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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME. A total of 1,672 reports were processed for this period.

Thirty nine cases of Q fever were reported (3 from New South Wales and 36 from Queensland). Occupational data were only available for 28 Queensland cases. The demographic details were as follow:-

OCCUPATION	(No. of cases)
VETERINARIAN	(2)
MEATWORKER	(17)
SHEARER	(6)
FARMER	(1)
MEAT INSPECTOR	(1)
WOOL SORTER	(1)

All cases were in males aged 15-47 years except a 54 year old female meatworker.

None of these patients were involved in the Q fever vaccine field trial conducted in South Australia.

Cytomegalovirus was isolated from the blood of a 30 year old leukemia patient who underwent bone marrow transplantation. The patient died of severe pneumonia.

One case of Australian Encephalitis has been reported in a 6 year old male who acquired the infection in the Fitzroy Crossing area of Western Australia.

One hundred and eighty one cases of Ross River virus were reported, 1 from New South Wales, 8 from Western Australia and 172 from Queensland.

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Q FEVER AMONG SLAUGHTERHOUSE WORKERS - CALIFORNIA
(Based on MMWR Vol. 35/No 14, 11 April 1986)

During May 1985, five workers at a local meatpacking plant that processes sheep, presenting with illness characterised by fever, malaise, myalgias, severe headache and abdominal pain, but not jaundice have been reported to the Solano County (California) Health Department as hepatitis cases. The patients exhibited symptoms which lasted at least 1 week, then gradually resolved. Hepatitis was suspected because all cases had moderately elevated SGOT levels. However, none had high IgM (antibody to surface antigen) titres consistent with either hepatitis A or B infection. Since all five patients were exposed to animal carcasses in the course of their work, the differential diagnosis included Q fever, brucellosis and leptospirosis. Sera from four of the above patients were positive for IgM antibody to Q fever by the immunofluorescent antibody test (IFA), indicating recent infection.

A subsequent serological survey was conducted on 42 of the approximately 100 employees, who agreed to participate, including the five employees described above. Nineteen (45%) of the surveyed employees were positive by IFA test (but negative by CF test) for IgG antibody, 11 (26%) were negative both by CF and IFA and 12 (29%) had complement-fixation (CF) titres to Q fever rickettsiae including 8 who had recently experienced a clinical illness compatible with Q fever. The 31 persons with serologic evidence of infection worked in a variety of jobs in areas throughout the plant, but no further investigation was performed to determine areas of highest risk.

However, an investigation conducted by the California Occupational Health and Safety Administration resulted in the implementation of a surveillance program that included screening for Q fever by serology and for valvular heart disease among new employees, but no feasible environmental control measures were identified. In addition, a Q fever awareness campaign has been instituted to:

- educate employees about the illness through printed material and a question and answer session, and
- inform by mail all physicians servicing the area in the vicinity of the meatpacking plant.

CDI EDITORIAL COMMENT:

Q fever notifications are routinely monitored by the CDI virus reporting scheme, as part of a morbidity survey assisting the ongoing Australian effort to develop a Q fever vaccine.

At its 97th session in June 1984, the National Health and Medical Research Council (NH & MRC) commended and endorsed a trial on vaccine prophylaxis of abattoir associated Q fever, conducted by personnel from the Institute of Medical and Veterinary Science, South Australia, the Commonwealth Serum Laboratories and South Australian medical officers. The group had developed the Q fever vaccine in the trial as there was no other commercially and readily available Q fever vaccine in the world.

At its 98th session in October 1984, the NH & MRC recommended that vaccine be indicated in groups at risk of contracting Q fever through occupational exposure as listed below:

Q FEVER VACCINE - LIST OF GROUPS AT RISK

1. The essential requirement that those to be vaccinated must first be serotested and skin tested to detect an existing immunity or hypersensitivity to the Q fever rickettsia imposes certain limits on the occupationally-exposed groups that can be vaccinated. The main targets have to be groups of workers with access to a medical room or medical station in which trained staff can take blood, do intradermal skin tests, read the results and maintain adequate records of the vaccination procedure.

2. Against this background, the following groups of workers, particularly new recruits to the industry, should be considered for vaccination:
   (a) Abattoir workers, including workers in subsidiary trades on the abattoir campus. Arrangements should be made to vaccinate frequent visitors to the plant, eg. equipment salesmen, meat wholesalers, catering school students.

   (b) Workers in large milk handling plants, shearing teams, wool sorting plants and tanneries, offal and glue factories, and any other organisation handling wool, hides, bones or entrails from cattle, sheep and goats.

   (c) Staff in medical and veterinary schools and research institutes experimenting with pregnant cattle, sheep or goats.

   (d) Veterinary students and other students in training for work in agriculture or agricultural laboratories that will bring them into contact with parturient cattle, sheep or goats or products of conception.

   (e) Laboratory workers preparing Q fever diagnostic antigen, vaccine or isolating the organism for diagnostic purposes.

The expanded Q fever vaccine trial, as required by the Australian Drug Evaluation Committee (ADEC) is nearing completion.

STREPTOCOCCAL PHARYNGITIS DIAGNOSIS - THROAT CULTURE OR LATEX AGGLUTINATION?

In a recently published American study, the sensitivity and specificity of an *in vitro* latex agglutination test for group A streptococci were compared with throat swab cultures[1].

Over a 4 month period, 100 consecutive throat swab specimens were tested for group A streptococci using both standard microbiological culture techniques and a latex agglutination test kit manufactured by a US Company. All specimens were collected from both the tonsillar and posterior pharyngeal area. Plating and agglutination testing were done on the same swab immediately after collection. Specimens were cultured on 5% blood agar plates at 37° in a candle jar. The agglutination test was performed according to the recommendations of the test kit manufacturer[2]. Culture

data were available approximately 24 hours after taking the specimen, and were read by a person who was blinded to the results of the agglutination test. The time taken for the latex agglutination test was not stated, but the experimental detail given indicates that results would have been available within about 20 min. Positive controls were performed daily.

Of 100 specimens, 22 were culture positive for group A streptococci (see table 1) of these, 20 were also positive in the latex test. Seventy eight of the 100 cultures were negative, and of these, 75 were latex test negative. The 3 culture negative/latex positive patients were later interviewed, and 1 admitted to having used antibiotics prior to providing the specimen. This patient, therefore, may not represent a false positive result. No explanation was provided for the 2 culture positive/latex negative patients.

Based on the limited data, the sensitivity of the latex test was estimated as 91% (20/22), and the specificity as 96% (75/78). The test sensitivity and specificity in this report are comparable with those reported in earlier publications[3-6] which are summarised in table 2. The variation in incidence of culture positives in these studies is possibly due to differences in patient age mix. Children have higher rates of streptococcal pharyngitis than adults, and one study[6] included only children and adolescents.

Table 1 Latex agglutination test performance compared with throat swab cultures.

Agglutination test	Throat Culture		
	Positive	Negative	Total
Positive	20	3	23
Negative	2	75	77
Total	22	78	100

Table 2 Agglutination test performance reported in the literature.

Reference	N	Sensitivity %	Specificity %	Incidence %
3	817	92.4	92.8	11.3
4	435	90	99.2	16
5	557	95.1	100	15
6	263	83	99	41

CDI EDITORIAL COMMENT

Based on the limited data examined[1], the latex agglutination test appears to be comparable with microbiological culture methods in the detection of group A streptococci. Whether the latex test finds a place in diagnostic laboratories will be determined by several factors.

First, the inherent reliability of the test will be of paramount importance. Large batch to batch variation in the incidence of incorrect results would significantly reduce the usefulness of the product.

Second, the degree of technical skill required to interpret test data might affect both the accuracy of the results and the cost in technician time.

Third, the cost of the latex test, calculated as US $2.50 per test is higher than throat cultures at less than US $1 per test[1].

Fourth, the speed with which results from the 2 tests can be obtained may be a decisive factor. The results of throat cultures are normally available between 24 and 48 hours after taking the specimen, while the latex test appears to take less than 20 min. The latter might be expected to have beneficial clinical and economic consequences for the patient. The more rapid diagnosis would allow appropriate therapy to begin almost immediately after consulting the physician, perhaps obviate a repeat visit and reduce time lost from school or work.

Clearly, the extent to which the latex agglutination test finds a place in diagnostic medicine will depend on a balance of all 4 factors.

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EPIDEMIC OF ACUTE GASTROENTERITIS AT A TERTIARY CARE HOSPITAL - ONTARIO

(based on CDWR Vol 12-6, 8 February, 1986)

On 14 November 1985, many health care personnel at a Toronto tertiary-care hospital had developed acute gastroenteritis. The incident was notified to the hospital infection control nurse who, on the following day, instituted a preliminary surveillance of all hospital staff and patients. The monitoring revealed several hundred additional cases and indicated that the disease had affected most areas of the hospital. Subsequently a decision was made to close the hospital to all admissions and emergency room visits as of 18.00 hours on 15 November in order to contain the outbreak.

Illness was characterised by fatigue, nausea, diarrhoea, abdominal cramps, headache, myalgia and vomiting. The case definition was satisfied by symptoms of vomiting and/or watery diarrhoea with 2 or more stools per day. A total of 673 hospital employees fitted the case definition for an attack rate of 25%. The highest attack rates were among staff in the emergency room (70%), respiratory therapy (69%) and the department of medicine (64%). Among hospitalised patients, 109 cases satisfied the case definition for an attack rate of 20%, with the highest attack rates being recorded on medical floors.

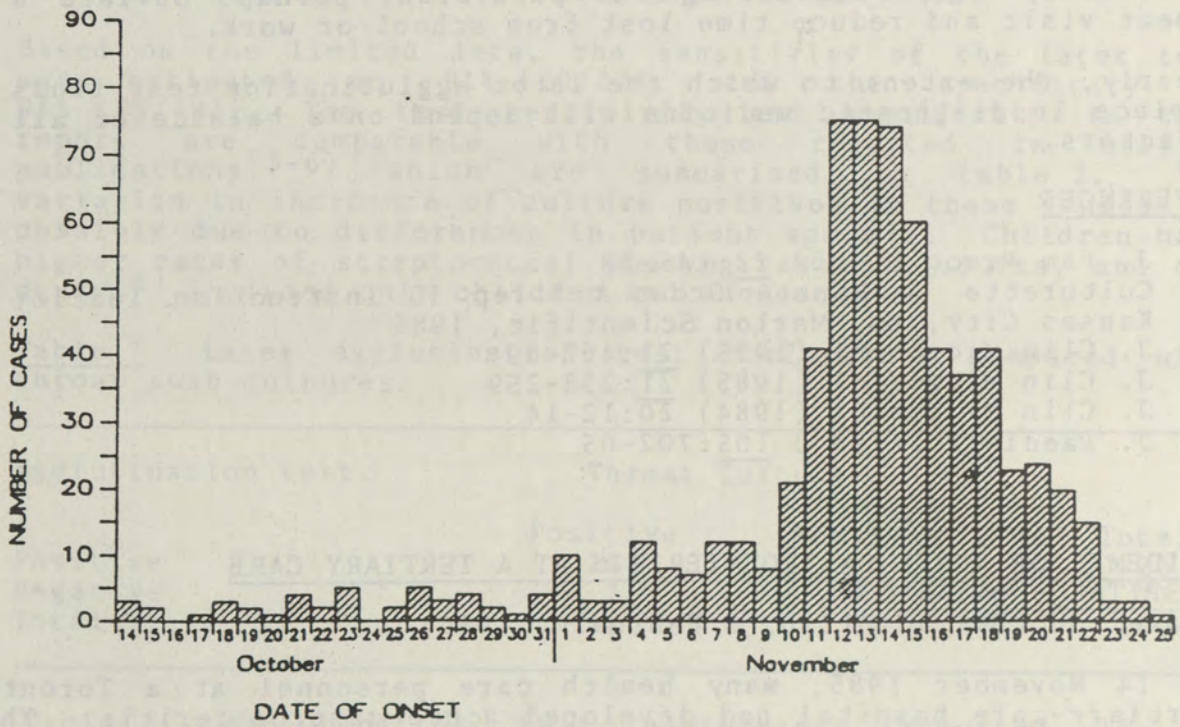
Illness was generally benign with the median duration of symptoms lasting between 24 and 48 hours. Stool specimens from 30 cases were negative for Salmonella, Shigella, Yersinia,

Campylobacter and toxogenic Escherichia coli. Electron microscopic examination identified 27 nm virus-like particles in 4 of 17 stool specimens.

Internal hospital investigation suggested that the outbreak period occurred between 1 and 22 November 1985 with the first in-patient case reported on 11 November. The epidemic curve (Figure 1) was consistent with person to person transmission of the virus. Subsequent environmental and staff case-control studies did not implicate food, water or ice as an infection source.

FIGURE 1

Cases of Gastroenteritis among Mount Sinai Hospital Staff Toronto, Ontario 14 October - 25 November 1985.



A subsequent community survey identified 102 people who satisfied the case definition, out of the 200 individuals who reported development of vomiting and/or diarrhoea symptoms following their visit to the hospital during the outbreak. Ninety four of these 200 callers had visited the Emergency Room. Of the 102 identified cases 35 (34%) had visited the Emergency Room prior to their illness. The infection was apparently not associated with touching staff, using the washroom, consuming food, drinking water or smoking.

The attack rate in a different area of the hospital during the same time period was also determined at 7% with 3 cases identified out of 41 randomly selected patients who had been seen in the family practice unit. No cases were identified among 18 randomly selected patients who had visited the Emergency Room on 8 November. The Emergency Room has been postulated as an apparent common source of infection for several days. The virus was probably spread by fomites and possibly via aerosols in the Emergency Room and then transmitted by person-to-person contact throughout the rest of the hospital leading to an outbreak of over 700 cases of acute gastroenteritis involving both staff and patients at the hospital.

I EDITORIAL COMMENT:

idemic viral gastroenteritis is characterised by:-
the absence of bacterial pathogens;
gastroenteritis with an incubation period of 16-48 hours, a rapid onset and recovery, a clinical course lasting 24-48 hours, and relatively mild systemic signs;
an epidemiologic pattern of a highly communicable disease that spreads rapidly with no particular predilection in terms of age or geography.

lthough symptoms include diarrhoea, nausea, vomiting, low-grade ver, abdominal cramps, headache and malaise, hospitalisation is rely required, treatment is symptomatic and no sequelae have en reported.

e 'Norwalk agent', a virus particle of 27 nm in diameter as monstrated by immune electron microscopy in stools from adults h acute gastroenteritis, appears to have at least 3 serotypes t as yet has not been grown in tissues culture. However, man volunteer experiments have clearly shown that the pearance of the virus coincides with the clinical illness. tibody develops during the illness and is protective against infection with that agent. Experimental infection of impanzees results in infection and seroconversion of animals in e absence of clinical illness.

ile immune electron microscopy was required initially for the tection of the virus and antibody, a radioimmunoassay blocking st and an immune adherence method can now detect antibody to rwalk type virus. Whereas rotavirus antibody develops early in ildhood, Norwalk virus antibody is acquired later in life; by e fifth decade, 50% of adults have such antibody.

AUTION:

ecause of the infectious nature of the stools, care should be ken in their handling and disposal.

OTAVIRUS VACCINE RESEARCH

uman rotaviruses were first identified about 12 years ago. otavirus infections, cause most episodes of diarrhoea in the irst 2 years of life particularly in tropical countries. In ommunities with poor hygiene practices these viruses cause a elatively high mortality in children during their first months of life.

he role of humoral immunity in the protection of infants from nfection is uncertain, although intestinal IgA appears to be mportant. It has also been suggested that transplacentally cquired IgG provides considerable protection to neonates, ince in this group, exposure to the virus usually results in a ild or subclinical infection[1-4]. Nevertheless, attempts o relate neonatal rotavirus resistance to maternal IgG levels ave been unsuccessful[5].

here are 4, and possibly 5 serotypes of human rotavirus. It s well established that rotaviruses from other species can nfect piglets causing either subclinical infection seroconversion) or diarrhoea, but it is not known whether uman rotaviral infection is a zoonotic disease.

Studies with piglets have shown that prior infection with one serotype did not adequately protect the animals against infection by other serotypes, although they were resistant to challenge by the homologous serotype[6]. This raises the possibility that it may be necessary to include antigens to all pathogenic rotavirus serotypes in a human vaccine. A preliminary study conducted in Finland suggests that this may not be so[7]. In a randomised, double blind, placebo-controlled clinical trial, 178 infants (aged 8 to 11 months) received a single oral dose of either a live attenuated bovine rotavirus vaccine (RIT 4237 subgroup 1) or placebo, during a seasonal epidemic of rotavirus, subgroup 2 infection. During the 5 months following vaccination, the subjects were followed up serologically and clinically. Two of 86 (2.3%) vaccine recipients and 18 of 92 (19.6%) placebo recipients had rotavirus diarrhoea lasting more than 24 hours ($p < 0.001$) in that time. The 2 vaccinated subjects who suffered disease symptoms were considered to be primary vaccination failures as there was no detectable antibody response after vaccination.

It has been reported[8] that similar clinical trials are, or will soon be under way in Peru and The Gambia. It will be important to discover whether vaccination is effective in tropical or sub-tropical climates, where the challenge dose of virus may be much larger.

Attempts are being made to develop vaccines using other strains of rotavirus. It appears that a vaccine derived from a human isolate has not yet been developed. The National Institutes of Health (NIH), Bethesda, Maryland have developed a rhesus derived vaccine which is reported to be on clinical trial in Venezuela [8], and work is continuing with the calf rotavirus vaccine (7-9).

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APPARENT TRANSMISSION OF HTLV-III IN THE HEALTH-CARE SETTING

(Based on MMWR (1986) 35: 76-79)

A report of the apparent transmission of HTLV-III from a child to its mother has recently been received by the Centers for Disease Control (CDC), Atlanta, Georgia, USA. Infection of the mother appears to have occurred while providing nursing care that involved extensive unprotected exposure to the child's blood and body secretions and excretions.

The child (M, 24 months as at February 1986) had been diagnosed as having a congenital intestinal abnormality at age 4 days. Over the next several months, numerous surgical procedures were performed, including colonic and ileal resections, repairs of ostomies, intravascular catheter replacements, and a liver biopsy. The child was hospitalised for 17 months and has required intravenous hyperalimentation and nasogastric feeding throughout his life. His illness was also characterised by frequent bouts of bacterial sepsis, many of which were apparently related to his gastrointestinal disease and indwelling intravascular catheter. Because of anaemia due to chronic illness, multiple surgical procedures, gastrointestinal bleeding, and frequent blood drawing, the child required multiple transfusions between birth (February 1984) and early June 1985.

Because of the child's history of both recurrent bacterial sepsis and multiple transfusions, blood samples were taken in May and August 1985 and tested for antibody to HTLV-III. Both samples were positive by enzyme immunoassay (EIA). The second sample was tested by Western blot assay and was positive. In June 1985 the T-helper to T-suppressor lymphocyte ratio (T_H/T_S) was 1.6, within the normal range. Serum obtained in December 1985 was strongly positive for antibody to HTLV-III by EIA (absorbance $>$ 2.0. negative cutoff = 0.083, absorbance ratio $>$ 24). Western blot assay at CDC was positive for both the p24 and gp41 bands. Cultures of the child's peripheral blood lymphocytes, saliva, and stools for HTLV-III were negative.

The child had been transfused with blood from 26 donors between birth and June 1985 (16 months). One of the donors was a 34-year-old female whose serum was strongly HTLV-III antibody positive as at January 1986, by both EIA and Western blot assay. The child had received her blood in May 1984. All other donors were seronegative.

The child's 32-year-old mother had been closely involved in the child's care during hospitalisation and at home, which required frequent contact with the child's blood and with other body fluids. Her activities included drawing blood through the child's indwelling catheter at least weekly, removing peripheral intravenous lines occasionally, emptying and changing ostomy bags daily for the 7 months these were in place, inserting rectal tubes daily to facilitate large-bowel clearing, changing nappies and surgical dressings, and changing nasogastric feeding tubes weekly. When interviewed, she did not recall any specific incidents of needlesticks or other parenteral exposures to the child's blood. However, the mother did not wear gloves, and on numerous occasions, her hands become contaminated with blood, faeces (which often contained blood), saliva, and nasal secretions. She did not recall having open cuts or an exudative dermatitis on her hands. However, she often did not wash her hands immediately after blood or secretion contact.

In March, June, and October 1985, the mother donated blood. None of the donated blood was given to her child. As part of routine blood-donor screening, the blood was tested for HTLV-III antibody. She was seronegative by EIA in March and June. In October, a serum sample was repeatedly positive by EIA and was confirmed by Western blot assay. Serum obtained

during an investigation in December 1985, and the October 1985 specimen, were both strongly positive by EIA and Western blot assay at CDC. The mother remains clinically well. However, her T_H/T_S ratio was 0.9 (normal > 1.0) when tested in December 1985. Culture of her peripheral blood lymphocytes for HTLV-III was negative.

Extensive investigations did not reveal any other risk factors for infection in the mother or child. The mother was employed in paramedical duties before the child's birth but denied needlestick injuries or exposure to AIDS patients. The child's father is negative for HTLV-III antibody and is clinically well with a normal T_H/T_S ratio of 2.4.

MMWR Editorial note

The child probably became infected with HTLV-III through a transfusion of blood donated in May 1984 by a person who was later found to be seropositive. It is postulated that the mother acquired HTLV-III from her son while providing nursing care that involved extensive contact with his blood and other body secretions and excretions. She did not take precautions, such as wearing gloves, and often failed to wash her hands immediately after exposure.

The mother did not appear to have been exposed to other risk factors for HTLV-III infections. That seroconversion occurred between June and October 1985 suggested that her exposure to the virus occurred after the child's birth. Limited data[1-3] suggest that seroconversion occurs approximately 1 to 6 months after exposure to HTLV-III. As at early February 1986, there are no published reports of seroconversion later than 6 months after exposure. Although virus could not be isolated from the mother or child, the EIAs were repeatedly reactive from multiple specimens in separate laboratories. The high absorbance ratios and presence of strong bands reacting to specific viral proteins on Western blot assay were most consistent with HTLV-III infection.

Transmission of HTLV-III infection from child to parent has not been previously reported. The contact between the reported mother and child is not typical of the usual contact that could be expected in a family setting. None of the family members of the over 17 000 AIDS patients reported to CDC have been reported to have AIDS, except a small number of sexual partners of patients, family members who themselves had other established risk factors for AIDS, or children born to infected women. In the latter group, both transplacental transmission[4,5] and infection of an infant during the perinatal period[6] have been reported.

Although transmission of HTLV-III in the health-care setting has been reported, such transmission appears to be extremely rare. In five separate studies, a total of 1 498 health-care workers were tested for antibody to HTLV-III. In these studies, 666 (44.5%) of the workers had direct parenteral (needlestick or cut) or mucous-membrane exposure to patients with AIDS or HTLV-III infection. Although 26 persons in these five studies were seropositive when first tested, all but three of these belonged to groups recognised to be at increased risk for AIDS.[7-10]

There appears to be only one other case in which HTLV-III transmission from a patient to a person providing care may have occurred through a nonparenteral route[11]. In this report, from the United Kingdom, a 44-year-old woman who was not a health-care worker, developed AIDS after she had provided home nursing care for a Ghanaian man who was diagnosed with AIDS at postmortem examination. The care involved prolonged and frequent skin contact with body secretions and excretions. The woman recalled having some small cuts on her hands and an exacerbation of chronic eczema. She denied any sexual contact with the patient.

The occurrences of the US and UK cases suggest that HTLV-III infection may, on rare occasions, be transmitted during unprotected contact with blood or other potentially infectious body secretions or excretions in the absence of known parenteral or sexual exposure to these fluids.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 12/5/86 - 25/5/86 BULLETIN NUMBER 86/11
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	1	1		4	1	3	1	15
0101 ADENOVIRUS TYPE 1	2			2				2	6
0102 ADENOVIRUS TYPE 2	6	1						1	8
0103 ADENOVIRUS TYPE 3	2			1					3
0104 ADENOVIRUS TYPE 4	2								2
0105 ADENOVIRUS TYPE 5	1								1
0106 ADENOVIRUS TYPE 6					1				1
0108 ADENOVIRUS TYPE 8	3		3	1					7
0109 ADENOVIRUS TYPE 9	1								1
0111 ADENOVIRUS TYPE 11	1								1
0113 ADENOVIRUS TYPE 13	1								1
0126 ADENOVIRUS TYPE 26				1					1
0127 ADENOVIRUS TYPE 27	1								1
0131 ADENOVIRUS TYPE 31	1								1
0199 ADENOVIRUS TYPING PENDING			3						3
0201 INFLUENZA A VIRUS	1		2						3
0203 INFLUENZA B VIRUS			3						3
0301 PARAINFLUENZA VIRUS TYPE 1	1				16		1		18
0302 PARAINFLUENZA VIRUS TYPE 2	1			3	15	5			24
0303 PARAINFLUENZA VIRUS TYPE 3					3	2			5
0399 PARAINFLUENZA VIRUS TYPING PENDING		3							3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	11	4	5	1	3	2			26
0500 RHINOVIRUS (ALL TYPES)	3				14	7			24
0600 MYCOPLASMA PNEUMONIAE	9		1			1	6	3	20
0700 ORNITHOSIS-PSITTACOSIS	1					3			4
1002 ECHOVIRUS TYPE 2								1	1
1003 ECHOVIRUS TYPE 3				1					1
1005 ECHOVIRUS TYPE 5	1								1
1007 ECHOVIRUS TYPE 7								1	1
1011 ECHOVIRUS TYPE 11	3	1							4
1013 ECHOVIRUS TYPE 13		1							1
1018 ECHOVIRUS TYPE 18	1								1
1020 ECHOVIRUS TYPE 20	3								3
1021 ECHOVIRUS TYPE 21	1			2					3
1022 ECHOVIRUS TYPE 22						1			1
1024 ECHOVIRUS TYPE 24	1								1
1100 POLIOVIRUS NOT TYPED			4						4
1101 POLIOVIRUS TYPE 1	1							1	2
1103 POLIOVIRUS TYPE 3	2								2
1200 MUMPS VIRUS	1			1	1			1	4
1300 HERPES VIRUS GROUP-NOT TYPED	28			4				2	34
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2				2		2	6
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	32	1	1					5	39
1303 VARICELLA-ZOSTER VIRUS	3			1		1	1	1	7
1306 HERPES SIMPLEX TYPE 1	34			34		24	23	14	129
1307 HERPES SIMPLEX TYPE 2	164		1	65		14	77	39	360
1399 HERPES VIRUS TYPING PENDING					3				3
1401 COXIELLA BURNETI	2		1				36		39
1502 PICORNA VIRUS-NOT TYPED	3	1	13				15	1	33
1521 MEASLES VIRUS		1							1
1522 RUBELLA VIRUS	2	1	1	6			6	1	17
1532 HEPATITIS B ANTIGEN	103	1	7	34	2	20	9	17	193
1535 HEPATITIS A ANTIBODY	13	1	2	2		13		18	49
1541 CHLAMYDIA A - C TRACHOMATIS	48		4	63		32	1	61	209
1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART)				1					1
1556 CMV - CYTOMEGALOVIRUS	16			31		6	3	7	63
1562 REOVIRUS (ALL TYPES)				1					1
1564 ROTAVIRUS	14	2	6		4	20		1	47
1571 ENTEROVIRUS TYPE 71 (BRCR)			4	4					8
1599 ENTEROVIRUS TYPING PENDING		3	12		14				29
9990 AUSTRALIAN ENCEPHALITIS								1	1
9992 ROSS RIVER VIRUS			1				173	8	182
9993 ASTROVIRUS	1								1
9994 SMALL VIRUS (LIKE) PARTICLE	1	4				1			6
9995 DENGUE							2		2
Total	530	28	75	259	80	155	356	189	1,672

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 12/5/86 - 25/5/86
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									
0101 ADENOVIRUS TYPE 1.....	1	1				1	2				
0102 ADENOVIRUS TYPE 2.....	2	4					2				
0103 ADENOVIRUS TYPE 3.....		2					1				
0104 ADENOVIRUS TYPE 4.....							1				
0105 ADENOVIRUS TYPE 5.....	1										
0106 ADENOVIRUS TYPE 6.....				1							
0109 ADENOVIRUS TYPE 9.....							1				
0111 ADENOVIRUS TYPE 11.....							1				
0113 ADENOVIRUS TYPE 13.....							1				
0131 ADENOVIRUS TYPE 31.....							1				
0201 INFLUENZA A VIRUS.....		2									
0203 INFLUENZA B VIRUS.....		1								1	
01 PARAINFLUENZA VIRUS TYPE 1....		18									
02 PARAINFLUENZA VIRUS TYPE 2....	1	22									
0303 PARAINFLUENZA VIRUS TYPE 3....		5									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	2	24									
0500 RHINOVIRUS (ALL TYPES).....		9									
0600 MYCOPLASMA PNEUMONIAE.....	3	12						1	1		1
0700 ORNITHOSIS-PSITTACOSIS.....		4									
1002 ECHOVIRUS TYPE 2.....							1				
1007 ECHOVIRUS TYPE 7.....						1					
1011 ECHOVIRUS TYPE 11.....						1	1				1
1013 ECHOVIRUS TYPE 13.....							1				1
1018 ECHOVIRUS TYPE 18.....	1										
1020 ECHOVIRUS TYPE 20.....	1					1	1				
1021 ECHOVIRUS TYPE 21.....	1			2							
1022 ECHOVIRUS TYPE 22.....		1									
1024 ECHOVIRUS TYPE 24.....				1							
1101 POLIOVIRUS TYPE 1.....							2				
1103 POLIOVIRUS TYPE 3.....	1						1				
1200 MUMPS VIRUS.....	1	1		1							
1300 HERPES VIRUS GROUP-NOT TYPED..											2
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1							3
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	7	2		2		1		4			1
1303 VARICELLA-ZOSTER VIRUS.....	1				1						5
1306 HERPES SIMPLEX TYPE 1.....	4	12	1							4	40
17 HERPES SIMPLEX TYPE 2.....	8			1							64
01 COXIELLA BURNETI.....	5	14						1	1		1
1502 PICORNA VIRUS-NOT TYPED.....		6	2	3			18		1		3
1521 MEASLES VIRUS.....			1								
1522 RUBELLA VIRUS.....	3	3									8
1532 HEPATITIS B ANTIGEN.....	69							110			
1535 HEPATITIS A ANTIBODY.....	14							32			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1			1							
1555 PAPOVAVIRUS GROUP (PAPILLOMA-HUMAN WART).....	1										
1556 CMV - CYTOMEGALOVIRUS.....	8	13				3		2		5	2
1562 REOVIRUS (ALL TYPES).....			1								
1564 ROTAVIRUS.....		1					46				
1571 ENTEROVIRUS TYPE 71 (BRCR)....				4							4
9990 AUSTRALIAN ENCEPHALITIS.....			1								
9992 ROSS RIVER VIRUS.....	37	4							1		48
9993 ASTROVIRUS.....							1				
9994 SMALL VIRUS (LIKE) PARTICLE...							6				
9995 DENGUE.....	1					1					
Total.....	174	162	6	17	1	9	88	150	5	9	184

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 12/5/86 - 25/5/86
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.....								2		
0104 ADENOVIRUS TYPE 4.....									1	
0108 ADENOVIRUS TYPE 8.....	7									
0126 ADENOVIRUS TYPE 26.....									1	
0127 ADENOVIRUS TYPE 27.....									1	
0201 INFLUENZA A VIRUS.....									1	
0203 INFLUENZA B VIRUS.....								1		
0302 PARAINFLUENZA VIRUS TYPE 2....								1		
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0500 RHINOVIRUS (ALL TYPES).....				1					1	
0600 MYCOPLASMA PNEUMONIAE.....								4	2	
0700 ORNITHOSIS-PSITTACOSIS.....								1		
1003 ECHOVIRUS TYPE 3.....								1		
1005 ECHOVIRUS TYPE 5.....									1	
1011 ECHOVIRUS TYPE 11.....									1	
1020 ECHOVIRUS TYPE 20.....									1	
1101 POLIOVIRUS TYPE 1.....										1
1200 MUMPS VIRUS.....				1						
1301 HERPES SIMPLEX VIRUS NOT-TYPED									2	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).		1	13	1		1	3	4	3	
1303 VARICELLA-ZOSTER VIRUS.....				1						
1306 HERPES SIMPLEX TYPE 1.....	3	63							3	
1307 HERPES SIMPLEX TYPE 2.....		292								
1401 COXIELLA BURNETI.....					7		1	32		
1502 PICORNA VIRUS-NOT TYPED.....					1		1	2	1	
1522 RUBELLA VIRUS.....					2	2	1	2		
1532 HEPATITIS B ANTIGEN.....						1			13	
1535 HEPATITIS A ANTIBODY.....									3	
1541 CHLAMYDIA A - C.TRACHOMATIS...	2	203				1			2	
1556 CMV - CYTOMEGALOVIRUS.....			2	1		5	3	11	18	
1564 ROTAVIRUS.....									1	
9992 ROSS RIVER VIRUS.....					125			41		
Total.....	12	559	17	3	135	10	9	103	56	1

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

1 January 1985 to 31 December 1985

Bulletin ...86/11...

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	520	105	332	223	336*	2	951	14	*WA 2,483
Tetanus		1	4	4	1				10
Trachoma	1	N.N.	3		59	N.N.	N.N.		63
Tuberculosis (all forms)	403	293	142	99	110	1	33	23	1,104
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.

IMPORTANT NOTICE Our ref 86/4465

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