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Communicable Diseases Intelligence

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This is the final issue of CDI for 1986 and includes a subject index for the year. The next issue will be published on 19 January 1987, and will contain a compilation of the reports for the two generations, 8-21 December and 22 December - 4 January 1987. The editorial staff takes this opportunity to thank all the participating laboratories for their regular contributions to communicable diseases surveillance and extend the seasons greetings to all our readers, with best wishes for the New Year.

VIRUS REPORTING SCHEME: A total of 1 405 reports were processed for this period.

Twenty cases of Q fever were reported, 11 from Queensland, 4 from New South Wales, 4 from Victoria and 1 from South Australia. Occupational exposure data were only available for 6 of the Queensland cases:-

- 4 male meatworkers (2 from Beenleigh aged 20 and 22 years respectively, 1 from Toowoomba aged 15 years and 1 from Townsville aged 29 years).
- one 46 year old female grazier from Roma.
- one 32 year old male truck driver from Mt Isa.

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

Specific IgM antibody to cytomegalovirus was detected in the blood of a 23 year old female who had a foetal death in utero.

An increase in the number of cases of aseptic (viral) meningitis caused by echovirus type 11 became apparent during the last 2 reporting periods. Routine monitoring indicates an apparent increase in:

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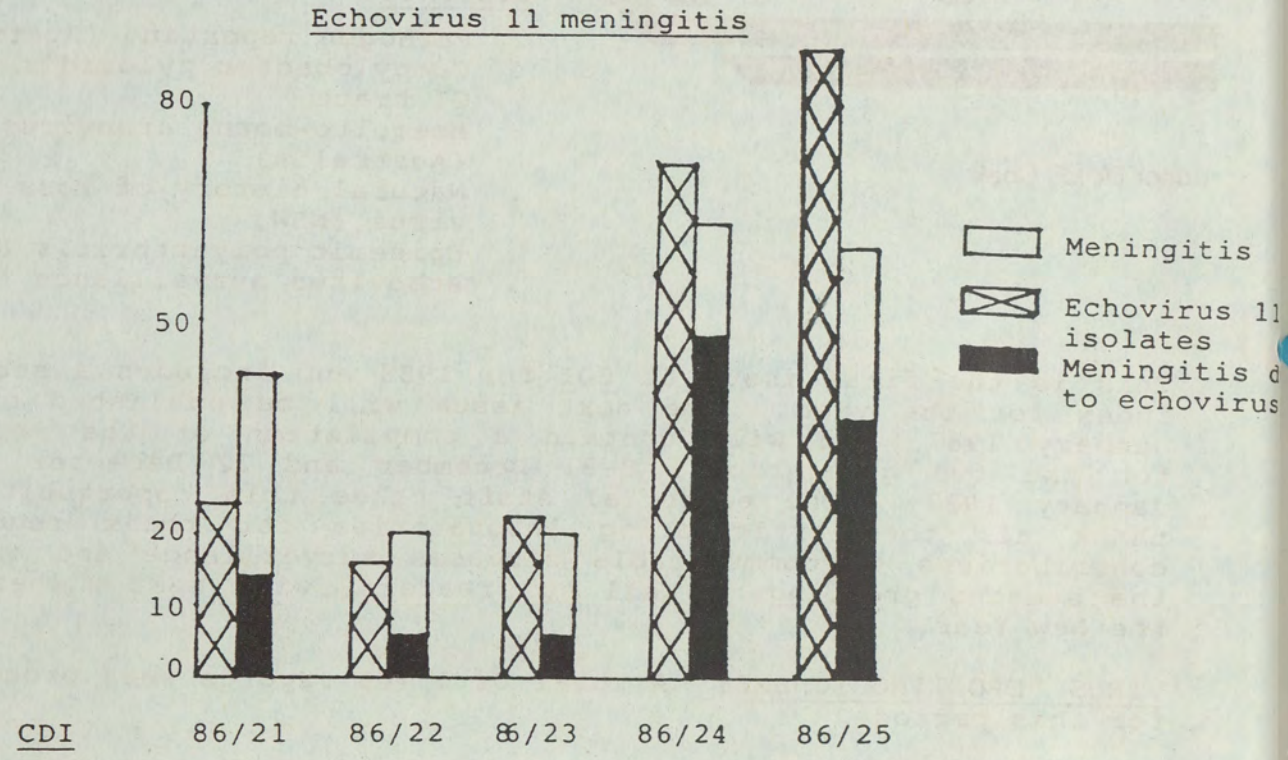
(i) the incidence of echovirus type 11 isolates
(ii) the number of reported cases of viral meningitis
(iii) the proportion of viral meningitis cases caused by echovirus 11. (Figure).



The viral meningitis cases reported for the period occurred almost exclusively in children with less than 20% of the cases in children less than one year. A number of studies have suggested that enteroviral meningitis in the first year of life may result in permanent neurologic sequelae, such as spasticity, diminished head circumference, and impaired intellectual functions. However, among echoviruses, type 11 is the most firmly established and possibly the most common cause of respiratory disease, although types 4, 8, 9, 20, 22 and 25 appear to be responsible for similar illnesses. Echovirus 11 produces sore throat, coryza, cough and sometimes fever. It has also been associated with croup.

Routine surveillance of echovirus activity is usually indicated in the summer months. While hospitalisation is not always necessary for all cases of aseptic meningitis during summer epidemics of enterovirus infections, it is advisable for sporadic cases or when the presence of disturbances of consciousness, muscle weakness, or a petechial rash suggest the possibility of a more serious illness, such as encephalitis, poliomyelitis, or meningococcaemia.

COMMUNICABLE DISEASE INTELLIGENCE EXPANSION SCHEME

PATHOGEN REPORT - SEPTEMBER 1986

PATHOGEN REPORTING SCHEME: A total of 388 reports (261 bacterial, 71 fungal, 45 protozoan and 4 helminthic infections) were processed for the month of September 1986.

These reports were contributed by the following laboratories:

Laboratories	Bacteria	Fungi	Protozoa	Helminths	Total
Commonwealth Laboratory (Toowoomba)	6	2	4		12
State Health Laboratory (Brisbane)	48	26	35	3	112
Royal Brisbane Hospital	111	39	6		156
Royal Newcastle Hospital	7	3			10
St. George Hospital	41			1	42
Institute of Clinical Pathology & Medical Research	46	1			47
Royal Hobart Hospital	9				9
TOTAL	268	71	45	4	388

Pathogen reports collected this month featured the following:

I. BACTERIAL INFECTIONS

A/Zoonoses:
- Brucella species (1) due to B. suis
- Leptospira species (8) which included
  - L. pomona (2)
  - L. hardjo (3)

B/Bacterial meningitis (10) due to:
- Streptococcus species (4) which included
  - S. pneumoniae (2)
- Staphylococcus species (1)
- Escherichia coli (1)
- Haemophilus influenzae (1)
- Neisseria meningitidis (2)

C/Bacteraemia (59) due to:
- Staphylococcus species (14) which included
  - S. aureus (3)
  - MRSA (methicillin - resistant S. aureus) (3)
- Streptococcus species (8) which included
  - Streptococcus agalactiae (1)
  - S. faecalis (1)
- Pseudomonas species (2) which included
  - P. aeruginosa (1)
- Bacteroides species (5) which included
  - Bacteroides fragilis (1)

- Proteus species (4) which included
  Proteus (Morganella) morgagnii (2)
- Providencia rettgeri (1)
- Providencia stuartii (1)
- Klebsiella species (5)
- Escherichia coli (16)
- Neisseria meningitidis (3)

II. FUNGAL INFECTIONS

- Candida species (46) which included
  C. albicans (7)
  Torulopsis glabrata (3)
- Cryptococcus species (5) which included
  C. neoformans (1)
- Trichophyton species (13) which included
  T. rubrum (3)
  T. mentagrophytes (4)
- Aspergillus species (2)
- Microsporum species (2)
- Epidermidophyton (species) floccosum (3)

III. PROTOZOAN INFECTIONS

- Cryptosporidium species (2)
- Giardia lamblia (10)
- Trichomonas vaginale (11)
- Plasmodium falciparum (17)
- Plasmodium vivax (5)

IV. HELMINTHIC INFECTIONS

- Necator americanus (1)
- Trichuris trichiura (1)
- Echinococcus granulosis (2)

CAMPYLOBACTER PYLORIDIS IN THE GASTROINTESTINAL TRACT

(based on CDR 86/36, 5 September 1986)

A new spiral Gram-negative bacterium has been recognised and isolated from the gastric mucosa of patients with gastritis[1,2]. It was been reported that:-

- the curved bacilli were almost present in 'active chronic gastritis',
- the bacteria were rare when there was no inflammation,
- the bacilli and the histological changes were most often seen in the gastric antrum but could be present in any part of the stomach.

In 1983 it was reported that these curved bacilli could be cultured on moist chocolate agar at 37°C in microaerophilic conditions where they produced a faint transparent layer of growth in 3 to 4 days.

It was observed that 58% of patients presenting for gastroscopy had spiral or curved bacilli in biopsy specimens of antral mucosa; leading to the conclusion that a new species of bacteria related to the genus campylobacter was present in almost all patients with active chronic gastritis, duodenal ulcer, or gastric ulcer and that the bacteria may be an important factor in the aetiology of these diseases[3].

The ultrastructure of these organisms, now called C. pyloridis, showed more morphological resemblances to Spirillum than to Campylobacter. The multiple flagella were sheathed and usually monopolar, and 12 nm protein subunits ("doughnuts") were arranged on the surface similar to those seen in Aquaspirillum. It was thus concluded that this gastric bacterium should be placed in a new genus[4]. The organism was characterised by researchers from Australia, Europe, Japan, Yugoslavia, Spain and North America who confirmed the association of C. pyloridis with gastritis, but the clinico-pathological correlation with gastric ulcer is less clear[5]. A review of early reports of gastritis, and the reconstruction of the natural history of antral gastritis demonstrated C. pyloridis in 114 of 267 patients who had undergone antral biopsy; and the bacterium was isolated from 88% of patients in whom it was detected histologically.

The original hypothesis that antral gastritis was caused by C. pyloridis was extended to suggest that the subsequent mucosal damage predisposed their patients to acid and peptic digestion (ulceration) by interfering with the mucus barrier. A further extension of this 'antral gastritis' hypothesis was made to propose that C. pyloridis also damaged the duodenal epithelium and was responsible for the duodenitis almost always present in patients with duodenal ulcer. It was therefore suggested that the acute form of the disease would be most common in previously unexposed individuals including children or young adults of families with a history of peptic ulceration or chronic dyspepsia. After ingestion of the infective agent (C. pyloridis) the patient develops, in about a week, an acute gastrointestinal disturbance characterised by epigastric discomfort, nausea and vomiting in 50% of cases, the remainder being asymptomatic. In the acute stage the vomitus will contain acetic and fatty acids with a reduced amount of hydrochloric acid. Halitosis may be a feature. A mild gastrointestinal disturbance may persist in some patients together with achlorhydra. This phase lasts 3 to 12 months during which time the histological pattern of chronic gastritis develops, with the polymorphs replaced by lymphocytes and plasma cells. As immunity to the infection increases, the inflammation in the body of the stomach regresses and acid secretion returns. The most severely affected mucosa will then be in the antrum and pyloric canal where bacterial growth will be less inhibited by acid secretion. In this final stage of the infection chronic inflammation of the gastroduodenal mucosa persists, but acid secretion returns to normal levels. Peptic ulceration may then develop. Serial microbiological and histological data from patients with the acute syndrome may

show that pyloric campylobacter infection is the factor linking acute achlorhydric gastritis, chronic gastritis, and peptic ulceration. In an attempt to prove Koch's postulate, a one-man volunteer study demonstrated that the ingestion of 10^9 C. pyloridis cells in 10 mL of alkaline peptone water after fasting overnight and taking a 600 mg dose of cimetidine to produce temporary achlorhydria, produced on the 7th day following ingestion a mild illness. The symptoms lasted 14 days and gastritis was histologically proven on the 10th day. It has been suggested that this acute disorder may progress to a chronic infection which predisposes to peptic ulceration.

However, does colonisation of the stomach by C. pyloridis cause infection? Antibody to the organism has been demonstrated in patients by complement fixation tests, ELISA and microagglutination[5]. A study comparing the histological grades of gastritis with isolation of C. pyloridis and the serum IgG values suggested that the isolation of this agent was associated with both superficial and chronic gastritis and chronic gastritis with intestinal metaplasia whereas atrophic gastritis was associated with a lower rate of isolation of C. pyloridis[6]. The mean serum IgG values were significantly different in the sera from patients with gastritis proven on antral biopsy as compared to the IgG levels in people with symptoms but without proven gastritis. The data also suggest that, using a C. pyloridis specific ELISA test, there is a difference in serum IgG, and to a lesser extent in serum IgA, between patients with gastritis, gastric or duodenal ulcer and age-matched controls. Such findings are of interest but are not evidence of pathogenicity.

In developing tools to investigate transmission and sources, the protein profiles were examined by SDS-polyacrylamide gel electrophoresis (PAGE) and enzymatic activity using 109 substrates[5]. Unpublished data from a collaborative study in Peru suggested that C. pyloridis strains isolated repeatedly from patients appear to be similar. If confirmed this could mean either that patients remain infected with one type of C. pyloridis or that they are reinfected with strains of the same source. Enzyme profile analysis may also prove useful for fingerprinting strains but the strong urease activity and characteristic morphology are the methods of choice for routine identification of C. pyloridis.

It has been suggested that since bacterial urease activity can be detected in the stomach of some patients, urease production may be a pathogenicity factor. Sections of gastric biopsy material show that the organisms lie in planes in the mucus layers covering the epithelium and on the epithelial surface at the intercellular junctions of the mucus secreting cells. This observation has led to the hypothesis that C. pyloridis has a spiral morphology which enables it to move efficiently in a viscous environment and that the bacteria are attracted to the epithelial surface by the chemotactic influence of preferred metabolites diffusing from the tissue. Levels of enzymatic activity reflects the bacterial environment and a high urease activity could mean that urea, being readily accessible at the cell surface, is one of the preferred metabolites.

Colonisation of the stomach by C. pyloridis and the presence of gastritis may alter gastric permeability so enhancing bacterial growth which in turn could lead to the bulk diffusion of the hydrogen ions necessary for gastric and duodenal ulceration.

Circumstancial evidence that C. pyloridis may be pathogenic is provided by two further areas of investigation:

- the demonstration by western blot techniques that the serum of patients with gastritis have specific antibody to outer membrane proteins;
- the effect of chemotherapeutic agents on C. pyloridis isolation and the response of the patient to such treatment. The protective and therapeutic effects of anti-ulcer drugs and antibiotics are best described by the anti-ulcer effect of furazolidine in gastric ulcer models which showed a 73% cure rate with short-term furazolidine therapy as against a 24% rate with placebo in the treatment of peptic ulcer in man.

As C. pyloridis appears to be susceptible to a variety of anti-ulcer drugs and antibiotics some of these could be used to study the aetiological role of C. pyloridis in various clinical disease states. In 82 patients who underwent endoscopy for abdominal complaints, the clinico- pathological effects of treated patients with agents known to reduce or eliminate C. pyloridis, indicated that C. pyloridis associated gastritis did not resolve spontaneously, and was eliminated by some anti-bacterial drugs but not by anti-ulcer drugs like cimetidine and sucralfate. However, resolution after treatment with amoxycillin led to the conclusion that C. pyloridis caused the associated gastritis[7].

A review of published evidence implicating C. pyloridis as a gastric pathogen is supported by:-

- a report of epidemic gastritis in which 17 of 37 healthy volunteers and one patient became profoundly hypochlorhydric and developed fundal and antral gastritis after endoscopy. C. pyloridis was subsequently demonstrated in one biopsy specimen[8],
- a report of epidemic hypochlorhydria[9].

Is C. pyloridis an aetiological factor in gastritis? The above evidence suggests that under certain host conditions C. pyloridis may be a pathogen.

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SEASONAL PATTERN OF MOSQUITO-BORNE ARBOVIRUS DISEASE IN AUSTRALIA

Three significant mosquito-borne arbovirus disease affect man in Australia. These are australian encephalitis (AE), dengue and epidemic polyarthritis (EPA).

AUSTRALIAN ENCEPHALITIS

AE is caused by infection with a flavivirus - Murray Valley Encephalitis virus (MVEv). There are also two reports in the literature where the aetiological agent has been identified as Kunjin virus a related flavivirus. The disease is characterized by sudden onset with fever, headache, nausea, vomiting and non-specific dizziness, followed after 2 to 5 days by symptoms of meningitis and brain dysfunction(1). Asymptomatic infections are very common(2). Sequellae are seen in about 30% of cases and case fatality rates range from 20-30%.

MVEv is active annually in northern WA (January to May/June)(3), with cases of AE recorded every 2-3 years between February and June(4). AE cases are less frequent (every 3-10 years) in north-west WA, NT and north Qld, with scattered single cases, or, at most, small outbreaks of up to 8 cases.(4) More serious epidemics, centred on the Murray Valley, occur every 15 to 30 years with cases recorded from early January to mid April. Occasional small outbreaks (2-3 cases) are also recorded in the south-east. The most recent epidemic occurred in 1974 and resulted in 58 cases (13 deaths), with cases recorded from all mainland States and the NT.(2,4) AE epidemics and activity in the south-east are associated with excessive summer rainfall(2).

MVEv is transmitted by the mosquito Culex annulirostris and has a natural cycle involving wild birds and possibly mammals(9). Other vectors may be involved, particularly in northern Australia. Recent speculation about the possible interepidemic survival of MVEv through trans-ovarial transmission (TOT) in Aedes species has yet to be demonstrated in the field, though laboratory evidence of TOT in Aedes aegypti (not a recognized vector) has been reported(5).

Kunjin virus has very similar ecology and activity, but the symptoms of Kunjin infection are generally less severe.

DENGUE

Dengue, a flavivirus transmitted by Ae aegypti, is maintained in a man-mosquito cycle. Both disease and vector are restricted to Queensland, particularly the northern coastal towns of the State(6). Four serotypes of dengue virus are recognized, and all cause similar disease. Dengue fever is characterized by sudden onset of high fever, myalgia, arthralgia and headache. The more serious manifestations of dengue haemorrhagic fever and dengue shock syndrome have not been recorded in the most recent dengue outbreaks in Australia.

Epidemics of dengue occur periodically, and generally last 1-2 years. In the intervening periods relatively fewer cases are reported, and most of these are probably imported. Cases in the most recent epidemic (1981-1983) occurred throughout the year, with the greatest number early and late in the Summer[6]. Although TOT of dengue in Ae aegypti has been demonstrated, this does not appear to be significant in long term maintenance of dengue in Australia.

EPIDEMIC POLYARTHRITIS (EPA)

EPA (Ross River fever) is caused by infection with the alphavirus, Ross River virus (RRv). Symptoms of RRv infection are rare in pre-pubertal infection and about 60% of infections are asymptomatic in adults. Clinical diagnosis is based on polyarthralgia, often accompanied by a variable rash, headache. fever, or muscle pain[7].

Numerous mosquito species have been found naturally infected with RRv, but the major vectors are Aedes vigilax (a tidal salt marsh species) near the coast, and Cx annulirostris in more inland sites[7]. The relevance of preliminary data demonstrating laboratory TOT of RRv by Ae vigilax[8] to the field survival of the virus is not known. Vertebrate hosts of RRv are mammals, particularly the large macropods[7,9], though man-mosquito cycles are also suspected[7].

EPA occurs as annual outbreaks throughout Australia. The intensity of local transmission is dependent on a number of factors, particularly the distribution and amount of rainfall, as this affects the populations of the vectors. Unseasonal or heavy rainfall in normally arid areas of Australia can sometimes result in outbreaks of EPA. The largest number of EPA cases are recorded in the Summer months when the mosquito vectors are most active.

CDI Editorial Comment

None of these diseases can be diagnosed with certainty on the basis of clinical signs and symptoms alone. Serological confirmation should be requested whenever an arboviral disease is being considered in a patient.

All arboviral diseases are notifiable in all States and Territories of Australia, and it is essential that reports are supported by appropriate serological data. The epidemiology of these diseases is being studied by a working group established under the auspices of the National Diseases Control Program, initiated by the Commonwealth Department of Health. One of the aims of the working group is to maintain a register of arboviral diseases in Australia and to distribute data therein regularly to medical practitioners. A prerequisite to the success of this venture is that notification be improved, as it is apparent that arboviral disease incidence is currently underreported.

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A STUDY* OF THE NATURAL HISTORY OF ROSS RIVER VIRUS ON THE SOUTH COAST OF NEW SOUTH WALES

(Contributed by M.J. Cloonan, Prince Henry Hospital, Sydney and R.C. Russell, School of Public Health and Tropical Medicine, University of Sydney)

*(Supported by the Commonwealth Department of Health under the National Diseases Control Program)

A longitudinal study to define the natural history of Ross River virus is currently being conducted on the south coast of New South Wales to complement previous studies[1-4] which have demonstrated arbovirus (including Ross River virus) activity in terms of seasonal prevalence, host attraction and longevity of the mosquito fauna in a native forest of that region.

As part of the study, adult (30 000 female mosquitoes trapped at field sites) and immature mosquitoes (11,500 larvae or pupae collected and maintained until adult emergence) were collected between September 1985 and June 1986 from:

- four sites around Batemans Bay (including one site in Mogo State Forest)
- two sites in both Termeil and Conjola State Forests.

A total of 29 mosquito species were identified. No viruses were isolated from adult mosquitoes emergent from field-collected larvae and pupae. However 68 isolates were obtained from field-collected adults which were processed for virus isolation by initial inoculation of C6/36 cells and subsequently BHK-21 and vero cells. Ninety four percent of viral isolates were identified as Ross River virus recovered from Aedes vigilax captured between January and March 1986. Further studies may elucidate the basic maintenance cycle of the virus - mosquito - vertebrate host relationships.

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EPIDEMIC POLYARTHRITIS IN NEW SOUTH WALES: OCTOBER 1985 - MAY 1986*

(Contributed by H.M. Naim, University of New South Wales, and M.J. Cloonan, Prince Henry Hospital - for New South Wales Arbovirus Control Committee)

* (Supported in part by the Commonwealth Department of health and the NSW Department of Health under the National Diseases Control Program)

Diagnostic laboratories in Queensland, New South Wales, Victoria and South Australia have confirmed the following number of cases of epidemic polyarthritis (Ross River virus infections) in relation to the biophysical zone[1] of New South Wales during the previous spring-autumn period:-

Ross River virus infections in New South Wales (Oct 85 - May 86)

Biophysical zone	No. of cases	Biophysical zone	No. of cases
Coast - Far North	5	Slopes - North West	5
- Mid North	9	- Central West	9
- Central	2	- South West	7
- South	2	Plains - North West	82
Tablelands - Northern	0	- Far West	0
- Central	0	- South West	88
- Southern	1	Not known	25
Hunter	2		
		TOTAL	237

The original data indicated that approximately 60% of cases occurred in people living in or around four country towns:-
. Narrabri, Moree and Bourke (in the North West Plains) and
. Griffith (in the South West Plains).

The number of epidemic polyarthritis infections recorded during 1985-86, although considerably lower than that reported during the 1983-84 epidemic[2], still indicates the annual recurrence of Ross River virus activity in New South Wales.

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ARBOVIRUS SURVEILLANCE* - NEW SOUTH WALES (1985-86)

(Contributed by H.M. Naim, R.A. Hawkes, C.R. Houghton, B. Myrick, L. Ramsay, R. Beattie - Arbovirus Research Unit, University of New South Wales).

* (Supported by the Commonwealth Department of Health and the NSW Department of Health under the National Diseases Control Program)

During the summer of 1985-86, throughout the state of New South Wales, sentinel chicken flocks (of approximately 15 chickens in each flock) were monitored for alphavirus and flavivirus antibodies, with the first bleed on 13 December 1985 and the final bleed at the end of April 1986:

- weekly bleeding was done at Wentworth, Berrigan, Leeton, Hay, Griffith and Deniliquin
- monthly bleeding was done at Moree, Menindee, Broken Hill, Gunnedah, Coffs Harbour, Kempsey, Narrabri, Macquarie Marshes, Warren and Bourke

The positive results of haemagglutination inhibition (HI) test for antibody against:

- alphaviruses, ie Sindbis (SIN) and Barmah Forest (BF)
- flaviviruses, ie Murray Valley Encephalitis (MVE), Kunjin (KUN), Alfuy (ALF), Stratford, Kokobera and Edge Hill are summarized below.

Seroconversions in Sentinel Chickens in NSW (1985-86)

TOWN	SIN (sero-conversions)	Date of detection	
Leeton	2	2/4/86	
Griffith	4	1/2/86	+ *1 flavivirus seroconversion
Macquarie Marshes	2	2/2/86	
Warren	1	8/2/86	
Bourke	6	3/2/86	

*HI antibodies to MVE 1:20, KUN 1:40 and ALF 1:40 were decteted on 6/1/86. Plaque neutralisation tests failed to serotype the virus but the chicken subsequently died.

The profile of seroconversions taken place between November 1985 and July 1986 included:

- 15 SIN seroconversions in chickens (Table above) which were detected later in the season than in previous years.
- of 23 dogs monitored in Griffith, 2 seroconverted to an alphavirus, (1 to Ross River virus, 1 to Sindbis virus) and 1 to both an alphavirus and a flavivirus.
- no seroconversion was detected in a sentinel cattle herd at Kempsey and sentinel dogs at Bourke.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 24-11-86 - 7-12-86 BULLETIN NUMBER 86/25
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	7	1	3		10	1	4	2	28
0101 ADENOVIRUS TYPE 1	2			2		1		1	6
0102 ADENOVIRUS TYPE 2	2			3		4		1	10
0103 ADENOVIRUS TYPE 3	1			1		4		1	7
0105 ADENOVIRUS TYPE 5				2		2			4
0108 ADENOVIRUS TYPE 8				1				1	2
0109 ADENOVIRUS TYPE 9	1								1
0111 ADENOVIRUS TYPE 11	1								1
0199 ADENOVIRUS TYPING PENDING		1			6				7
0201 INFLUENZA A VIRUS	2		2						4
0203 INFLUENZA B VIRUS	3		1	1					5
0206 INFLUENZA A VIRUS SUBTYPE H1N1				1					1
0301 PARAINFLUENZA VIRUS TYPE 1					1	1			2
0302 PARAINFLUENZA VIRUS TYPE 2				1		1			2
0303 PARAINFLUENZA VIRUS TYPE 3	3	3		1	6	11	3		27
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	4		1		1	9		12	27
0500 RHINOVIRUS (ALL TYPES)	1	1		1	13	19	1	1	37
0600 MYCOPLASMA PNEUMONIAE	9		4	5		4	5	13	40
0700 ORNITHOSIS-PSITTACOSIS	3			8		1			12
1002 ECHOVIRUS TYPE 2						1			1
1005 ECHOVIRUS TYPE 5				6				2	8
1011 ECHOVIRUS TYPE 11	9	2		16	10	4		1	42
1014 ECHOVIRUS TYPE 14					1				1
1018 ECHOVIRUS TYPE 18					2				2
1100 POLIOVIRUS NOT TYPED			3						3
1101 POLIOVIRUS TYPE 1						1			1
1103 POLIOVIRUS TYPE 3						1		1	2
1104 POLIOVIRUS-VACCINAL STRAIN					1	1			2
1200 MUMPS VIRUS				2				1	3
1300 HERPES VIRUS GROUP-NOT TYPED	38			1		1		1	41
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1		3					4
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	11		4	12			12	12	51
1303 VARICELLA-ZOSTER VIRUS	2		2	1	4			1	10
1306 HERPES SIMPLEX TYPE 1	23			40		19	31	22	135
1307 HERPES SIMPLEX TYPE 2	111			70		11	64	38	294
1399 HERPES VIRUS TYPING PENDING				1	7				8
1401 COXIELLA BURNETI	4			4		1	11		20
1502 PICORNA VIRUS-NOT TYPED	6		5	1			6	4	25
1521 MEASLES VIRUS				6				2	8
1522 RUBELLA VIRUS	18		4	8		7		6	43
1531 HEPATITIS B VIRUS				1					1
1532 HEPATITIS B ANTIGEN	83		4	17		9	16	22	151
1535 HEPATITIS A ANTIBODY	2		1	4		3	1	9	20
1541 CHLAMYDIA A - C TRACHOMATIS	24					57	30	82	193
1556 CMV - CYTOMEGALOVIRUS	10	1	2	8	2	10	2	11	46
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	19		4	1	3	11			38
1571 ENTEROVIRUS TYPE 71 (BRCR)	1			1					2
1599 ENTEROVIRUS TYPING PENDING		1	10		14				25
9994 SMALL VIRUS (LIKE) PARTICLE		1							1
Total	400	12	53	230	81	195	186	248	1,405

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24-11-86 - 7-12-86

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir-atory	Enceph-alitis	Mening-itis	Para-lysis	CNS other unspec	GI	Hepa-tic	CVS	Urin-ary	Skin/ mucs memb
0100 ADENOVIRUS NOT TYPED.....							1				
0101 ADENOVIRUS TYPE 1.....		5					1				1
0102 ADENOVIRUS TYPE 2.....		8					1				
0103 ADENOVIRUS TYPE 3.....		2					1				
0105 ADENOVIRUS TYPE 5.....		2									
0109 ADENOVIRUS TYPE 9.....	1										
0111 ADENOVIRUS TYPE 11.....							1				
0201 INFLUENZA A VIRUS.....		3							1		
0203 INFLUENZA B VIRUS.....	1	3					1				
0301 PARAINFLUENZA VIRUS TYPE 1....		1									
0302 PARAINFLUENZA VIRUS TYPE 2....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....		27									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1	26									
0500 RHINOVIRUS (ALL TYPES).....		35									1
0600 MYCOPLASMA PNEUMONIAE.....	5	29						1			1
0700 ORNITHOSIS-PSITTACOSIS.....	1	11									
1002 ECHOVIRUS TYPE 2.....				1							
1005 ECHOVIRUS TYPE 5.....	1	1		6							
1011 ECHOVIRUS TYPE 11.....	3	4		18		1	3				2
1014 ECHOVIRUS TYPE 14.....											1
1018 ECHOVIRUS TYPE 18.....		1		2							
1101 POLIOVIRUS TYPE 1.....							1				
1103 POLIOVIRUS TYPE 3.....	1					1					
1104 POLIOVIRUS-VACCINAL STRAIN....	1	1									
1300 HERPES VIRUS GROUP-NOT TYPED..											2
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	10	6			1			2			3
1303 VARICELLA-ZOSTER VIRUS.....	3	3	1			1					2
1306 HERPES SIMPLEX TYPE 1.....	2	6		1							69
1307 HERPES SIMPLEX TYPE 2.....	7									1	76
1401 COXIELLA BURNETI.....	5	1									
1502 PICORNA VIRUS-NOT TYPED.....	3			1							1
1521 MEASLES VIRUS.....	2	2									4
1522 RUBELLA VIRUS.....											38
1531 HEPATITIS B VIRUS.....		1									1
1532 HEPATITIS B ANTIGEN.....	51	1						88			1
1535 HEPATITIS A ANTIBODY.....	7							12			
1541 CHLAMYDIA A - C.TRACHOMATIS...	18										
1556 CMV - CYTOMEGALOVIRUS.....	10	13	1	1			1	1		1	
1563 CORONAVIRUS.....		1									
1564 ROTAVIRUS.....							38				
1571 ENTEROVIRUS TYPE 71 (BRCR)....											1
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	133	195	2	30	1	3	50	104	1	2	20

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 24-11-86 - 7-12-86
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
0100 ADENOVIRUS NOT TYPED.....									1	
0102 ADENOVIRUS TYPE 2.....									1	
0103 ADENOVIRUS TYPE 3.....	4									
0105 ADENOVIRUS TYPE 5.....	1							2		
0108 ADENOVIRUS TYPE 8.....	1	1								
0203 INFLUENZA B VIRUS.....								1		
0206 INFLUENZA A VIRUS SUBTYPE H1N1								1		
0301 PARAINFLUENZA VIRUS TYPE 1....							1			
0500 RHINOVIRUS (ALL TYPES).....							1			1
0600 MYCOPLASMA PNEUMONIAE.....		1						3	4	
1011 ECHOVIRUS TYPE 11.....					2		1	4	1	3
1200 MUMPS VIRUS.....			1							
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			17	5			3	11	3	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	3	54							2	
1307 HERPES SIMPLEX TYPE 2.....		209				1				
1401 COXIELLA BURNETI.....					1			12	2	
1502 PICORNA VIRUS-NOT TYPED.....					1			4		
1522 RUBELLA VIRUS.....			1	1	1		1	2	5	
1532 HEPATITIS B ANTIGEN.....									11	
1535 HEPATITIS A ANTIBODY.....									1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	174								
1556 CMV - CYTOMEGALOVIRUS.....		6		1		3	2	4	3	
1564 ROTAVIRUS.....								1		
Total.....	10	445	19	7	5	4	9	45	35	4

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 8 - 12 July 1986 to 8 August 1986

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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	30
Ankylostomiasis				6	1		NN		7	22
Anthrax									-	-
Arbovirus infection	3		NN		1				4	1,063
Brucellosis									-	11
Campylobacter infections	93	8	NN	128	3	NN	2	1	235	1,540
Chancroid				NN	1				1	8
Cholera									-	-
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria							2		2	25
Donovanosis				NN	7		4		11	90
Giardiasis	15		NN	73	9	NN	NN	NN	97	787
Genital herpes	105				NN	NN	3	NN	108	830
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	-
Gonorrhoea	86			69	99	1	34	7	296	2,989
Hepatitis A (infectious)	43	7		58	40	1	2	1	152	1,188
Hepatitis B (serum)	43	30		8	28	1	2	1	113	* 1,137
Hepatitis – unspecified	5			2	NN	NN			7	96
Hydatid disease									-	6
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	5		NN	1	1	NN		NN	7	50
Leprosy	4	1			1				6	15
Leptospirosis	1	1		1	1	1			5	116
Lymphogranuloma venereum				NN	NN	NN		NN	-	2
Marburg disease			NN			NN		NN	-	-
Malaria	20	10		4	2		2	2	40	* 428
									-	-
Meningococcal infections	2	1				NN			3	25

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	266		NN	NN	NN	NN	NN	NN	266	2,836
Ornithosis				1					1	26
Pertussis (whooping cough)	2	1	NN	1	14	NN		NN	18	413
Plague									-	-
Poliomyelitis									-	-
Q. fever	5			6					11	179
Rabies				NN		NN		NN	-	-
Salmonella infections	50	8		22	17	1	10	2	110	1,721
Shigella infections	14	4		4	7		20		49	560
Smallpox									-	-
Syphilis	19			13	15		54		101	1,375
Tetanus									-	4
Trachoma		NN			27	NN	NN		27	62
Tuberculosis (all forms)	33	21		9	4		1	1	69	* 635
Typhoid fever	2								2	24
Typhus (all forms)									-	12
Vibrio parahaemolyticus infections			NN			NN		NN	-	5
Yellow fever									-	-
Malaria infections	1		NN	1		NN		NN	2	53

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.)

++ No figures due to 7 week return - appearing in Period 9

* Adjustment to the Cumulative Total since last report:

Hepatitis B (serum)	+1 South Australia
Malaria	+1 South Australia
Malaria	+1 Tasmania
Tuberculosis (all forms)	+3 Tasmania

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