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Communicable Diseases Intelligence

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Contents:

Editor Dr I.F. Cook

VIRUS REPORTING SCHEME: A total of 1 096 reports were processed for this period.

Three cases of Q fever were reported, 2 from New South Wales (a 10 year old male and a 37 year old female) and a 56 year old male from Western Australia. No details of occupational exposure were available for these patients, however none was involved in the Q fever vaccine field trial conducted in South Australia.

Measles virus was isolated from the cerebrospinal fluid of an 11 year old male who has been diagnosed as having subacute sclerosing panencephalitis (SSPE).

Cytomegalovirus was isolated from the lung lavage of a 24 year old male presenting with severe urinary tract infection and an atypical pneumonia manifested as a diffuse interstitial infection of the lower respiratory tract.

Specific IgM antibody to rubella virus was detected in the blood of a 30 year old woman who was 17 weeks pregnant. It is not known whether this woman was previously vaccinated against rubella.

Herpes Simplex virus was isolated from the throat swabs of a 12 year old male with acute lymphoblastic leukaemia (ALL) who presented with fever, pharyngitis and ulceration of the mouth and tongue.

Poliovirus type 2 was isolated from the post-mortem specimens of the digestive tract of a 2 month old male who died of Sudden Infant Death Syndrome.

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FULMINANT HEPATITIS B IN SUCCESSIVE FEMALE SEXUAL PARTNERS OF TWO ANTI-HBe-POSITIVE MALES

Fulminant hepatitis B, although rare in incidence, is lethal in 60-90% of cases and is highly correlated with age. In most patients who survive, complete restoration of the hepatic parenchyma and normal liver function is the rule. However patients who develop confluent hepatic necrosis have a poor prognosis, and 15-30% of those who survive develop chronic active hepatitis.

Since HBsAg can be detected in most body fluids including saliva, nasopharyngeal washings, semen, menstrual fluid, and vaginal secretions as well as in blood, transmission from carriers to close contacts by the oral route or by sexual or other intimate exposure can occur. There is particularly strong evidence that persons with subclinical infection can transmit the disease. Carriers of HBsAg can also infect long-term homosexual and heterosexual partners, although the precise mechanism of transmission is not clear. Transmission by the faecal-oral route has not been documented.

The present article (Lancet (1986) ii: 538-40) describes two separate families in which consecutive, unrelated female partners of a symptom-free, male, HBsAg-positive carrier died of fulminant hepatitis B.

FAMILY I

A 27 year old woman, married as a second wife to subject I for 12 months, died from severe cerebral oedema within 24 hours of admission to hospital with fulminant hepatic failure (grade 4 encephalopathy) following 2 days of malaise and jaundice. Her prothrombin time was 72 s prolonged, serum aspartate aminotransferase (AST) 1800 IU/L.

The family medical history revealed that subject I's first wife had also died of fulminant hepatic failure, 6 years earlier. She was a 29 year-old woman (unrelated to the second wife) who was admitted to hospital with a grade 4 encephalopathy following a 2-week illness commencing with malaise and jaundice. She died within 72 hours of admission from severe cerebral oedema. Her maximum prothrombin time was 178s prolonged, AST > 1000 IU/L. She had been married to subject I for 10 months.

Investigation of subject I, a 41 year-old, symptom-free, male, after the death of his second wife revealed that:-

- he was seropositive for HBsAg (1/6400) and anti-HBe
- he was seronegative for IgM anti-HBc and antibodies against hepatitis delta virus
- his serum was negative for DNA polymerase activity and HBV DNA
- liver serum biochemistry was normal
- histology of a liver biopsy sample, taken 6 months later, showed chronic persistent hepatitis.

Family II

A 45 year-old woman, engaged to subject II, was admitted to hospital with fulminant hepatic failure (grade 2 encephalopathy progressing to grade 4) following 6 days of epigastric pain and 2 days of vomiting and jaundice. She died within 72 hours of admission from severe cerebral oedema. Her maximum prothrombin time was 202s prolonged, AST 2750 IU/L. She had had regular sexual contact with subject II for 6 months before death.

The family medical history revealed that subject II's wife (a 54 year old woman, unrelated to the fiancee) had also died of fulminant hepatic failure, 1 year earlier. She died from severe cerebral oedema within 72 hours of hospital admission with grade 3 encephalopathy progressing to grade 4 following 2 weeks of a flu-like illness and 1 day of jaundice. Her maximum prothrombin time was 170s prolonged, AST 3132 IU/L. She had been married to subject II for 30 years.

Histology of a post-mortem liver biopsy sample showed massive confluent necrosis in both women who were:-

- seropositive for HBsAg and IgM anti-HBc,
- seronegative for IgM antibodies against hepatitis A virus and total antibodies against hepatitis delta virus.

Subject II, a 53 year old, symptom free, male, was investigated after the death of his first wife:-

- seropositive for HBsAg ($>1/512$), HBeAg, and total (IgM + IgG) anti- HBc,
- liver serum biochemistry was normal except for
  - raised alkaline phosphatase (185 IU/L, normal $<$ 100 IU/L)
  - AST levels (65 IU/L, normal $<$ 40 IU/L).

One year later, at the time of his second partner's death he was:-

- still HBsAg positive ($>1/512$) but had become anti- HBe positive
- seronegative for delta antigens and antibodies
- found to have - normal serum AST levels (23 IU/L) but
  - elevated serum alkaline phosphatase (152 IU/L).

Seven months later, when molecular hybridisation was carried out, his status was reported as:-

- seropositive for HBsAg and anti- HBe
- negative for DNA polymerase activity and HBV DNA
- histology of a liver biopsy showed persistent hepatitis with pronounced fibrosis
- aspermic following a vasectomy performed 2 months prior to his fiancee's death.

The presence of HBeAg, or a high concentration of HBsAg, in a person's serum may be a useful marker for infectivity. Conversely, lower levels of infectivity correlate with the presence of anti- HBe. Hence the risk of transmission of hepatitis B virus (HBV) by sexual contact is well recognised for HBeAg-positive subjects but is thought to be much less for those who are anti-HBe positive.

In the above cases it appeared that some chemical, humoral, and/or infective factors localised to the body secretions together with HBV accounted for the extraordinary virulence in the women but not in the men.

After recent conversion from seropositivity to HBeAg to anti-HBe, HBV DNA and DNA polymerase activity may be retained in serum. Although a recent seroconversion could be implicated in subject II, both men were not only positive for anti-HBe but also seronegative for DNA polymerase activity and HBV DNA when their second partners died.

Despite the lack of serological markers of viral replication which would indicate high infectivity, both men had evidence of monomeric HBV DNA in various secretions, including seminal fluid, 6 months after they were shown to be anti-HBe positive. Electrophoretic analysis of DNA from body fluids and liver indicated:

- the 3.2 kb band for HBV DNA in samples of serum, seminal fluid, saliva and liver
- the 4.0 kb band in seminal fluid samples probably representing the nicked-circle configuration of the HBV genome rather than viral integration
- the 6.4 kb band in patient I's sputum, the dimeric form of HBV DNA.

These distinctions are important since the non-integrated forms of HBV are more likely to be associated with transmission.

Integrated HBV DNA was not detected in either man's liver biopsy specimens. The persistence of serum HBsAg with HBV DNA in body fluids, but not in serum, appeared to indicate that viral replication can be maintained at privileged extrahepatic sites that can evade the host's immune cytotoxic attack.

In subject II who was known to be aspermic at the time of analysis, an attempt to detect free and integrated forms of HBV DNA in leucocytes indicated that monomeric (3.2 kb) but not integrated HBV DNA was present in mononuclear cell preparations from peripheral blood as well as seminal fluid pellets. In subject I there was some indirect evidence for the presence of HBV DNA in leucocytes, 3.2 kb bands were detected in seminal fluid pellets shown by microscopy to contain leucocytes and in sputum during a respiratory tract infection.

In studies where HBV DNA, as well as monomeric HBV DNA, has been detected in the peripheral blood leucocytes of HBeAg - positive patients, higher molecular weight HBV DNA sequences indicative of polymeric or integrated forms were found in over half of the patients, but sequences less than 3.2 kb, indicative of viral replicative intermediates, were rare. However, molecular biology studies of HBV strongly point to the 3.2 kb monomer as representing a stage of viral replication. Even if replication has ceased the presence of complete viral genome suggests the potential for transcription of viral genes and particle assembly.

Until such time as persistence of HBV in body secretions, particularly seminal fluid, is known and the potential infectivity defined, HBsAg-positive subjects must be considered potentially infectious, at least by way of sexual contact, regardless of the lack of serological evidence for active viral replication.

THE SAFETY OF HEPATITIS B VACCINE

Hepatitis B vaccine has been available in the United States since 1982 and in Australia since 1983. It is prepared from the plasma of carriers of hepatitis B surface antigen (HBsAg). The manufacturing process involves procedures designed to eliminate infectious hepatitis B virions by biophysical and biochemical means, the latter of which includes pepsin treatment at pH2, 8M urea and formalin.

A theoretical side effect of vaccination against hepatitis B is infection with a different virus present in blood of a plasma donor. However a 1983 review concluded that vaccine preparation procedures were adequate to inactivate representative viruses of what was then all the known types[1].

Since 1983, the Human Immunodeficiency Virus (HIV), the aetiological agent of AIDS has been identified, and despite the safety record of hepatitis B vaccine in respect of other viral infections, many people are now reluctant to be vaccinated against hepatitis B. A recent report, however, provides evidence that there is no demonstrable risk of contracting AIDS through vaccination with the currently available hepatitis B vaccine[2].

HIV in lymphocyte cultures were treated independently with 2 of the 3 viral inactivation procedures used in vaccine manufacture, namely -
1) 8M urea at 37°C for 4 hours; and
2) 0.01% formaldehyde at 37°C for 72 hours.

The treatment was performed on cultures of 3 different isolates of HIV, which had initial titres of $10^{4.2}$, $10^{4.9}$ and $10^{5.1}$ median tissue culture infectious doses ($TCID_{50}$) respectively. Post treatment viral titres were less the 10^{1} TCID in each case (ie the limit of the assay) which indicates that each procedure independently completely inactivated each HIV isolate.

Some vaccine batches have been identified as being derived from plasma pools which, retrospectively were found to include plasma from at least one donor in whom AIDS subsequently developed. Sera were taken from 50 recipients of such vaccine batches, one month after the last dose and tested for AIDS antibody using an enzyme - linked immuno-sorbent assay. No antibody was detected.

Clinical experience also indicates the safety of vaccine in humans. It has been estimated[2] that in the US, over 1 million persons have received the hepatitis B vaccine licenced in that country. To July 1986, no case of AIDS had been reported in a recipient who did not have other risk factors.

Taken together, the data clearly support the view that the risk of infection with AIDS from hepatitis B vaccination is minimal.

REFERENCES

1. CDI (1983) 83/1:5
2. JAMA (1986) 256 (7) 869-72

HEPATITIS A - AN EPIDEMIC IN OHIO

Between June 1, 1983 and August 30, 1984, an epidemic involving 313 cases of hepatitis A occurred in Muskingum County (population, 85 000), Ohio[1]. Of these, 197 cases occurred in the city of Zanesville (population, 28 500), with 34.7% of cases concentrated in two neighbourhoods in the eastern part of the city. Of the 313 cases of hepatitis A, 273 (87.2%) experienced jaundice, 47 (15%) were hospitalized and there were no fatalities. Eleven of the cases (3.5%) were non-white; eight were black and three were American Indians.

The mean age of cases was 18.6 years (standard deviation, 12.5 years) with the highest attack rate occurring in children aged 5-9 years. The attack rate declined with age, although there was a significant secondary rise in females aged 20-24 years. Adults over age 30 years had the lowest rates, possibly as a result of immunity gained during the epidemics of 1952, 1960 and 1969[1]. The overall attack rate was 7.4 per 1000 and 6.4 per 1000 for males and females, respectively. However, the difference in attack rate for males and females was not statistically significant for any age group.

The mean number of members for case households in Zanesville was 4.93, with 82 of the case households containing four or more persons. Members of case households tended to be less educated, more likely to have changed address in the previous 5 years and more likely to live in rented housing than the mean for households in the city.

Of the 208 cases in Muskingum County available for review, 78 (37.5%) reported definite exposure to other cases 14-42 days prior to the onset of symptoms, 22 (10.6%) had possible exposure and 108 (51.9%) had no known exposure to other cases. For the reported exposures, contact with infected household members or visitors was the most common type of encounter (42.8%). Casual exposures, such as baby-sitting group contacts, friends, schoolmates and neighbours were reported less frequently. There was no temporal or geographic clustering of the cases with no reported exposure to other cases.

A case-control study failed to show differences in several behavioural factors between case and control households, but did confirm that a lower socio-economic status was a risk factor for the disease.

Early during the outbreak, the county and state health departments formulated a policy recommending immunoglobulin prophylaxis within 2 weeks of exposure for certain groups of case contacts. These included: household members; visitors who consumed food or beverages at the patient's house; persons who had direct contact with the patient or with members of the patient's household who were less than 10 years old; people who were exposed in day-care, nursery, or baby-sitting groups which enrolled children who were less than 6 years old; and boyfriends and girlfriends. The administration of immunoglobulin to other than household members constituted a broader use of immunoglobulin than recommended under the guidelines of the Immunisation Practices Advisory Committee of the Centers for Disease Control[2].

In households with more than one member, 540 persons were candidates for immunoglobulin prophylaxis according to the criteria listed above. Of these, 446 (82.6%) received immunoglobulin; 369 (68.3%) received it within 2 weeks of the onset of symptoms in the index case, 45 (8.3%) received it later than 2 weeks and 32 (5.9%) received it at an unknown time. A total of 94 persons (17.4%) did not receive immunoglobulin prophylaxis. Of these, 21 were not immunised because of a history of hepatitis and 73 either refused the injection or failed to appear at the clinic.

Two (0.5%) of the persons who received immunoglobulin within the 2 week period became secondary cases. Of those who received it after the 2 week period, two (4.4%) later developed the illness. Eleven (11.7%) of the 94 persons who did not receive immunoglobulin subsequently became ill. Although the broad use of immunoglobulin was effective in preventing clinical disease among family contacts, it did not halt the outbreak.

CDI Editorial Comment:

Hepatitis A virus is endemic world-wide. In developing countries, most individuals become infected (usually asymptomatically) early in life, leading to a high degree of herd immunity among adults. In most Western countries, infection may be acquired at any age, and around 40-70 percent of adults are immune. In some Scandinavian countries, infection is becoming more uncommon and a majority of adults are susceptible. Vaccines to confer active immunity to hepatitis A are under development but at the present time none have been approved for clinical use.

Normal immunoglobulin (human)

Immunoglobulin may not always prevent infection, but it can be expected to modify the resulting disease so that it is mild, or undetectable clinically.

Routine passive protection is recommended for the following categories of individuals:

- Household contacts of an index case, who have not already had hepatitis A.
- Common source exposures. When a vehicle such as food or water is identified as a common source of infection for multiple hepatitis cases, administration of immunoglobulin should be considered for all those exposed to the source.

These classes should both be given a dose of gamma globulin equivalent to 0.06 ml/kg body weight for long term protection or 0.03 ml/kg body weight for short term protection.

- Institutional contacts, who should be given a higher dose.
- Staff in institutions where hepatitis is endemic. A large dose (0.06 ml/kg body weight) should be offered at the time of employment, and this should be repeated at six-monthly intervals if the risk persists.

Routine prophylaxis is not recommended for school, office, factory or hospital contacts.

Recommendations for use

Provided it is given within 7-10 days after exposure, normal immunoglobulin (human) is of proven benefit in preventing or modifying the severity of hepatitis A infection. Thus, by the time an index case of hepatitis A infection is clinically apparent, other individuals who were exposed simultaneously from a common source will not be protected by administration of normal immunoglobulin (human). It is, however, of value in protecting secondary contacts of an index case. In practice, the usefulness of immunoglobulin depends on identification of persons at risk within a few days of exposure.

Post-exposure prophylaxis

Normal immunoglobulin (human) is recommended for household contacts of a case of hepatitis A. It should be given as soon as possible after onset of jaundice in the index case.

In institutions caring for young children, particularly those children likely to soil nappies or clothing, the risk of cross-infection is high. In such centres, immunoglobulin is advised for both staff and children in close contact with the index case.

The routine administration of immunoglobulin to contacts of a case of hepatitis A in the school or workplace is generally not necessary, but may be warranted in certain circumstances. These may include regular and close contact with the patient, as distinct from occasional contact only, during the two weeks prior to the commencement of illness. A decision on the matter should be made by an appropriate medical practitioner after the relevant facts of the particular situation have been ascertained.

Pre-exposure prophylaxis

Normal immunoglobulin (human) is recommended for travellers to areas where hepatitis is highly endemic (Asia, India, Africa) if the individual involved is likely to be eating and drinking from sources where hygiene is not strictly controlled. Short term visits under first-class tourist facilities, do not normally warrant use of immunoglobulin.

The following dosages are recommended:

Persons' weight (kg)	Dosage	
	Short-term prophylaxis (ml)	Long term (5 months) prophylaxis (ml)
under 25	0.5	1.0
25 to 50	1.0	2.5
over 50	2.0	5.0

Administration

Normal immunoglobulin (human) should be given by deep intramuscular injection, using a large (20) gauge needle. Because of possible untoward reactions, this product should not be administered by the intravenous route. A suitable intravenous preparation - normal immunoglobulin (human, intravenous) - is now available for use in patients requiring large doses of immunoglobulin.

Side effects and adverse reactions

Local

Local tenderness and stiffness of the muscles occur occasionally at the site of injection, and may persist for several hours after injection.

Systemic

Urticaria and angioedema may occur. An occasional patient may react more strongly with erythema or low grade fever.

Anaphylactic reactions

Although rare, anaphylactic reactions have been reported following the injection of human immunoglobulin preparations. Anaphylaxis is more likely to occur if normal immunoglobulin is given intravenously. In highly allergic individuals, repeated injections may lead to anaphylactic shock.

Precautions and contraindications

Passively acquired antibody can interfere with the response to live, attenuated virus vaccines. Therefore, administration of such vaccines, e.g. poliomyelitis or measles, should be deferred until approximately three months after passive immunisation. By the same token, immunoglobulins should not be administered for at least two weeks after a vaccine has been given.

Pregnancy is not a contraindication to administration of immunoglobulin.

Management of outbreaks

Control of hepatitis A outbreaks is achieved by enforcement of hygienic measures designed to limit faecal-oral spread, e.g., appropriate care with food preparation, water sources, faecal disposal, handwashing; and by isolation of cases using enteric precautions. In addition, individuals at risk can be protected from symptomatic infection by the administration of normal immunoglobulin (human) as described above.

REFERENCES

1. Am J Epidemiol (1986); 123: 1057-65
2. MMWR (1985); 34: 313-24, 329-35

TYPHOID FEVER (U.K. ex Spain)
(based on CDR 86/34, 22 August 1986)

Typhoid fever was confirmed in 3 British holiday-makers who visited Salou near Tarragona in Spain during July 1986. Salmonella typhi isolated from these patients were of different phage types. Problems with the local main water supply have been reported.

CDI Editorial Comment:
Early information on places visited by Australian patients with enteric fever should be reported to:
The National Salmonella Surveillance Scheme
Microbiological Diagnostic Unit
University of Melbourne VIC 3000

GASTROINTESTINAL INFECTIONS FOLLOWING A TRIP TO RUSSIA
(based on CDR 86/33 15 August 1986)

Thirty-one persons including teachers, relatives and teenage pupils from a school in North Yorkshire (UK) made a one week trip to Russia in mid-February 1986. The trip included 3 days in Moscow followed by 3 days in Leningrad. On return from the trip 2 people consulted their doctors complaining of diarrhoea and it became apparent several weeks later that many others had been affected. Each tourist was sent a questionnaire requesting details of any illness or exposure and asked to submit a sample of faeces to the laboratory.

Of the 25 people (81%) who completed the questionnaire, 17 reported gastro-intestinal symptoms featuring diarrhoea, abdominal pain and nausea, lasting 3-6 days in most cases but extending to several weeks in some. The dates of onset of illness were established for 16 patients:

. one became ill on the tour
. two became ill on the day of return from Russia
. eleven became ill within 2 to 7 days
. two became ill on days 10 and 17 respectively.

The examination of faecal samples from 19 subjects identified pathogens in 11 of 13 persons with gastrointestinal symptoms and 2 of 6 persons who had remained asymptomatic. Salmonella agona was isolated from the 2 index cases who had become ill on the day of return; one of the 2 cases was also infected with Giardia lamblia and cryptosporidium. Faecal samples from other patients, collected 25-31 days after return, yielded G. lamblia in 11 specimens including 2 which also yielded cryptosporidium. Apart from the early illness in the salmonella-infected patients there appeared to be no relationship between dates of onset of illness and type of organism found.

CDR Comment:

A recent prospective study from Finland demonstrated that cryptosporidium was commonly acquired by students visiting Leningrad[1]. G. Lamblia infection was also common and has been known for some years as a constant hazard to visitors[2]. The outbreak detailed above reported cryptosporidial infection for the first time amongst British tourists to Russia. The Finnish study demonstrated a shorter incubation period for cryptosporidium than for G. Lamblia; hence it is likely that a number of patients with early onset illness had salmonella or cryptosporidial infections which resolved naturally before the faeces could be examined. It was therefore not possible to determine which of these organisms was primarily responsible for the outbreak.

Many of the tourists commented on the high standards in hotel accommodation and cuisine; and it was not possible to determine the sources of infections, including the details of water consumption.

REFERENCE

1. Lancet (1985) ii 487-89.

2. CDI (1986) 17: 2

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD 27-10-86 to 9-11-86 BULLETIN NUMBER 86/23
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ PCH (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED	1	1	5			4	1	2	14
0101 ADENOVIRUS TYPE 1	1			1		4		1	7
0102 ADENOVIRUS TYPE 2				2		3		2	7
0103 ADENOVIRUS TYPE 3				6		3			9
0105 ADENOVIRUS TYPE 5				1				1	2
0106 ADENOVIRUS TYPE 6	1					1			2
0108 ADENOVIRUS TYPE 8				1					1
0113 ADENOVIRUS TYPE 13	1								1
0119 ADENOVIRUS TYPE 19	1								1
0199 ADENOVIRUS TYPING PENDING			1		2				3
0201 INFLUENZA A VIRUS	1								1
0203 INFLUENZA B VIRUS	1								1
0206 INFLUENZA A VIRUS SUBTYPE H1N1				1					1
0303 PARAINFLUENZA VIRUS TYPE 3	2			1	9	13			25
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)			1	6	7	12	1	28	55
0500 RHINOVIRUS (ALL TYPES)					25	12		1	38
0600 MYCOPLASMA PNEUMONIAE	5					10		11	26
0700 ORNITHOSIS-PSITTACOSIS	4					1		1	6
0816 COXSACKIEVIRUS A16						1			1
0906 COXSACKIEVIRUS B6						1			1
1005 ECHOVIRUS TYPE 5				2					2
1011 ECHOVIRUS TYPE 11	2			3				6	11
1012 ECHOVIRUS TYPE 12						1			1
1022 ECHOVIRUS TYPE 22	1	1							2
1027 ECHOVIRUS TYPE 27						1			1
1100 POLIOVIRUS NOT TYPED			2						2
1101 POLIOVIRUS TYPE 1						1			1
1102 POLIOVIRUS TYPE 2		1				2			3
1103 POLIOVIRUS TYPE 3						1			1
1200 MUMPS VIRUS								2	2
1300 HERPES VIRUS GROUP-NOT TYPED	16			1				1	18
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2							2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	9							7	16
1303 VARICELLA-ZOSTER VIRUS	6		1		1	1		6	15
1306 HERPES SIMPLEX TYPE 1	26		13	37		33		18	127
1307 HERPES SIMPLEX TYPE 2	86		42	54		17		64	263
1399 HERPES VIRUS TYPING PENDING				2	4				6
1401 COXIELLA BURNETI	2							1	3
1502 PICORNA VIRUS-NOT TYPED			4					8	12
1521 MEASLES VIRUS	2	1			1				4
1522 RUBELLA VIRUS	33			8		5		12	58
1532 HEPATITIS B ANTIGEN	43		3	21		15		7	89
1535 HEPATITIS A ANTIBODY	3		2	3		12		7	27
1541 CHLAMYDIA A - C TRACHOMATIS	26					43	6	57	132
1556 CMV - CYTOMEGALOVIRUS	1		2	18	4	3		4	32
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	12	1		5	1	11	11	4	45
1566 NORWALK AGENT	2								2
1571 ENTEROVIRUS TYPE 71 (BRCR)	2	1				1			4
1599 ENTEROVIRUS TYPING PENDING			1		8				9
9993 ASTROVIRUS	1								1
9994 SMALL VIRUS (LIKE) PARTICLE		1							1
9998 ARBO. GROUP B. ...						1			1
Total	291	9	77	173	62	213	19	252	1,096

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 27-10-86 to 9-11-86 BULLETIN NUMBER 86/23

Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; 03 -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
0101 ADENOVIRUS TYPE 1.....		6					1				
0102 ADENOVIRUS TYPE 2.....		5					2				
0103 ADENOVIRUS TYPE 3.....		5					2				
0105 ADENOVIRUS TYPE 5.....							2				
0106 ADENOVIRUS TYPE 6.....		1					1				
0201 INFLUENZA A VIRUS.....		1									
0206 INFLUENZA A VIRUS SUBTYPE H1N1							1				
0303 PARAINFLUENZA VIRUS TYPE 3....		25					1				
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		53					1				
0500 RHINOVIRUS (ALL TYPES).....		32					2				
0600 MYCOPLASMA PNEUMONIAE.....	3	20						1			
0700 ORNITHOSIS-PSITTACOSIS.....	1	3		1						1	
0816 COXSACKIEVIRUS A16.....							1				
0906 COXSACKIEVIRUS B6.....		1									
1005 ECHOVIRUS TYPE 5.....				2							
1011 ECHOVIRUS TYPE 11.....	2	1		3			1				1
1012 ECHOVIRUS TYPE 12.....		1									
1022 ECHOVIRUS TYPE 22.....		2									
1027 ECHOVIRUS TYPE 27.....							1				
1101 POLIOVIRUS TYPE 1.....		1									
1102 POLIOVIRUS TYPE 2.....	1	1									
1103 POLIOVIRUS TYPE 3.....							1				
1200 MUMPS VIRUS.....				1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED											2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	1	1						1			
1303 VARICELLA-ZOSTER VIRUS.....	6	1				1					7
1306 HERPES SIMPLEX TYPE 1.....	5	8									66
1307 HERPES SIMPLEX TYPE 2.....	6	1									61
1401 COXIELLA BURNETI.....	1										
1502 PICORNA VIRUS-NOT TYPED.....				1			3		1	1	
1521 MEASLES VIRUS.....			1								2
1522 RUBELLA VIRUS.....	15	1	2			1					17
1532 HEPATITIS B ANTIGEN.....	23							49			
1535 HEPATITIS A ANTIBODY.....	4						1	17			
1541 CHLAMYDIA A - C.TRACHOMATIS...	23									1	
1556 CMV - CYTOMEGALOVIRUS.....	1	3				1		2		5	
1564 ROTAVIRUS.....		1					44				
1566 NORWALK AGENT.....							2				
1571 ENTEROVIRUS TYPE 71 (BRCR)....	1			2		1					
9993 ASTROVIRUS.....							1				
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
9998 ARBO. GROUP B.			1								
Total.....	93	174	4	10		4	69	70	1	8	156

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 27-10-86 to 9-11-86 BULLETIN NUMBER 86/23

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.....	2							2		
0108 ADENOVIRUS 8.....	1									
0113 ADENOVIRUS TYPE 13.....	1									
0119 ADENOVIRUS TYPE 19.....		1								
0203 INFLUENZA B VIRUS.....							1			
0206 INFLUENZA A VIRUS SUBTYPE H1N1								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1	2	
0500 RHINOVIRUS (ALL TYPES).....								2	1	4
0600 MYCOPLASMA PNEUMONIAE.....							2	1	2	
0700 ORNITHOSIS-PSITTACOSIS.....							1			
1011 ECHOVIRUS TYPE 11.....							1	3		
1102 POLIOVIRUS TYPE 2.....										1
1200 MUMPS VIRUS.....								1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			7	4			2	1	1	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	2	46							1	
1307 HERPES SIMPLEX TYPE 2.....		192							3	
1401 COXIELLA BURNETI.....			1				1			
1502 PICORNA VIRUS-NOT TYPED.....								1	1	
1521 MEASLES VIRUS.....								1		
1522 RUBELLA VIRUS.....		1	6	3	3		1	4	9	
1532 HEPATITIS B ANTIGEN.....									17	
1535 HEPATITIS A ANTIBODY.....									5	
1541 CHLAMYDIA A - C.TRACHOMATIS...	2	106								
1556 CMV - CYTOMEGALOVIRUS.....		1	1					2	17	
1563 CORONAVIRUS.....									1	
Total.....	8	347	15	7	3		9	20	61	5

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 6 - 17 May 1986 to 13 June 1986 Bulletin.....

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis		1			1				2	24
Ankylostomiasis			2	1	1		NN		4	13
Anthrax									-	-
Arbovirus infection	12		141		1				154	965
Brucellosis			1						1	11
Campylobacter infections	98	3	NN	83	3	NN	4	NN	191	1 114
Chancroid	1			NN					1	5
Cholera									-	-
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria							6		6	20
Donovanosis			2	NN	9		4		15	63
Giardiasis	32		NN	59	7	NN	NN	NN	98	585
Genital herpes	70			18	NN	NN	1	NN	89	614
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	-
Gonorrhoea	98		16	43	135	2	36	4	334	2 312
Hepatitis A (infectious)	29	9	11	37	56	2	7		151	* 866
Hepatitis B (serum)	43	15	46	15	31		4	3	157	* 880
Hepatitis – unspecified	6		6		NN	NN			12	76
Hydatid disease									-	6
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	1		NN	2		NN		NN	3	37
Leprosy			1						1	8
Leptospirosis	2		17	1					20	104
Lymphogranuloma venereum				NN	NN	NN		NN	-	2
Marburg disease			NN			NN		NN	-	-
Malaria	13	1	15	1	2	3		4	39	327
									-	-
Meningococcal infections			1			NN			1	15

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Non-specific urethritis	294		NN	31	NN	NN	NN	NN	325	2 256
Ornithosis *		1		4					5	21
Pertussis (whooping cough)	5	1	NN	7	7	NN		NN	20	381
Plague									-	-
Poliomyelitis									-	-
Q. fever	6		26	5					37	137
Rabies									-	-
Salmonella infections	52	20	71	20	11	1	20	1	196	1 429
Shigella infections	10		8	5	14		33		70	445
Smallpox									-	-
Syphilis	36		17	26	36		112	1	228	1 113
Tetanus					1				1	4
Trachoma		NN				NN	NN		-	35
Tuberculosis (all forms)	27	26	17	6	6	4	3	2	91	* 455
Typhoid fever	1								1	16
Typhus (all forms)			6						6	* 13
Vibrio parahaemolyticus infections	1		NN			NN		NN	1	5
Yellow fever									-	-
Yersinia infections	3		NN	1		NN		NN	4	46

NN - Not Notifiable

(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.)

* Adjustment to the Cumulative Total since last report:

Hepatitis A	+5	South Australia
Hepatitis B	+1	South Australia
Tuberculosis	+3	South Australia
Typhus (all forms)	-2	New South Wales