating the patient with the same combination of drugs which had earlier provided inadequate prophylaxis. As P. falciparum has no exo-erythocytic stages, a single dose of 45 mg primaquine base would have been sufficient to eliminate the sexual forms of this parasite. In addition, the daily 15 mg primaquine prescribed for the 14 days after hospital discharge appeared inappropriate for a person

living in an area experiencing winter weather conditions where anopheline mosquito activity would be expected to be nil.

- during the relapse the trophozoites were suggested to be P. vivax and sexual forms of P. falciparum were also noted, yet the treatment for chloroquine-resistant P. falciparum malaria was instituted.

AIDS SURVEILLANCE - AUSTRALIA

To 3 September 1986, 282 cases of AIDS fulfilling the criteria of case definition have been reported to the National Health and Medical Research Unit in AIDS Epidemiology and Clinical Research. The distribution of those patients by State or Territory of notification and by risk group are shown below:-

Table 1: AIDS patients by State or Territory of notification

STATE/TERRITORY	CASES			DEATHS		
	Male	Female	Total	Male	Female	Total
NSW	188	6	194	84	6	90
VIC	43	-	43	22	-	22
QLD	21	2	23	14	2	16
WA	13	2	15	5	-	5
SA	2	-	2	-	-	-
NT	2	-	2	1	-	1
TAS	1	-	1	1	-	1
ACT	2	-	2	1	-	1
	272	10	282	128	8	136

Table 2: AIDS patients by risk category

RISK GROUP	CASES	DEATHS
Homo-/Bi-sexual	249	108
IV drug abuser	1	-
Homo-/Bi-sexual IV drug abuser	4	2
Blood transfusion recipient	21	20
Person with haemophilia	4	4
Heterosexual transmission	2	2
None of the above	1	-
	282	136

PREVENTION OF PERINATAL HEPATITIS B

(Based on California Morbidity No 20 May 23 1986)

Transmission of hepatitis B virus (HBV) plays an important role in propagating high rates of hepatitis B infection in many groups including Australian Aborigines, and many South East Asian and Pacific island communities. The rate of development of the chronic HBV carrier state in infants born of HBsAg-positive mothers may be reduced by as much as 80-90% with the timely use of hepatitis B immunoglobulin (HBIG) and hepatitis B vaccine.

In 1984 the Immunisation Practices Advisory Committee of the United States Public Health Services (USPHS) and California's Infectious Disease Branch recommended that infants born of HBsAg positive women receive HBIG (0.5mL Im) within 12 hours of birth and hepatitis B vaccine (10ug/0.5mL) at birth and at 1 and 6 months after the original dose.

A recent retrospective medical records review in the San Francisco region has determined the extent to which Asian and Pacific island women delivering at a county hospital during the time period 1984-85 were screened antenatally for HBsAg, and ascertained the timing and completeness of HBIG and hepatitis B vaccine administration to infants born to HBsAg-positive mothers.

545 women in Asia or the Pacific islands gave birth to a total of 572 infants at the hospital. 484 of the 545 women (89%) were screened antenatally for HBsAg. Antenatal HBsAg screening ranged from a high of 100% (100/100) in Laotians to a low of 54% (32/59) in Filipinos.

Forty-nine of the 484 women (10.1%) were HBsAg-positive. For countries having at least one HBsAg positive woman, HBsAg-positive ranged from a high of 20.7% (6/29) in women from Tonga to a low of 3.1% (1/32) in Filipino women; HBsAg-positivity varied from a high of 17.7% (5/29) in women from Tonga to a low of 0% in Filipino and Korean women.

Fifty two infants were born to the 49 HBsAg-positive mothers. 40/52 (77%) of the infants received HBIG within 12 hours of birth; 7 infants (13%) got HBIG more than 12 hours after birth; and 5 infants (10%) received no HBIG. Analysis of vaccine utilisation excluded 14 of the 52 infants: 12 infants received an experimental hepatitis B vaccine as part of an on-going study by the New York Blood Centre, while 2 other infants were born after December 20, 1985 (and thus the 6-month follow-up period had not elapsed at the time of data analysis). Of the remaining 38 infants, 30 (79%) received HBIG and at least one dose of vaccine; 3 (8%) received HBIG only; 2 (5%) got vaccine only; and 3 (8%) got neither HBIG nor vaccine.

Only 14 of the 38 infants (37%) received the recommended treatment of HBIG and 3 doses of vaccine.

CDI Editorial Comment

Subsequent to the Commonwealth/State Meeting on Hepatitis B in June 1984, the Commonwealth Department of Health is currently preparing a mechanism to control Hepatitis B vertical transmission by sponsoring a program of vaccine/immunoglobulin administration to all neonates born to women who belong to a high rish group for hepatitis B. The implementation of this program is currently proceeding and should be finalised in the very near future.

The present report from California indicates that even in a hospital setting, there may be problems with delivery of hepatitis B prophylactic measures. The reason for poor compliance with the recommended procedures in this study were not stated.

In respect of 'at risk' babies born in hospital, it should be possible to assure a near 100% compliance rate with the first dose of vaccine and HBIG, provided that adequate information is provided to health care professionals and optimal patient education is available as to the need for vaccination.

Vaccination of babies born at home may pose a greater challenge. It is important that prophylactic treatment of babies at risk is undertaken soon after birth. In addition, it is essential that the second and third doses of vaccine be given. It has been estimated that less than 10% of babies receiving only the first dose are adequately protected against the disease. Medical practitioners should be made aware of the great importance of parent education, particularly where language problems or cultural background may pose a barrier, in the parents' eyes, to the medical treatment of an apparently healthy child.

AMODIAQUINE-INDUCED HEPATITIS

As previously reported in the CDI (No. 86/7: 5-6), several cases of agranulocytosis have been associated with the use of amodiaquine for malaria prophylaxis[1,2,3]. A recent report from France documents 7 cases of amodiaquine - induced hepatitis [4].

The 7 patients had no history of alcohol abuse or diseases of the liver or biliary tract. There was no evidence of recent infections with hepatitis A or B viruses, cytomegalovirus, Epstein-Barr virus or herpes simplex virus. In addition, no other factors likely to result in the induction of hepatitis were identified.

There was a close temporal relationship between the administration of amodiaquine and the onset of hepatitis; the patients developed hepatitis after receiving the drug for malaria prophylaxis for 4 to 15 weeks.

Symptoms such as asthenia and vomiting preceded jaundice or, in the absence of jaundice, were the only manifestations of hepatitis. Four patients experienced a relatively minor form of hepatitis: jaundice was mild or absent; serum alanine aminotransferase (ALT) levels were slightly elevated; and recovery was virtually complete within 3 months after the administration of amodiaquine ceased. The other 3 patients suffered a major form of hepatitis: jaundice was intense and persisted for 3 to 6 months; serum ALT levels were markedly elevated; the prothrombin time was decreased; and liver function tests were still abnormal 7 to 27 months after the onset of hepatitis.

In two of the patients the readministration of the drug was followed by a rapid increase in serum ALT levels. Although the mechanism for amodiaquine hepatotoxicity has not been established, this observation is consistent with an immunoallergic mechanism for amodiaquine-induced hepatitis[4].

CDI Editorial Comment

Amodiaquine is not currently available in Australia but is widely used in many malaria endemic countries. As a

consequence of the possible risk of agranulocytosis associated with the use of amodiaquine, the U.S. Centers for Disease Control recommended that the drug no longer be used for malaria prophylaxis[3]. The possibility of amodiaquine-induced hepatitis adds further weight to this recommendation.

REFERENCES

1. Lancet (1986) i: 411-414
2. Lancet (1986) i: 556
3. MMWR (1986) 35: 165-166
4. Ann Intern Med (1986) 104: 801-803.

HEPATITIS B ASSOCIATED WITH JET INJECTIONS IN CALIFORNIA.
Based on California morbidity No 23, 13 June 1986

During routine investigation of hepatitis B (HB) case reports, an epidemiologist in a local health authority in Los Angeles noted in March of 1985 that 3 HB cases had each received injections at the same weight reduction clinic (Clinic A) before disease onset. When a review of previous case records and of newly reported cases identified 5 additional HB cases at Clinic A, the California Department of Health Services joined the investigation.

Clinic A belongs to a chain of 29 similarly operated clinics in Southern California. Patrons of the clinic typically received daily parenteral injections of human chorionic gonadotropin (HCG). These were usually given by jet injectors (Med-E-Jet Corp., Cleveland, Ohio), although some patrons received injections by single-use disposable needle and syringe. A standard regimen consisted of 30 doses. However, there was much individual variability in duration of treatment and number of doses received.

The investigation focused on a cohort of 341 persons who attended Clinic A in the first 6 months of 1985. Clinical histories, a review of risk factors for acquiring HB, serological testing for HB markers and quantitation of parenteral exposures at the clinic were obtained from 287 (84.2%) cohort members. For comparison, 93 new patrons at Clinic A at the time of their initial visit and a random sample of 100 prior as well as 70 new patrons at a non-implicated clinic of the chain in Long Beach (Clinic B) were tested for serological evidence of HB infection.

Ultimately, 31 cases of clinical HB were identified among patrons of Clinic A. Onset dates ranged from January 1984 to November 1985, with 84% after February 1985. Only 2 (6%) of the clinical cases had other identified risk factors for acquiring HB infection in the 6 months before their illness.

The serological survey revealed that 21% (60/287) of the cohort attending Clinic A in the first 6 months of 1985 had evidence of acute HB infection, including 27 clinical cases and 33 subclinical cases (anti-HBc IgM positive), compared with none of the 93 new patrons of Clinic A. When all serologic markers of HB infection were included, 42.5% (122/287) of the cohort of prior Clinic A patrons had evidence of HB infection compared

with 6.5% (6/93) of new patrons at Clinic A, 8% (8/100) of persons having attended Clinic B and 6% (4/70) of new patrons at Clinic B. Prior attendance at Clinic A was significantly associated with being seropositive for a marker of HB infection.

Among Clinic A cohort members, exposure to the jet injector and HCG were both significantly associated with the development of acute HB infection. However, when cohort members were classified by type of parenteral injection (jet gun only vs. syringe only during the study period), it was found that 24% (57/239) of the jet gun only group developed acute infection compared with none of the syringe only group.

The protocols at Clinic A for administering the shots and cleaning the jet injectors appeared adequate. However, some patients did report that they had sustained lacerations and bruising in the course of receiving jet injections. In vitro studies of this particular brand of jet injector carried out at the Centers for Disease Control, Atlanta, Georgia (CDC) showed that if this gun is contaminated with HBsAg positive serum, there is the potential for subsequent HB transmission. No cases of hepatitis B have been identified among persons treated at Clinic A since July 2 1985 when the jet injectors were removed and no cases to date have been associated with any other clinic in the chain.

CDI editorial comment

This report emphasises the risks of infectious disease transmission associated with reuseable therapeutic devices. As noted in an earlier CDI article[1] such devices should be autoclaved after thorough cleaning. Disinfection is inadequate, particularly where there is a risk of transmission of highly infectious diseases, such as hepatitis B. This disease may be transmitted by inoculation with as little as 1 uL of infected blood.

Reference

1. Communicable Disease Intelligence bulletin 86/9

SPREAD OF HEPATITIS B BY ADOPTED CHILDREN IN SWEDEN

In Sweden it is extremely rare to find native-born children carrying hepatitis B virus. However, most of the adopted children in this country come from regions of the world where the disease is endemic and where widespread carriage or immunity is common. Of the 1 400 to 1 900 foreign children adopted by Swedish families each year, 66% come from South East Asia, 30% from Latin America, 3% from Europe and 1% from Africa.[1]

In the period from 1977 to 1981, a total of 114 HBsAg - positive, adopted, foreign children were reported to the Department of Epidemiology at the National Bacteriological Laboratory in Stockholm. Of these, 96 could be traced and subsequently followed up in an epidemiological survey designed to evaluate the risk of hepatitis B transmission in family settings.[1] The majority of the children reported (92%) were of South East Asian origin.

A total of 36 (37.5%) HBsAg - positive children were found to be responsible for the transmission of hepatitis B to 54 family members, there being 35 cases of clinical disease and 19 cases of subclinical disease detected. The risk of transmission by younger children was high; 16/43 children aged $<$ 1 year on arrival caused 26 of the secondary cases, 10/21 children aged 1-2 years on arrival caused 14 cases and 7/11 children aged 2 years but $<$ 3 years on arrival caused 10 cases. Of the 21 children aged $\geq$ 3 years on arrival, 3 were responsible for 4 secondary cases.

The greatest risk of transmitting acute hepatitis B was found to occur during the first year after the arrival of the child, when 22 of the cases of acute disease occurred. A further 8 clinical cases occurred in the second year with only sporadic cases occurring $\geq$ 24 months after arrival.

Of the 96 HBsAg - positive children, 65 were HBeAg positive and 5 had anti-HBe. The HBe status of 26 children was not known. Liver enzyme data was available for 67 of the children. Of these, 15(22%) had normal values while 52(78%) had an increase in liver transferases. During the follow-up, 4 children became anti-HBe positive at the ages of 3,7 (2 children) and 8 years. None of the children with known anti-HBe status was demonstrated to have spread hepatitis B.

As not all members of families containing adopted children were tested for hepatitis B markers, it is possible that the overall transmission of the virus may have been greater than actually detected during the survey. The study clearly demonstrated that there was a high risk of hepatitis B transmission by HBsAg - positive children within family units, an observation that is consistent with previous findings for adopted children in Sweden.[2]

REFERENCES

1. Scand J Infect Dis (1986) 18:105-109
2. Scand J Infect Dis (1978) 10:161-163

REVISED NOTIFIABLE DISEASES AUSTRALIA - 1985

The NSW notifiable disease statistics as reported in CDI 86/11 for the period 1 January to 31 December 1985 have now been revised. The corrected figures for NSW are included in the revised 'Notifiable Diseases Reported in Australia - 1 January 1985 to 31 December 1986' appearing on page 14 of this issue.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD 19/8/86 - 1/9/86 BULLETIN NUMBER 86/18
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	4		7		8	1	8	1	29
0101 ADENOVIRUS TYPE 1				1				4	5
0102 ADENOVIRUS TYPE 2				2	1	4		3	10
0103 ADENOVIRUS TYPE 3				1		1			2
0105 ADENOVIRUS TYPE 5	2					4		1	7
0107 ADENOVIRUS TYPE 7								1	1
0108 ADENOVIRUS TYPE 8				1					1
0111 ADENOVIRUS TYPE 11	1								1
0119 ADENOVIRUS TYPE 19	1								1
0128 ADENOVIRUS TYPE 28	1								1
0199 ADENOVIRUS TYPING PENDING			1		1				2
0201 INFLUENZA A VIRUS	1				2				3
0203 INFLUENZA B VIRUS	1								1
0301 PARAINFLUENZA VIRUS TYPE 1				1	4		1	1	7
0302 PARAINFLUENZA VIRUS TYPE 2	1			2	4	7	2		16
0303 PARAINFLUENZA VIRUS TYPE 3	1			2	2	1	3		9
0399 PARAINFLUENZA VIRUS TYPING PENDING				1					1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	36	49	20	48	54	8	62	4	281
0500 RHINOVIRUS (ALL TYPES)	2			3	10	13		2	30
0600 MYCOPLASMA PNEUMONIAE	3		3			1	1	4	12
0700 ORNITHOSIS-PSITTACOSIS								1	1
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED						1			1
0906 COXSACKIEVIRUS B6						1			1
1005 ECHOVIRUS TYPE 5	1								1
1007 ECHOVIRUS TYPE 7								1	1
1008 ECHOVIRUS TYPE 8						1			1
1011 ECHOVIRUS TYPE 11	4	1	2	1				7	15
1012 ECHOVIRUS TYPE 12						1			1
1014 ECHOVIRUS TYPE 14				1		1			2
1020 ECHOVIRUS TYPE 20				1					1
1021 ECHOVIRUS TYPE 21				2				1	3
1100 POLIOVIRUS NOT TYPED			2						2
1101 POLIOVIRUS TYPE 1				1		3		1	5
1102 POLIOVIRUS TYPE 2						1			1
1103 POLIOVIRUS TYPE 3			1	2		2			5
1200 MUMPS VIRUS	2							2	4
1300 HERPES VIRUS GROUP-NOT TYPED	15			4				4	23
1301 HERPES SIMPLEX VIRUS NOT-TYPED								2	2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	8						2	8	18
1303 VARICELLA-ZOSTER VIRUS	3			1		1		4	9
1306 HERPES SIMPLEX TYPE 1	23			31		24	37	15	130
1307 HERPES SIMPLEX TYPE 2	56			66		25	77	65	289
1399 HERPES VIRUS TYPING PENDING					2				2
1401 COXIELLA BURNETI	4					2	2		8
1502 PICORNA VIRUS-NOT TYPED	1		3				19	6	29
1521 MEASLES VIRUS	1								1
1522 RUBELLA VIRUS				5		2			7
1532 HEPATITIS B ANTIGEN	30		9	23		10	13	11	96
1535 HEPATITIS A ANTIBODY	2		1	5		10	3	6	27
1541 CHLAMYDIA A - C TRACHOMATIS						40	21	45	106
1556 CMV - CYTOMEGALOVIRUS	2		2	20	4	5	17	17	67
1563 CORONAVIRUS								2	2
1564 ROTAVIRUS	26	9	28	11	20	16			110
1571 ENTEROVIRUS TYPE 71 (BRCR)	2			23		5			30
1599 ENTEROVIRUS TYPING PENDING		1	9		3				13
9992 ROSS RIVER VIRUS			3				6	1	10
9994 SMALL VIRUS (LIKE) PARTICLE		2		1					3
9995 DENGUE								1	1
Total	234	62	91	260	115	191	274	221	1,448

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 19/8/86 - 1/9/86
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.....							3				
0101 ADENOVIRUS TYPE 1.....		1					2		1		
0102 ADENOVIRUS TYPE 2.....		4		1			2				1
0103 ADENOVIRUS TYPE 3.....		2									
0105 ADENOVIRUS TYPE 5.....	1	3					2				
0128 ADENOVIRUS TYPE 28.....							1				
0201 INFLUENZA A VIRUS.....		3									
0301 PARAINFLUENZA VIRUS TYPE 1....		7									
0302 PARAINFLUENZA VIRUS TYPE 2....		16									
0303 PARAINFLUENZA VIRUS TYPE 3....		8					1				
0399 PARAINFLUENZA VIRUS TYPING PENDING.....		1									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	4	272		1			3		1	1	3
0500 RHINOVIRUS (ALL TYPES).....		1									
0600 MYCOPLASMA PNEUMONIAE.....	1	12									1
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....											1
1005 ECHOVIRUS TYPE 5.....				1							
1007 ECHOVIRUS TYPE 7.....				1							
1008 ECHOVIRUS TYPE 8.....		1									
1011 ECHOVIRUS TYPE 11.....	1	7		3			1				1
1012 ECHOVIRUS TYPE 12.....	1										
1014 ECHOVIRUS TYPE 14.....				1			1				
1021 ECHOVIRUS TYPE 21.....	1			1		1					
1101 POLIOVIRUS TYPE 1.....		3					2				
1102 POLIOVIRUS TYPE 2.....							1				
1103 POLIOVIRUS TYPE 3.....		1					1				
1200 MUMPS VIRUS.....	1										
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	5	1				1		1			
1303 VARICELLA-ZOSTER VIRUS.....											8
1306 HERPES SIMPLEX TYPE 1.....	5	2	2		1						60
1307 HERPES SIMPLEX TYPE 2.....	16	1									73
1502 PICORNA VIRUS-NOT TYPED.....	1	9		1			6				6
1521 MEASLES VIRUS.....											
1522 RUBELLA VIRUS.....	1										
1532 HEPATITIS B ANTIGEN.....	29							60			
1535 HEPATITIS A ANTIBODY.....	7							16			
1556 CMV - CYTOMEGALOVIRUS.....	6	15				4		2	1	7	
1563 CORONAVIRUS.....		1									
1564 ROTAVIRUS.....		2					107			1	
1571 ENTEROVIRUS TYPE 71 (BRCR)....	2	7		4							14
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
Total.....	82	381	2	14	1	6	135	79	3	9	173

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 19/8/86 - 1/9/86
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
0101 ADENOVIRUS TYPE 1.....	1									
0102 ADENOVIRUS TYPE 2.....			1							2
0103 ADENOVIRUS TYPE 3.....								1		
0105 ADENOVIRUS TYPE 5.....								1		
0107 ADENOVIRUS TYPE 7.....									1	
0108 ADENOVIRUS TYPE 8.....	1									
0111 ADENOVIRUS TYPE 11.....							1			
0119 ADENOVIRUS TYPE 19.....	1									
0203 INFLUENZA B VIRUS.....			1							
0301 PARAINFLUENZA VIRUS TYPE 1....					1					
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1						1	2		
0906 COXSACKIEVIRUS B6.....									1	
1020 ECHOVIRUS TYPE 20.....								1		
1103 POLIOVIRUS TYPE 3.....									1	1
1200 MUMPS VIRUS.....							1	1		
1301 HERPES SIMPLEX VIRUS NOT-TYPED	1									
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			6				1	2	2	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	8	52							2	
1307 HERPES SIMPLEX TYPE 2.....		203								
1401 COXIELLA BURNETI.....					2		4	2	2	
1502 PICORNA VIRUS-NOT TYPED.....		1			1			1		1
1521 MEASLES VIRUS.....							1			
1522 RUBELLA VIRUS.....				1	2			1	1	
1532 HEPATITIS B ANTIGEN.....									7	
1535 HEPATITIS A ANTIBODY.....									4	
1541 CHLAMYDIA A - C.TRACHOMATIS...		106								
1556 CMV - CYTOMEGALOVIRUS.....		5	1	4		4		5	17	
1563 CORONAVIRUS.....		1								
1564 ROTAVIRUS.....										1
1571 ENTEROVIRUS TYPE 71 (BRCR)....							1	4		
9992 ROSS RIVER VIRUS.....					9			4		
9994 SMALL VIRUS (LIKE) PARTICLE...								1		
9995 DENGUE.....							1			
Total.....	13	368	9	5	15	4	11	26	39	5

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

1 January 1985 to 31 December 1985

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	TOTAL
Amoebiasis	9	52	11	10	3			2	87
Ankylostomiasis			16	27			N.N.		43
Anthrax	1								1
Arbovirus infection	76	5	575	1	3				660
Brucellosis	4	1	16		1				22
Campylobacter infections	1,025	N.N.	N.N.	1,242	36	N.N.	40	N.N.	2,343
Chancroid	3		1	N.N.		N.N.	1		5
Cholera		1	1						2
Congenital rubella syndrome	1	N.N.	N.N.	2		N.N.		N.N.	3
Diphtheria			2				15		17
Donovanosis	1	N.N.	26	N.N.		N.N.	46		73
Giardiasis	342	N.N.	N.N.	735	14	N.N.	N.N.	N.N.	1,091
Genital herpes	1,054	N.N.	355	256	N.N.	N.N.	31	11	1,707
Gonococcal ophthalmia neonatorum		N.N.	N.N.		N.N.	N.N.	14	N.N.	14
Gonorrhoea	1,855	1,274	1,213	631	1,690*	52	807	83	*WA 7,605
Hepatitis A (infectious)	200	72	262	139	116	4	54	1	848
Hepatitis B (serum)	548	151	364	182	305	9	67	19	1,645
Hepatitis - unspecified	68	3	5	2	32	N.N	12		122
Hydatid disease	8		3			2		1	14
Lassa Fever		N.N	N.N.			N.N.	N.N.	N.N.	-
Legionnaires' disease	16	4	N.N.	5	3	N.N.		N.N.	28
Leprosy	16	6	1	1	5	1	8		38
Leptospirosis	43	34	76	9	9	14			185
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.	5		5
Malaria	132	99	72	47	37	5	13	16	421
Marburg Disease		N.N.	N.N.			N.N.	N.N.	N.N.	-
Meningococcal infections	21	9	8	9	5	N.N.		1	53
Non-specific urethritis	3,629	N.N.	104	1,139		N.N.		N.N.	4,872
Ornithosis	1	5	2	7	2				17
Pertussis (whooping cough)	303	140	N.N.	136	7	N.N.	1	N.N.	587
Plague									-
Poliomyelitis									-
Q. fever	33	2	112	52	3		N.N.		202
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
Salmonella infections	1,002	158	427	391	169	72	373	76	2,668
Shigella infections	149	30*	135	84	82	1	252	1	*VIC 734
Smallpox									-
Syphilis	1560	105	332	223	336*	2	951	14	*WA 3,523
Tetanus	1	1	4	4	1				11
Trachoma	1	N.N.	3		59	N.N.	N.N.		63
Tuberculosis (all forms)	387	293	142	99	110	1	33	23	1,088
Typhoid fever	16	9	5					1	31
Typhus (all forms)	1	2*	6		1				*VIC 10
Vibrio parahaemolyticus infections	4	N.N.	N.N.			N.N.		N.N.	4
Yellow Fever									-
Yersinia enterocolitica infections	48	N.N.	N.N.	10		N.N.	2	N.N.	60

(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 dependent upon voluntary reporting by medical practitioners etc.)

N.N. Not Notifiable

* Unconfirmed figures.