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Communicable Diseases Intelligence

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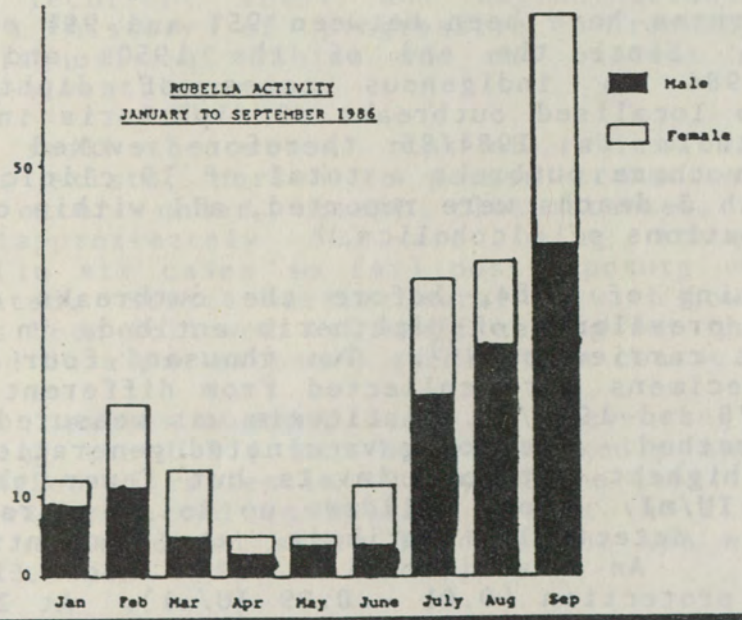
Contents:

Editor Dr I.F. Cook

VIRUS REPORTING SCHEME: A total of 1 676 reports were processed for this period.

Twenty cases of Q fever were reported (15 from New South Wales, 2 from Victoria, 2 from South Australia and 1 from Western Australia). Occupational exposure data were only available for the two South Australian cases, a 27 year old male butcher and a 44 year old male farm worker. None of the twenty patients were involved in the Q fever vaccine field trial conducted in South Australia.

An apparent rubella epidemic has been reported in North Queensland (Dr Lynch - Rockhampton). Rubella activity in the area has been monitored since the beginning of the year (Graph below) following an increase in rubella incidence observed in the month of February 1986.



The Bulletin is compiled and distributed by the Communicable Diseases Branch, Department of Health, P.O. Box 100, Woden, A.C.T. 2606, Australia, and is available on request.

Herpes Simplex Virus type 1 was isolated from:

- the saliva of a 27 year old male presenting with fever and splenomegaly due to a severe urinary tract infection following the rejection of a renal transplant
- the lesion on the nose of an adult male who was neutropenic due to acute leukaemia.

Cytomegalovirus was isolated from:

- the saliva and urine of a one year old male whose mother had toxoplasmosis and a suspected CMV infection
- the breastmilk of an adult female whose other clinical details were not available
- the bronchial washing of a 22 year old female who had juvenile dermatomyositis and pulmonary fibrosis.

IMMUNITY TO DIPHTHERIA IN THE SWEDISH POPULATION
(Based on CDR 86/32, 8 August 1986)

The first vaccination campaign against diphtheria in Sweden was started in 1943, when all school children where offered one or two diphtheria vaccine. Triple vaccine comprising diphtheria and tetanus toxoids together with the pertussis component was introduced in the beginning of the fifties and three doses were given at 4-6 week intervals, the first injection at 2-3 months of age. The diphtheria vaccines given between 1943 and 1962 were alum adsorbed, and between 1962 and 1978 plain. A booster dose of tetanus and diphtheria toxoids containing 0.5 Lf of diphtheria toxoid (Td) was introduced in 1957 for schoolchildren aged 8 years, changed in 1980 to 10 years.

The coverage rates have been between 95% and 99% of the child population[1]. Since the end of the 1950s and until the autumn of 1984 no indigenous cases of diphtheria were reported. Two localised outbreaks of diphtheria in Gothenburg and in Stockholm in 1984/86 therefore evoked justifiable attention. In these outbreks a total of 19 clinical cases of diphtheria with 3 deaths were reported, all within or connected with sub-populations of alcoholics.

At the beginning of 1984, before the outbreaks occurred, a survey of the prevalence of diphtheria antibody in the Swedish population was carried out[2]. Two thousand four hundred and nine serum specimens were collected from different age groups between 1977/78 and 1983/84. Antitoxin was measured by a micro cell culture method. The young vaccinated generation was found to have the highest antibody levels but fewer than 50% had levels 0.1 IU/ml. Among children up to 15 years of age 15% were without detectable antibody at concentrations of 0.01 IU/ml. An additional 35% had insufficient immunological protection (0.01 - 0.09 IU/ml). At 21-40 years, 43% of men and 64% of women had no immunity and at 41-65 years, innumity rates were even lower, at 56% for men and 81% for women. Only 8% of the women and 25% of the men had a reliably protective antibody titre.

An antibody concentration of 0.1 IU/ml is protective; between 0.01 and 0.09 IU/ml it will not with certainty protect against diphtheria, but may influence the course and outcome of the disease. There are other factors which influence vulnerability to diphtheria - the dose and pathogenicity of the diphtheria bacilli involved, as well as the general immunological status of the person infected. That there have been no known diphtheria cases since the fifties, in spite of the unsatisfactory immunity in the population, suggests that there are other important factors in resistance to diphtheria.

REFERENCE

1. Svenska Lakartidn (1984) 81: 1633-36
2. J Infect Dis (1986) 18: 227-33

HUMAN RABIES (U.K. ex Zambia)
(based on CDR 26/33, 15 August 1986)

In mid-June 1986, a 46 year old British woman residing in Lusaka, Zambia, was bitten on the arm and forearm when she attempted to break up a fight between her guard dog and a stray dog which broke into the compound of her home. The woman did not seek medical advice following dog bite injuries, believing herself to have been bitten by her own dog which has been fully immunised against rabies.

Two weeks following the incident she travelled to the U.K. to visit her family. She remained in good health until the evening of Saturday 9 August when she complained of extreme fatigue. Two days later, she developed fever, flu-like symptoms, and bradycardia, all of which had resolved within 24 hours. However, her condition deteriorated over the next three days with recurrent fever and hallucinations. By Friday 15 August a history of progressive hydrophobia led to a clinical diagnosis of rabies and the patient remained under sedation on a ventilator.

Although a small theoretical risk of rabies transmission via body fluids exists, person to person transmission of rabies does not occur under normal circumstances. However all contacts (approximately 50) are being traced and where necessary (in six cases so far) post-exposure vaccination has been initiated. Strict barrier nursing, with gowns, gloves and goggles has been followed and staff caring for the patient have been started on a pre-exposure vaccination course.

In the U.K., rabies control based on preventing animal rabies from entering the U.K., has been remarkedly successful since the elimination of rabies in 1902 when the last indigenous case of rabies was reported in Wales. During the first world war, rabies was reintroduced again in dogs but was eradicated once more in 1922.

The present case is the 18th imported case of human rabies reported in the U.K. since the beginning of the century. Most of the cases were reported following dog bites in the Indian sub-continent, one in Indonesia and the current case in Zambia.

CDI Editorial Comment:
If rabies occurs in Australia it will most likely follow the illegal entry by sea or air vessel of a dog, cat or other warm-blooded animals. In the event of rabies being detected in a human or animal case, a series of contingency procedures, as outlined in the National Health and Medical Research Council document, will be activated. The document entitled 'Amended Plan for the Eradication of Rabies' which was revised and endorsed by Council at its Ninety-Eighth Session in October 1984, appeared at Appendix XXVIII to the NH&MRC 1984 Session Report.

However, in addition to the information provided by the contingency plan the following advice on rabies, as published by May & Baker Australia Pty Ltd, would assist the person at risk to prevent, recognise and treat the disease.

Although Australia is at present rabies free, rabies is present in animal reservoirs in Europe, North and South America, Africa and Asia. Currently rabies control programs are being instituted in Sri Lanka, Malaysia and Indonesia in an effort to eradicate this disease. Bali is considered to be a rabies risk area by the NH&MRC and the Commonwealth Department of Health. It is considered that Australians who have been bitten by dogs or monkeys whilst visiting Bali, be offered post-exposure treatment. Rabies is spread by warm-blooded animals such as stray dogs, cats, monkeys, foxes, racoons, skunks and vampire bats.

Infections in humans:
Rabies is an almost invariably fatal disease of the central nervous system. Infection commonly occurs through being bitten by an infected animal as the virus lives in its saliva.

Protecting those at risk:
The NH&MRC recommended the following vaccination schedules:

A. Pre-exposure Management

. Pre-exposure prophylaxis should be considered for veterinarians, agricultural advisers and laboratory workers who may be exposed to the rabies virus. It is not recommended for those travellers who would have a limited chance of exposure.

. Pre-exposure Vaccination Schedule uses Merieux Inactivated Rabies Vaccine (MIRV)

Volume	Route	Schedule (days)	Booster*
1ml MIRV	Intramuscular	0, 7, 30	Every 2 years

The costs of this regime have to be met by either the individual or the employer.
This immunisation schedule should commence at least one month prior to the patient's planned departure.

*If sufficient antibody to rabies virus can be demonstrated serologically the booster is not recommended.

B. Post-exposure Management
Guide to post-exposure treatment with Merieux Inactivated Rabies Vaccine (MIRV).

1. The Unprotected Patient:

Dose	Route	Schedule (days)
1ml MIRV	Intramuscular	0, 3, 7, 14, 30 & 90.
20iu/Kg Rabies Immunoglobulin	A portion should be used to infiltrate the wound, the remainder is injected intra-muscularly	Given at the time of the first dose of vaccine

2. The Protected Patient:

Dose	Route	Schedule (days)
1ml MIRV	Intramuscular	0 and 3

N.B. Tetanus prophylaxis and measures to control bacterial infection should also be taken.

How to obtain Merieux Inactivated Rabies Vaccine

1. Pre-Exposure Prophylaxis:
A major pharmaceutical wholesaler in each State keeps stocks of MIRV and rabies immunoglobulin. Enquiries can be made to May & Baker Australia Pty. Ltd., the distributor of this product.

2. Post-Exposure Prophylaxis:
MIRV and rabies immunoglobulin are issued free of charge from the Commonwealth Serum Laboratories (CSL) to patients on an authority from the Commonwealth Department of Health (Central or Regional offices) or the Director of CSL.

The patient's case history:
The incubation period may be long and allow the contact to return home even if infection occurred thousands of kilometres away. The following details should be recorded:
- Was the person bitten or scratched by an animal while abroad?
- Where did the incident occur?
- What animal was involved?
- The reason for the exposure - was the animal provoked?

Precautions to be observed:
- Do not approach dogs
- Do not feed monkeys or hungry cats
- Do not approach wild animals no matter how friendly they might appear - remember a rabid animal often loses its fear of humans.

Action to be taken by the patient if bitten:
- Report all bites immediately
- If bitten or scratched, wash the wound at once with soap and water, or detergent and water - or at least flush it thoroughly with clean water. If possible, apply alcohol to it as well. Medical attention must be sought IMMEDIATELY. Ideally washing of the wound should be performed under medical supervision.
- Even if the patient has received, before travelling, vaccination against rabies, booster doses of vaccine will still be required in the event of suspected exposure to rabies.

GONOCOCCAL SURVEILLANCE - AUSTRALIA

(Contributed by the Australian Gonococcal Surveillance Programme (AGSP) Co-ordinator Dr J.W. Tapsall, Prince of Wales Hospital, Randwick, New South Wales 2031)

The present report provides details of penicillin sensitivities for the periods:

. January-March 1986 with 1091 isolates (Table 1)
. April-June 1986 with 1046 isolates (Table 2)

of Neisseria gonorrhoeae, examined by participating State and Territories laboratories using Standard Techniques and Procedures (1).

JANUARY-MARCH 1986

In this quarter penicillinase-producing gonococci (PPNG) were isolated in all centres with the exception of Hobart. High rates of PPNG infection , observed in Sydney and Melbourne, suggested that endemic cycles of PPNG transmission are now established. In other centres PPNG were isolated principally from travellers entering/returning to Australia, or their immediate contacts, although local contacts were responsible for the transmission of infection in some instances in Brisbane and Darwin. It was also noted that, in Canberra and Adelaide, PPNG were isolated from patients who acquired their infection in other parts of Australia emphasising the likelihood of further spread of these strains within Australia from centres where PPNG are endemic.

Gonococcal resistance to the penicillins is controlled by both chromosomal and extra chromosomal mechanisms with the chromosomal resistance manisfested as a series of gradual increases in intrinsic resistance to the penicillins. However, an exception to this trend is noted in the data from Adelaide where fully sensitive strains predominate.

Table 1: Penicillin sensitivity of isolates of N. gonorrhoeae(2) January - March 1986

Centre	Percentage of isolates		
	Sensitive	less sensitive	PPNG
Brisbane	27 (26.5)	60 (63.3)	9.2 (7.2)
Sydney	7.5 (20.5)	54.5 (59.3)	27.6 (8.8)
Melbourne	15.5 (18.2)	53 (60.2)	12.6 (10.2)
Adelaide	47.9 (46.4)	37.3 (38.3)	3.5 (2.7)
Perth	32.9 (25.8)	34.1 (48.4)	8.5 (5.7)

(Figures in parenthesis represent data for the corresponding period in 1985)

APRIL - JUNE 1986

In this quarter the incidence of PPNG isolates reported for Sydney and Melbourne remained high with 27.6% of Sydney isolates recorded as PPNG, the highest incidence reported so far. PPNG were isolated in all AGSP centres including Hobart, Canberra and Darwin, with high PPNG isolation rates reported in

Brisbane and Perth. However it is believed that, apart from Sydney and Melbourne, travellers entering or returning to Australia account for most of the cases of PPNG infection in other centres.

Over the past 18 months, strains fully sensitive to penicillin accounted for the majority of isolates reported from Adelaide. However during this quarter the trend was reversed with the less sensitive strains predominating.

The report for this quarter marks the completion of five years of gonococcal surveillance in Australia. The first report of the AGSP appeared in CDI 81/25.

Table 2: Penicillin sensitivity of isolates of N. gonorrhoeae[2] April - June 1986

Centre	Percentage of isolates		
	Sensitive	less sensitive	PPNG
Brisbane	21.6 (31.5)	58.6 (51.5)	13.8 (6.2)
Sydney	9.7 (10.2)	51.8 (70.0)	27.6 (6.7)
Melbourne	15.1 (13.4)	51.9 (55.2)	15.7 (15.2)
Adelaide	36.9 (64.0)	50.8 (22.5)	4.9 (3.4)
Perth	28.6 (36.2)	34.9 (44.1)	17.0 (3.3)

(Figures in parenthesis represent data for the corresponding period in 1985)

N.B. The author wishes to make the following corrections to the October-December 1985 AGSP report published in CDI 86/13:

1. in relation to an outbreak of infections with relatively resistant gonococci in Western Australia, the author wishes to add: "The report also features the appearance of a cluster of cases of relatively penicillin-resistant non PPNG which occurred in Western Australia during this period but which were all amenable to conventional treatment"

2. the Adelaide data reported in the Table is amended to read: "Sensitive 58.7% - Less sensitive 34.1% - PPNG 1.6%".

REFERENCES

1. Br J Vener Dis (1984) 60: 226-230
2. Communicable Disease Intelligence (CDI) Bulletin 85/23

TETRACYCLINE RESISTANT NEISSERIA GONORRHOEAE (TRNG) IN BRITISH COLUMBIA (CANADA)

(Based on Disease Surveillance Vol. 7/No. 8, 28 July 1986)

Neisseria gonorrhoea with high-level resistance to tetracycline was isolated in Vancouver in January 1986 from two patients who had treatment failures with tetracycline and from one patient who had been treated with tetracycline but could not be contacted for follow-up.

The isolates were resistant to tetracycline with a minimal inhibitory concentration (MIC) of 16 ug/ml but were uniformly susceptible to penicillin (MIC=0.124 ug/ml), erythromycin, spectinomycin and ceftriaxone. The isolated gonococci contained plasmids of approximately 24.5 and 2.6 megadaltons.

Isolates of N. gonorrhoeae that show high-level resistance to tetracycline (MIC = 16-32 ug/ml) but are susceptible to penicillin and other antibiotics were first reported in the United States in February 1985[1] but no Canadian data on similar isolates are available.

The appearance of high-level tetracycline resistance in British Columbia, supports the adoption of the Centers for Disease Control (CDC) recommendation that all positive test-of-cure cultures be screened for tetracycline resistance by disc diffusion in addition to recommended procedures for penicillinase - producing strains (PPNG) and chromosomally-resistant N. gonorrhoea (CMRNG) testing.

REFERENCE

1. Communicable Disease Intelligence bulletin 86/13.

TWAR: A NEW CHLAMYDIAL STRAIN

(based on Infectious Disease Alert: Vol 5 No 22, 15 August 1986)

Of the 2 species of chlamydia, C. psittaci and C. trachomatis, the type that causes Psittacosis has long been associated with an atypical pneumonia related to close exposure to certain birds. This often severe pneumonia can involve high fevers, hypoxia, and even pulmonary haemorrhage. C. trachomatis infections were originally seen primarily as trachoma, an illness of repeat exposure to this agent which leads to recurrent conjunctivitis, scarring, and eventual blindness, often through superinfections with other bacteria. C. trachomatis is now considered the most frequent cause of sexually transmitted diseases. In neonates, C. trachomatis strains can cause pneumonia from chlamydial infections, although it is not yet clear whether adults develop pneumonias from these agents.

A chlamydial strain morphologically similar to psittacosis was recently isolated from several adults with lower respiratory tract illnesses. Antibodies to the strain, named TWAR in recognition of the 2 laboratories where it was identified, has been shown to occur in 25-45% of adults. Prevalence of this antibody appears to increase precipitously with the onset of puberty and to stabilize (but not decrease) around age 30. During a 30 month study, 386 students with acute respiratory illness were evaluated for the presence of TWAR so that its importance as a respiratory pathogen could be determined.

Patients were students seen at the University of Washington Student Health Centre. Three throat swab specimens were obtained from each patient, and culture for chlamydial strains

was performed both in the yolk sac of embryonated chicken eggs and in HeLa 229 cell cultures. Staining was performed in the traditional manner and using TWAR monoclonal antibodies. Serology was performed using both chlamydial group complement-fixation tests and the microimmunofluorescence test with IgM and IgG antibody.

Of the 386 patients, 139 had infections of the lower respiratory tract which included bronchitis and pneumonia. Fevers of undertermined origin were mostly influenza-like illnesses, whereas TWAR infections presented primarily as pneumonia and bronchitis.

The TWAR organism was isolated from 8/13 patients with serological evidence of recent TWAR infections; there were no isolates without serological evidence of infection. Of the 13 patients, 4 did not have markedly elevated titres of IgG antibody; instead, they maintained markedly elevated fractions to the IgM component. Chlamydia complement fixation antibodies were found in 6/8 patients with TWAR isolates.

Fluorescent staining with monoclonal antibody appeared to be the only successful way to detect the antigen in cell culture. Throat specimens, for example, were stained with TWAR monoclonal antibody, and 6/8 specimens stained were positive for antigen. No C. trachomatis or C. psittaci strains other than the TWAR strain were isolated from any of the 386 patients studied. Chronic antibody to TWAR was seen in 80/191 (42%) men and 68/195 (35%) women.

The other significant causes of respiratory illnesses in this patient group were influenza A or B and M. pneumoniae. Most patients with TWAR or mycoplasma had lower respiratory tract diseases, whereas those with influenza usually had nonspecific febrile illnesses or upper respiratory tract diseases.[1]

REFERENCES

1. N Engl.J.Med. (1986) 315: 161

METRONIDAZOLE-RESISTANT TRICHOMONAS VAGINALIS INFECTION

Metronidazole has been used for the treatmemt of vaginal trichomoniasis for over 20 years and although resistance to the drug has been reported[1,2], it is generally effective. A study of metronidazole efficacy reported in 1978 found a cure rate of 97% with a single 2g dose in patients who presented no problems with compliance or reinfection[3].

Recently a report of persistent vaginal trichomoniasis due to metronidazole resistant Trichomonas vaginalis was published[4]. The patient was an otherwise healthy 30 year old woman who presented with a 1 1/2 year history of recurrent vulvovaginitis characterised by green-white leukorrhoea and vulval pruritis. Many vaginal mucosal petechiae were seen on physical examination. Hanging-drop examination and culture of vaginal discharge revealed T.vaginalis.

Initial treatment with metronidazole-nystatin vaginal cream and metronidazole, 250 mg QID for 7 days was unsuccessful. Although there was symptomatic improvement, her infection recurred after about 1 month, confirmed by hanging-drop examination and culture. A repeat course of the same treatment was prescribed but the infection recurred 5 more times. On the first occasion, the initial treatment was prescribed (ie a total of 3 courses of metronidazole-nystatin cream and oral metronidazole). The next 2 times she was treated with sodium borate douches and sulphanilamide-aminacrine HCl-allantoin cream, and the last 2 with a 14 day course of oral metronidazole (dosage not stated) and a 20 day course of metronidazole-nystatin suppositories administered nightly.

All relapses were confirmed by hanging-drop examination and culture of vaginal discharge. The patient denied having sexual intercourse and claimed full compliance with prescribed therapy. In vitro culture and sensitivity tests indicated that the organism was moderately to highly resistant to metronidazole, with an aerobic minimum lethal concentration (MLC) of 400 µg/mL and an anaerobic MLC of 12-5 µg/mL.

The patient was then treated with oral metronidazole, 750 mg QID for 14 days, combined with a 14 day course of 500 mg metronidazole-nystatin vaginal suppositories, administered nightly. The treatment regimen was tolerated well, the only side affect noted being a metallic taste. Her haemoglobin level, leukocyte and platelet count, levels of serum aspartate aminotransferase, bilirubin, alkaline phosphatase and creatinine and of blood urea nitrogen (serum urea) and results of urinalysis continued to be within normal limits.

The patient improved symptomatically and was still well 1 year after the final treatment. Cultures of vaginal discharge made on 2 occasions after treatment were negative for T.vaginalis.

This report confirms the observations made by others that, while metronidazole is usually effective in the treatment of vaginal trichomoniasis, recalcitrant cases do occur. In the present case, resistance to metronidazole was relative rather than absolute and in such infections, the major limiting factor in successful treatment with metronidazole appears to be drug toxicity.

REFERENCES

1. Can Med Assoc J (1962) 86:665
2. Br J. Vener Dis (1973) 49:531-35
3. Obstet Gynecol (1978) 56:508-10
4. Can. Med. Ass J (1986) 134:1373-74

ERRATUM:

The CDI issue of 7 October 1986 should have read Bulletin Number 86/20 in lieu of CDI 86/19 as appearing on the front page of that issue.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 30/9/86 to 12/10/86 Bulletin 86/21
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	2		12		11	1	7		33
0101 ADENOVIRUS TYPE 1		1			9	3		1	14
0102 ADENOVIRUS TYPE 2	3				13			3	19
0103 ADENOVIRUS TYPE 3				2	3	2		3	10
0105 ADENOVIRUS TYPE 5	1	1			3	4		1	10
0107 ADENOVIRUS TYPE 7	1								1
0108 ADENOVIRUS TYPE 8				1					1
0199 ADENOVIRUS TYPING PENDING		1			4				5
0201 INFLUENZA A VIRUS	4	1	6	3	2				16
0203 INFLUENZA B VIRUS	3	1							4
0301 PARAINFLUENZA VIRUS TYPE 1						1		3	4
0302 PARAINFLUENZA VIRUS TYPE 2						2			2
0303 PARAINFLUENZA VIRUS TYPE 3	2	1		2	5	7	6	4	27
0399 PARAINFLUENZA VIRUS TYPING PENDING							1		1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	7	1	19	13	55	24	27	10	156
0500 RHINOVIRUS (ALL TYPES)	1		1	2	26	8			38
0600 MYCOPLASMA PNEUMONIAE	11		7	6				7	31
0700 ORNITHOSIS-PSITTACOSIS				5				2	7
0809 COXSACKIEVIRUS A9								1	1
0816 COXSACKIEVIRUS A16				1		15			16
0901 COXSACKIEVIRUS B1								1	1
0902 COXSACKIEVIRUS B2				1					1
1003 ECHOVIRUS TYPE 3				2					2
1005 ECHOVIRUS TYPE 5				1					1
1011 ECHOVIRUS TYPE 11	2				7			3	12
1012 ECHOVIRUS TYPE 12						2			2
1014 ECHOVIRUS TYPE 14					1	2			3
1018 ECHOVIRUS TYPE 18								1	1
1020 ECHOVIRUS TYPE 20				2					2
1021 ECHOVIRUS TYPE 21					1				1
1022 ECHOVIRUS TYPE 22				1					1
1100 POLIOVIRUS NOT TYPED			1		15				16
1101 POLIOVIRUS TYPE 1						1			1
1102 POLIOVIRUS TYPE 2	1					1			2
1103 POLIOVIRUS TYPE 3	1					1			2
1200 MUMPS VIRUS	1			2				1	4
1300 HERPES VIRUS GROUP-NOT TYPED	12			2				3	17
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1				1	2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	2		15	3				9	29
1303 VARICELLA-ZOSTER VIRUS	9		2	2	1	1	1	4	20
1306 HERPES SIMPLEX TYPE 1	21			37	1	15	24	11	109
1307 HERPES SIMPLEX TYPE 2	93			68		23	88	23	295
1399 HERPES VIRUS TYPING PENDING			2		8	1			11
1401 COXIELLA BURNETI	15			2		2		1	20
1502 PICORNA VIRUS-NOT TYPED	7		10				15	1	33
1521 MEASLES VIRUS		1		2	5				8
1522 RUBELLA VIRUS	1			4		2	4	3	14
1532 HEPATITIS B ANTIGEN	79	1	15	23	1	16	17	13	165
1535 HEPATITIS A ANTIBODY	9	2		13		7		10	41
1541 CHLAMYDIA A - C TRACHOMATIS	25	1	18	91		29	16	42	222
1556 CMV - CYTOMEGALOVIRUS	8	1	5	38	7	4	14	10	87
1564 ROTAVIRUS	24	8	13	4	35	13			97
1571 ENTEROVIRUS TYPE 71 (BRCR)	2	3		2	20	6			33
1599 ENTEROVIRUS TYPING PENDING		1	2		7				10
9992 ROSS RIVER VIRUS			3	1			7		11
9994 SMALL VIRUS (LIKE) PARTICLE		1		1					2
9998 ARBO. GROUP B.				2					2
Total	347	26	131	340	240	193	227	172	1,676

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 30/9/86 to 12/10/86
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	Respir-atory	Enceph-alitis	Mening-itis	Para-lysis	CNS other unspec	GI	Hepa-tic	CVS	Urin-ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....		10		1			2				
0102 ADENOVIRUS TYPE 2.....	1	13					4			1	
0103 ADENOVIRUS TYPE 3.....		8									
0105 ADENOVIRUS TYPE 5.....		5		1			3				
0107 ADENOVIRUS TYPE 7.....	1										
0201 INFLUENZA A VIRUS.....		11							1		
0203 INFLUENZA B VIRUS.....	1	1							1		
0301 PARAINFLUENZA VIRUS TYPE 1....		3									
0302 PARAINFLUENZA VIRUS TYPE 2....		2									
0303 PARAINFLUENZA VIRUS TYPE 3....		25									1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	6	144					1				1
0500 RHINOVIRUS (ALL TYPES).....		34									
0600 MYCOPLASMA PNEUMONIAE.....	11	18									1
0700 ORNITHOSIS-PSITTACOSIS.....	2	3							1		
0809 COXSACKIEVIRUS A9.....											1
0816 COXSACKIEVIRUS A16.....	1	1									13
0901 COXSACKIEVIRUS B1.....											1
1003 ECHOVIRUS TYPE 3.....											2
1005 ECHOVIRUS TYPE 5.....				1							
1011 ECHOVIRUS TYPE 11.....				7		1	4				
1012 ECHOVIRUS TYPE 12.....		1					1				
1014 ECHOVIRUS TYPE 14.....		1					2				
1018 ECHOVIRUS TYPE 18.....											1
1020 ECHOVIRUS TYPE 20.....				1							
1022 ECHOVIRUS TYPE 22.....											1
1100 POLIOVIRUS NOT TYPED.....							1				
1102 POLIOVIRUS TYPE 2.....	1	1									
1103 POLIOVIRUS TYPE 3.....	1			1							
1200 MUMPS VIRUS.....				1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED		2									
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	6	7						2		1	
1303 VARICELLA-ZOSTER VIRUS.....	2					2		1			1
1306 HERPES SIMPLEX TYPE 1.....	5	6								1	4
1307 HERPES SIMPLEX TYPE 2.....	8	1								1	43
1401 COXIELLA BURNETI.....	6	2				1		1			
1502 PICORNA VIRUS-NOT TYPED.....							1				
1521 MEASLES VIRUS.....	2										6
1522 RUBELLA VIRUS.....	4										6
1532 HEPATITIS B ANTIGEN.....	61							95			
1535 HEPATITIS A ANTIBODY.....	10							25			
1541 CHLAMYDIA A - C.TRACHOMATIS...	16	1					3				7
1556 CMV - CYTOMEGALOVIRUS.....	9	17						1		5	2
1564 ROTAVIRUS.....							94				
1571 ENTEROVIRUS TYPE 71 (BRCR)....	1	13	1	8			1		1		5
9992 ROSS RIVER VIRUS.....	1										3
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
9998 ARBO. GROUP B.			1								
Total.....	156	330	2	21		4	119	125	4	9	153

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 30/9/86 to 12/10/86
Viral Identifications by Clinical Information Table 2.
Code 10 -Eye; 59 -Genital; 39 -Endo/sal gland;
38 -RES; 29 -Muscle/joint; 69 -Congenital; P8 -PUO;
G8 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....										1
0103 ADENOVIRUS TYPE 3.....	3							1		
0105 ADENOVIRUS TYPE 5.....	1									
0108 ADENOVIRUS TYPE 8.....	1									
0201 INFLUENZA A VIRUS.....									4	
0203 INFLUENZA B VIRUS.....									1	
0301 PARAINFLUENZA VIRUS TYPE 1....					1		1			
0303 PARAINFLUENZA VIRUS TYPE 3....								2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								3	1	2
0500 RHINOVIRUS (ALL TYPES).....							1	1		2
0600 MYCOPLASMA PNEUMONIAE.....	1						1		1	
0700 ORNITHOSIS-PSITTACOSIS.....							1			
0816 COXSACKIEVIRUS A16.....									1	
0902 COXSACKIEVIRUS B2.....								1		
1011 ECHOVIRUS TYPE 11.....							1			
1020 ECHOVIRUS TYPE 20.....								1		
1021 ECHOVIRUS TYPE 21.....					1					
1101 POLIOVIRUS TYPE 1.....										1
1200 MUMPS VIRUS.....			3							
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			6	2			6	2	4	
1303 VARICELLA-ZOSTER VIRUS.....					1				2	
1306 HERPES SIMPLEX TYPE 1.....	5	42	1					1	3	
1307 HERPES SIMPLEX TYPE 2.....		243								
1401 COXIELLA BURNETI.....							2	3	6	
1522 RUBELLA VIRUS.....					2				4	
1532 HEPATITIS B ANTIGEN.....									9	
1535 HEPATITIS A ANTIBODY.....									6	
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	189							3	
1556 CMV - CYTOMEGALOVIRUS.....		8		1		4	5	8	31	2
1564 ROTAVIRUS.....		1						1		1
1571 ENTEROVIRUS TYPE 71 (BRCR)....							1	1	1	
9992 ROSS RIVER VIRUS.....			1		7			4		
9998 ARBO. GROUP B.									1	
Total.....	14	483	11	3	12	4	19	29	78	9

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Period 4 - 22 March 1986 to 18 April 1986

Bulletin 86/21....

Disease	N.S.W.	VIC.	QLD.	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	Cumulative Total to Date for Year
Amoebiasis		2	2	1				2	7	16
Ankylostomiasis				1			NN		1	* 4
Anthrax									-	-
Arbovirus infection	15	17	161		7				200	506
Brucellosis			1						1	7
Campylobacter infections	65		NN	72	5	NN	7	NN	149	* 795
Chancroid	3			NN					3	3
Cholera									-	-
Congenital rubella syndrome			NN			NN		NN	-	-
Diphtheria							2		2	8
Donovanosis				NN	4		4		8	24
Giardiasis	35		NN	72	11	NN	NN	NN	118	* 385
Genital herpes	72		4	23	NN	NN	1	NN	100	413
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	-	-
Gonorrhoea	100		10	56	95	2	63	1	332	1,458
Hepatitis A (infectious)	14	14	12	46	53				139	586
Hepatitis B (serum)	49	30	8	10	32	1	3	4	137	* 576
Hepatitis – unspecified	14		2		NN	NN	1		17	55
Hydatid disease	1							1	2	* 4
Lassa fever			NN			NN		NN	-	-
Legionnaires disease	2		NN	1		NN		NN	3	32
Leprosy									-	4
Leptospirosis	5	2	11	1					19	65
Lymphogranuloma venereum	1			NN	NN	NN		NN	1	2
Marburg disease			NN			NN		NN	-	-
Malaria	17	10	6	2	10		2	3	50	230
									-	-
Meningococcal infections		1		1	1	NN			3	12

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	271		NN	73	NN	NN	NN	NN	344	1,491
Ornithosis				3				1	4	15
Pertussis (whooping cough)	7	3	NN	11	1	NN		NN	22	344
Plague									-	-
Poliomyelitis									-	-
Q. fever	7		18	4					29	* 80
Rabies				NN		NN		NN	-	-
Salmonella infections	94	23	49	28	22	14	33	8	271	* 999
Shigella infections	21	3	27	9	4		24		88	* 309
Smallpox									-	-
Syphilis	37		44	23	15		109	2	230	643
Tetanus									-	3
Trachoma		NN				NN	NN		-	5
Tuberculosis (all forms)	23	37	14	1	11			3	89	* 276
Typhoid fever		1							1	14
Typhus (all forms)									-	2
Vibrio parahaemolyticus infections	1		NN			NN		NN	1	* 4
Yellow fever									-	-
Yersinia infections	7		NN	3		NN		NN	10	32

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.

Ankylostomiasis	+1	South Australia	Ornithosis	+1	Western Australia
Campylobacter infec.	+1	South Australia	Q fever	-2	New South Wales
Campylobacter infec.	-11	New South Wales	Salmonella infec.	+3	South Australia
Giardiasis	+5	South Australia	Salmonella infec.	-3	New South Wales
Hepatitis B (serum)	+1	South Australia	Shigella infec.	+1	South Australia
Hydatid disease	+1	South Australia	Shigella infec.	+1	New South Wales
Non-spec. urethritis	-1	Western Australia	Tuberculosis	+2	New South Wales