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Contents:

VIRUS REPORTING SCHEME: A total of 1,164 reports were processed for this period.

Six cases of Q fever were reported. Details of occupational exposure were not available for two of these patients. The other patients were males from Queensland: a 20 year old meatworker from Maryborough, a 36 year old butcher from Brisbane, a 36 year old meatworker from Wondai and a draftsman from Maroochy Shire. None of these patients was involved in the Q fever vaccine field trial conducted in South Australia.

In the current reporting period there have been 6 reports of echovirus type 7 compared with an average of 3 for the past three periods. The virus was isolated from:

- the bronchial washings of a 3 month old male with myocarditis and a 5 month old microcephalic male with severe cough;
- the nasal aspirate of a 9 year old male with encephalitis;
- the faeces of a 2 year old female with mild fever and a 2 month old female who had a more than one month history of rash in the nappy area;
- the throat swab of a 26 year old male with pyrexia of unknown origin and a history of weight loss and sore throat for the preceeding 3 weeks.

Sixty cases of Ross River virus were reported: 1 from South Australia, 5 from Western Australia, 10 from Queensland and 44 from Victoria.

One case of dengue fever (serotype 3) was reported in a 24 year old male presenting with gastrointestinal symptoms and a mild fever. The patient had become infected with the virus in India.

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Figures given may be subject to revision.

AIDS SURVEILANCE IN CANADA

(based on CDWR, Vol 12-2, 11 January 1986)

The following table outlines the distribution of adult AIDS cases reported in Canada for the period 1 January to 29 November 1984 and 1985.

	CUMULATIVE 1/1/84 - 29/11/84	1/1/85 - 29/11/85
Newfoundland	Nil	Nil
Prince Edward Island	Nil	Nil
Nova Scotia	1	4
New Brunswick	Nil	1
Quebec	34	56
Ontario	42	99
Manitoba	Nil	1
Saskatchewan	1	Nil
Alberta	8	8
British Columbia	21	41
Yukon	Nil	Nil
North West Territories	Nil	Nil
CANADA	107	210

In addition to the above adult cases, 3 and 11 paediatric cases respectively have been reported in the prescribed periods.

AIDS IN THAILAND

Based on Asian Pacific J Allerg. and Immun. 3:195-199

Significant HTLV-III related infections, AIDS and AIDS related complex have recently been diagnosed in 3 patients at the Chulalongkorn Hospital Medical School in Thailand. One case was a 30 year old American who had been in Thailand for two years. The other two were Thais, one a 27 year old male and the other a 26 year old female, one of three mistresses of the second patient.

The American was a homosexual who had moved to Thailand 2 years earlier with his male partner. AIDS was diagnosed on the basis of poor T-cell response to PHA *in vitro*, recurrent dermatomycoses, and *Pneumocystis carinii* pneumonia confirmed by lung biopsy.

The Thai male had been bisexual for the last 4-5 years; the only male homosexual with whom he had had contact was a 50 year old German who visited Thailand regularly. AIDS was diagnosed in this patient on the basis of bilateral cervical lymphodadenopathy disseminated cryptococcal infection, a depressed T4/T8 ratio of 0.39 and a suppressed T-cell response to PHA *in vitro*.

The Thai female had engaged in regular vaginal intercourse with the second patient. She denied ever having had anal intercourse or intercourse with other men. AIDS related complex was diagnosed on the basis of generalised lymphoadenopathy and a progressive reduction in T4/T8 ratio (to below 0.61).

Sera from all 3 patients, tested subsequently at the Centres for Disease Control in Atlanta, Georgia, (CDC) were HTLV-III positive.

The sexual partner of the first patient presented with P.carinii pneumonia about 5 months after his partner.

The male sexual contact of the second patient was asymptomatic, HTLV-III antibody negative and had normal T-cell numbers.

The third case probably reflects heterosexual transmission of HTLV-III related syndrome. Male to female heterosexual transmission of this virus has been well documented.(1) By contrast female to male heterosexual transmission of the virus has been less well documented.

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USE OF LIVE VACCINES IN PERSONS WITH HTLV-III INFECTION

(based on CDWR Vol 12-2, 11 January 1986)

The use of live vaccines in individuals with impaired immune mechanisms is contraindicated. Since patients with symptomatic HTLV-III infection (AIDS and AIDS-related complex) have abnormalities of their immune system, they should not be given live vaccines.

Children born to mothers infected with HTLV-III are at appreciable risk of developing symptomatic infections due to this virus. Live vaccines should be given to infants born of infected women only when appropriate laboratory investigations have excluded HTLV-III infection.

Adverse events have not been reported after administration of live vaccines to individuals with asymptomatic HTLV-III infection. Nevertheless, infected persons are at theoretical risk of a severe reaction since some may have a clinically silent immune defect. Accordingly, certain live vaccines including BCG, rubella, and mumps vaccine should be avoided in such individuals because of the possibility of increased risk of complications. Inactivated poliomyelitis vaccine should be used in place of live oral vaccine for infected persons and their household contacts. Administration of measles vaccine should be deferred in individuals infected with HTLV-III until further information on its safety in these persons is available. In certain high risk situations, however, such as during an outbreak, use of measles vaccine should be considered. Travel by infected persons to areas where yellow fever vaccination is mandatory should be discouraged. If such travel is unavoidable, theoretical risks of vaccination should be discussed with the individual before vaccine is given.

ACYCLOVIR AND HERPES SIMPLEX INFECTIONS

Herpes simplex virus (HSV) infection is characterised by localised primary lesions, latency and a tendency to recurrence. HSV types 1 and 2 have different characteristics in tissue culture, embryonated eggs and experimental animals, and can be differentiated serologically. Primary infection with HSV type 1 is often asymptomatic but may be marked by gingivostomatitis, keratoconjunctivitis, generalised cutaneous eruption, meningoencephalitis or a generalised infection in neonates.[1] Genital infection is usually caused by HSV type 2 but 5-30% of cases may be due to HSV type 1. Reactivation of HSV type 1 infection commonly results in herpes labialis.

Topical therapy with 5% acyclovir cream shortens the duration of lesions in patients with recurrent herpes labialis[2,3] and herpes genitalis[4] but does not appear to provide adequate protection against lesion development when used prophylactically[5]. In contrast, prophylaxis with oral acyclovir has been shown to decrease the recurrence of lesions in patients with recurrent genital HSV infection[6,7,8]. In addition, oral acyclovir treatment of primary genital HSV infection is clinically effective but may not prevent virus latency or associated recurrent disease[9].

In a recent double-blind, placebo-controlled, cross-over trial[10] 11 patients suffering eight or more episodes of recurrent non-genital HSV infection per annum, only 2 patients experienced a recurrence during treatment with oral acyclovir (200mg qid) for up to twelve weeks, compared with 9 during placebo treatment ($p=0.016$). Although lesion development was effectively suppressed in nine of the patients whilst taking acyclovir, the development of prodromal symptoms, and occasionally erythema, was reported by five. There was no difference between acyclovir and placebo in the time to the next recurrence following completion of treatment. No patient reported any side effects of either placebo or acyclovir therapy.

Since 1977 the National Institute of Allergy and Infectious Disease (NIAID) Collaborative Antiviral Study Group has defined the effectiveness of vidarabine for treating herpes simplex encephalitis. With the introduction of acyclovir, the study group undertook a controlled, comparative evaluation of vidarabine versus acyclovir in biopsy-proven herpes encephalitis[11].

The group studied 208 patients suspected of herpes simplex-related encephalitis who underwent brain biopsy; herpes simplex was isolated from 69. A randomization schedule was used to determine who would receive each agent; 37 received vidarabine and 32 acyclovir, with therapy initiated at the time of brain biopsy. Vidarabine was given intravenously at a dosage of 15 mg/kg of body weight over 12-hour periods daily for 10 days. Acyclovir was administered at 30 mg/kg divided into 3 dosages daily for the same time period. At presentation of each patient, clinical assessment focused on the time, progression, and severity of the neurological dysfunction. The Glasgow Coma Scale was used to assess level of coma with eye, motor, and verbal scores being granted (higher scores for higher levels of consciousness). Morbidity was defined in terms of the level of patient impairment.

The demographics of the 2 treatment groups were similar, although more patients < 30 years old were assigned to receive acyclovir. Duration of the disease, administration of mannitol, corticosteroids, or both, and presence of concomitant bacterial infections was comparable in the 2 groups. Mortality at 1 and 6 months and overall for the vidarabine and acyclovir groups was 43% vs 13%, 54% vs 19%, and 54% vs 28%, respectively. Patients < 30 years had only a 6% mortality using acyclovir, compared to a 45% mortality with vidarabine. Levels of consciousness and coma scores of the recipients at time of treatment were also predictors of effectiveness, with the significant differences favouring acyclovir. The lighter the coma and the higher the level of consciousness, the better the prognosis. All patients receiving acyclovir within 4 days of onset of fever, headache, and focal neurologic findings survived. A 35% mortality rate was associated with those patients treated later than the fourth day.

Six months after therapy, 65% of the vidarabine recipients were either dead or had severe sequelae, 22% had moderate sequelae, and 13% had either no impairment or minor abnormalities. The acyclovir recipients fared better - 53% were dead or severely impaired, 9% were moderately impaired, and 38% had either no residual deficits or minor abnormalities. Laboratory abnormalities and clinical sequelae or treatment again favoured the acyclovir recipients. In both groups, the toxicity of drug therapy and laboratory abnormalities were minimal.

Although early recognition followed by early treatment with acyclovir improves the prognosis for most patients with herpes simplex encephalitis, further treatment options must be examined.[12] Alternatives may be required because of the emergence of resistant strains and the failure of acyclovir therapy in a number of patients. The use of vidarabine and acyclovir or acyclovir in combination with an immune-enhancing agent should be investigated.

The liability in antiviral applications, as with other antimicrobials, is the potential emergence of resistant strains. Although the in vitro passage of HSV in the presence of acyclovir can be demonstrated to select for resistant strains with alteration in thymidine kinase (TK) expression, it has been difficult to develop conditions which select out resistance by altering the viral DNA polymerase. Those strains which are TK-deficient have had less pathogenicity and a reduced ability to produce latency. DNA polymerase variants, on the other hand, have maintained their virulence.

A recent study[13] reported on the properties of HSV isolates obtained from 3 immunocompromised patients treated with acyclovir (ACV), with special emphasis on the mixed pattern of drug resistance associated with different combinations of low TK expression and decreased TK affinity for both thymidine and the nucleoside analogs. Because a number of other nucleoside analogs have been proposed for therapy against HSV, they were included in this study. The study also included phosphonoformic acid (PFA), the only drug in the group acting on the DNA polymerase of the virus.

The ACV-resistant strain from patient 1 was cross-resistant to dihydroxypropoxymethylguanine and bromovinyldeoxyuridine but still sensitive to the 3 fluoro-substituted pyrimidines

tested. The TK from the resistant strain showed a 50-fold reduction in affinity for thymidine, although the total enzyme activity compared to the sensitive strain did not change. The isolate from patient 2 was resistant to all of the analogs in which TK dependency was a criteria. TK activity in this isolate was about 1% of the patient's pretherapy isolate. Of interest, a later isolate from this patient in a subsequent recurrence regained its initial sensitivity to all drugs tested. The third patient developed a mixed resistance which did not fit into either of the first 2 patterns. In this case, there was reduced affinity and reduced presence of the TK, but neither was as severe as the changes in affinity in patient 1's isolate or the change in the level in patient 2's isolate.

None of the isolates demonstrated any change in activity to the PFA. Compared to pretreatment isolates, there was a 30-fold decrease in neurovirulence in the first 2 resistant strains which emerged and no change in the last mixed pattern strain.

TK enzyme production, and particularly its affinity, appears to be the primary mechanism of resistance. Within the same patient, it is apparent that isolates may rapidly adapt, and they have the ability to readapt in future recurrences of the infection. Changes in viral DNA polymerase should be continuously sought because this type of factor may have greater import in the clinical course of disease. As with bacteriology, the epidemiology of viral infections must be closely monitored to define the use or abuse of drugs which can affect these illnesses. Inhibitory and virucidal levels of antiviral drugs will become as common-place as they are in treating bacterial infections, permitting more accurate and appropriate treatment.(14)

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HUMAN SALMONELLOSIS SURVEILLANCE

(Contributed by J. Powling, J. Taplin and L. Scott, Microbiological Diagnostic Unit (MDU), University of Melbourne)

A total of 1 279 Salmonella (92 serottypes), 219 Shigella and 515 campylobacter isolates from human cases were reported to the National Salmonella Surveillance Scheme during April-June 1985.

Salmonella typhi:

- S.typhi E1 was isolated from a 23 year old male who recently arrived from India.
- S.typhi M1 was isolated from a 40 year old male whose history was not available.
- S.typhi 38 was isolated from a 8 year old male migrant from El Salvador
- S.typi untypable was isolated from a 7 year old male who recently arrived from Indonesia.

Salmonella paratyphi:

- S.paratyphi A
  - phage type 6 were recovered from a 33 year old female who recently arrived from Iran and also from a 28 year old male who had just returned from India.
  - phage type untypable were isolated from a 33 year old male on his return from India and from a 20 year old male whose history was unavailable.

- S.paratyphi B
  - type 1 was isolated from a 18 month old female
  - type Taunton was recovered from the femur biopsy of a 51 year old male who had suffered from recurrent osteomyelitis for the past 25 years.

- S.paratyphi C
  - Vi negative strain was isolated from a 28 year old female social trainer at a centre for the handicapped. The patient had had a previous shigella infection.

OTHER SALMONELLA INFECTIONS

A. ISOLATIONS FROM URINE:- comprised the following serotypes:
- S.anatum was isolated from a 16 year old female
- S.birkenhead was isolated from a 79 year old female
- S.bovismorbificans was isolated from a 75 year old male
- S.havana was isolated from a 79 year old female
- S.kottbus was isolated from a 13 year old female
- S.ohio var 14 + was isolated from a 78 year old female
- S.ohio was isolated from an 88 year old female
- J.virchow was isolated from a female of unknown age.

B. ISOLATIONS FROM BLOOD: Cases of repticaemia involved the following serotypes:
- S.enteritidis was isolated from a 2 year old female with bowel obstruction.
- S.bovismorbificans was isolated from a 17 year old male with acute lymphatic leukaemia and who subsequently died.
- S.chester was isolated from a 13 year old male with fever after returning from a trip to Bali and Singapore.
- S.stanley was isolated from a 22 year old female with pyrexia of unknown origin and rigors after returning from Bali.

. S.virchow was isolated from two 9 year old males.
. S.waycross was isolated from a baby girl of unknown age.
. S.typhimurium
  - phage type 12a was isolated from a 34 year old male with diarrhoea and fever
  - phage type 135 was isolated from a febrile 37 year old male renal transplant patient and from a 69 year old male with fever, rigors, dysuria and abdominal pain.
  - phage type 156 was isolated from a 1 year old male whose hostory was not available
  - phage type 175 was isolated from a 16 year old male bone marrow transplant patient who had returned from Malaysia 2 days before surgery.

Other isolations of interest included the following serotypes:
. S.muenchen was isolated from the peritoneal fluid of a 8 year old female who undervent laparotomy for possible appendicitis.
. S.potsdam was isolated from a high vaginal swab for post partum infection in a 29 year old patient.
. S.virchow was isolated from the pus exudate of an infected ovarian cyst in a 34 year old patient
. S.typhimurium 202 was isolated from the pus of an abscess due to a perforated appendix in a male of unknown age.

New serotypes reported for the first time in the Surveillance scheme included S.duesseldorf, S.molade, S.paratyphi C (Vi negative), S.rissen, S.staouli, S.tananarive and S.typhimurium phage types 53 and 81.

ENTERIC PATHOGENS ACQUIRED OUTSIDE AUSTRALIA

In addition to the cases of enteric fever detailed above, the following serotypes were isolated from travellers returning from overseas visits and/or from migrants screened for enteric pathogens upon arrival in Australia.

Salmonella species:
. S.agona, S.blockley, S.hadar, S.havana, S.hvittingfoss, S.saintpaul and S.tananarive were acquired in South East Asia.
. S.braenderup, S.indiana, and S.emek were acquired in India
. S.alachua and S.bareilly were acquired in Sri Lanka
. S.cerro, S.chester, S.weltevreden and S.typhimurium 81 were acquired in Singapore.
. S.duesseldorf was acquired in Thailand
. S.enteritidis and S.virchow were acquired in Fiji
. S.heidelberg was acquired in the Philippines
. S.litchfield, S.molade, S.montevideo, and S.panama were acquired in Indonesia
. S.ohio were acquired in Hong Kong and India
. S.oranienburg were acquired in Iran
. S.typhimurium phage type 175 was acquired in Malaysia.

Clusters of enteric infections

S.typhimurium 64 were isolated from 29 patients, the majority of whom were children in the metropolitan area of Perth. Subsequent investigations have not revealed the source of infection. However, S.typhimurium 64 was implicated in a large scale outbreak of infection among sheep (see CDI 86/4) and were subsequently isolated from a variety of meats, food handlers employed by a smallgoods producer, and abattoir effluents.

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S.aberdeen	3				3				
S.abony	2				1		1		
S.adelaide	13		4		1	1	4		3
S.agona	10		1	8	1				
S.alachua	2						2		
S.anatum	18		1		10	2	2		3
S.anatum var 15+	4		1		3				
S.arizonae	7		1		5		1		
S.bahrenfeld	2		1				1		
S.ball	2								2
S.bareilly	5		2	3					
S.birkenhead	17	1	11	2	3				
S.blockley	12		2	5		2	3		
S.bonn	1					1			
S.bournemouth	3						3		
S.bovismorbificans	10		1	3	1		5		
S.bovismorbificans 07	2					1			1
S.bovismorbificans 21	1		1						
S.bovismorbificans 23	4					2	2		
S.bovismorbificans 24	1		1						
S.bovismorbificans 4	1		1						
S.bovismorbificans 7	6		1			4			1
S.braenderup	2		1	1					
S.bredeney	2		1						1
S.breukelen	1				1				
S.cerro	5						5		
S.charity	1					1			
S.chester	43		9	2	6		18		8
S.cholerae suis v kunz	1		1						
S.decatur	1						1		
S.derby	12		2	4	3		3		
S.duesseldorf	1			1					
S.eastbourne	2				1		1		
S.emek	2		1	1					
S.emmasted	1						1		
S.enteritidis	21		4		17				
S.fremantle subgenus II	1						1		
S.give	6					1	3		2
S.hadar	2						2		
S.havana	47		6	4	3	7	14		13
S.heidelberg	7			1	6				
S.hessarek	2						2		
S.hvittingfoss	6			2	2	1			1
S.indiana	1			1					
S.infantis	19		1	3	2	2	10		1
S.jangwani	3						3		
S.java	2		1				1		
S.java Battersea	1					1			
S.java Dundee	2		1		1				
S.java Untypable	12					1	2		9
S.kentucky	2		2						
S.kottbus	8		1		3	4			
S.krefeld	2						2		
S.lansing	6				5		1		
S.litchfield	14			2	5		3		4
S.livingstone	2		1			1			
S.mbandaka	2						2		
S.meleagridis	1			1					
S.mississippi	6		1				5		
S.molade	2						2		

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S.montevideo	6			3	2		1		
S.muenchen	33		8		10		10		5
S.muenster	1					1			
S.newport	14		12			2			
S.ohio var 14+	3	1	2						
S.ohio	13	8	1	1		2		1	
S.ohlstedt	2								2
S.onderstepoort	1								1
S.oranienburg	25		3	2	2	2	13	1	2
S.orientalis	2				2				
S.orion	8					3	4		1
S.orion var 15+	1		1						
S.panama	4		2			1	1		
S.paratyphi A6	2		1			1			
S.paratyphi untyp.	3		1			1	1		
S.paratyphi B1	1		1						
S.paratyphi B Taunton	1			1					
S.paratyphi C (vi neg)	1						1		
S.potsdam	27		2		14	4	5		2
S.reading	3				2		1		
S.rissen	1						1		
S.rubislaw	5						2		3
S.saintpaul	77		1	3	53	1	11		8
S.schwarzengrund	1		1						
S.senftenberg	10	1		2					7
S.singapore	23	3	9	3		2	4		2
S.stanley	12	1	1	3	1		6		
S.staoueli	1								1
S.tananarive	2			2					
S.tennessee	5						4		1
S.typhi*	16		8	5	3				
S.typhimurium*	469	6	148	76	37	86	88	13	15
S. 4,12:d:	7		4		3				
S.untypable 17:a:-	3								3
S.untypable 38:1v:-	1								1
S.untypable 4,5,12;-;1,2	1				1				
S.untypable 47:Z4Z23:-	1		1						
S.untypable 6.7:K:-	3								3
S.untypable 6.8:-:-	1			1					
S.untypable rough:-:-	2			1	1				
S.untypable rough:-:1,5	1			1					
S.urbana	5								5
S.virchow	75		9	4	56		2	1	3
S.wandsbek subgenus II	1			1					
S.wandsworth	2						2		
S.waycross	7		1		6				
S.welikade	14		3		6				5
S.weltevreden	4			1					3
S.zanzibar	1				1				
TOTAL	1279	20	283	152	285	138	263	16	122

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S.typi*									
S.typhi 38	1			1					
S.typhi A	3				3				
S.typhi E1	7		4	3					
S.typhi M1	1		1						
S.typhi O	1			1					
S.typhi T	1		1						
S.typhi untypable	2		2						
TOTAL	16		8	5	3				

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S.typhimurium*									
S.typhimurium	21	3	3			2	13		
S.typhimurium RDNC	26		9	4		3	8	2	
S.typhimurium untypable	28	1	13	6	1	5	1		1
phage type 1	1			1					
phage type 4	16		3	10		2	1		
phage type 5	26		9		1	11	1	1	3
phage type 6	4		1	2				1	
phage type 8	7			4	1	2			
phage type 9	25		1	9	10	3	2		
phage type 12	1				1				
phage type 12a	38		9	5	3	18	3		
phage type 13	1					1			
phage type 16	2		1			1			
phage type 21	3			1			2		
phage type 22	4		2		1		1		
phage type 23	1						1		
phage type 26	19	4	7		1	7			
phage type 27	5	1		1			3		
phage type 29	2						2		
phage type 30	1						1		
phage type 31	4			2	1		1		
phage type 32	1						1		
phage type 41	3		2		1				
phage type 44	6		2	2	2				
phage type 46	1		1						
phage type 52	2		1				1		
phage type 53	2		1				1		
phage type 55	1						1		
phage type 58	6					1	5		
phage type 64	35		5		1		29		
phage type 66	1								1
phage type 81	3			3					
phage type 90	5		1	2		2			
phage type 92	1		1						
phage type 101	18	6		1	10		1		
phage type 102	2		2						
phage type 108	9		4		2	2	1		

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
phage type 124	8		6		2				
phage type 138	87	1	41	13	4	16		6	6
phage type 136	1						1		
phage type 141	7		2				1	4	
phage type 145	2		2						
phage type 156	1								1
phage type 167	1		1						
phage type 168	1				1				
phage type 170	6		4	1	1				
phage type 175	1		1						
phage type 176	1			1					
phage type 179	8	1	4	2		1			
phage type 202	14		5	1	2	5			1
TOTAL	469	6	148	76	37	86	88	13	15

GONOCOCCAL SURVEILLANCE AUSTRALIA
JULY - SEPTEMBER 1985

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

During this period the AGSP recorded the lowest number of strains examined in a single quarter since it began reporting in 1981. This report provides details of penicillin sensitivities for the period July-September 1985 of 991 isolates of Neisseria gonorrhoea, examined by participating State and Territory Laboratories using standardised techniques and procedures(1).

The intrinsic resistance of gonococci describes the sum total of gonoccocal resistance to penicillin, effected by bacterial chromosome alterations. Strains of gonococci may be classified as sensitive, less sensitive or relatively resistant according to their in-vitro antibiotic sensitivity(2). Sensitive and less sensitive strains usually respond to standard doses of the penicillin group of antibiotics whereas treatment failures occur with infections caused by relatively resistant gonococci. These strains are still relatively uncommon in Australia, although the tendency towards increased intrinsic resistance reported this quarter, continued with a greater proportion of strains falling into the less sensitive category, particularly in Sydney and Melbourne.

A fourth category comprises those gonococci which have acquired additional chromosomal genetic material (plasmids) which codes for elaborate penicillinase production (PPNG). These strains are totally resistant to the penicillins.

The continued monitoring of PPNG infections in Sydney and Melbourne still indicates an on-going outbreak (Table). During this period 33 PPNG isolates have been reported for Sydney including 20 isolates recovered from patients with no history of travel indicating that these strains are now endemic in Sydney. The other 13 isolates recovered included 12 cases of infections acquired in South East Asia and the source was not identified in the remaining case.

During the same period, 22 PPNG were isolated in Melbourne, comprising 12 infections acquired overseas, 2 infections acquired locally and in the remaining 8 isolates the source of infection was not determined. No PPNG were reported from Adelaide, Canberra or Hobart, however in Darwin, Perth and Brisbane PPNG were noted in locally acquired infections.

Table: Penicillin sensitivity of isolates of N. gonorrhoeae July - September 1985

Centre	Percentage of isolates		
	Sensitive	Less Sensitive	PPNG
Brisbane	37.0 (29.5)*	51.9 (61.8)	5.1 (2.0)
Sydney	9.4 (15)	66.6 (56.9)	13.2 (13.0)
Melbourne	12.8 (26)	59.3 (44.8)	10.7 (6.4)
Adelaide	38.4 (47.9)	46.4 (44.7)	0 (1.1)
Perth	14.9 (32.5)	51.9 (42.7)	7.4 (8.2)

* Figures in parenthesis represent data for the corresponding period in 1984.

References

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

Q FEVER

Q fever is an acute rickettsial disease due to Coxiella burnetii which infects phagolysosomes of mononuclear phagocytes.(1) Human infection occurs from exposure to infected sheep, goats, cattle or their products. Infection can also occur following exposure to infected dust. Q fever is an important cause of morbidity in Australian meatworkers, particularly since abattoirs started to process feral goats for export.

Q fever has been reported from all continents. The incidence is greater than that reported because of limited clinical suspicion and the few laboratories testing for Coxiella burnetii. A few cases of imported Q fever have been reported from Scandinavia,(2) but the first human case of domestic Q fever in Sweden has only recently been documented.(3) The patient, a previously healthy 56 year old male, experienced

sudden onset of pyrexia, back pain and frontal headache. On admission, the patient's temperature was 40^{0}C, pulse rate 94/min and arterial blood pressure 90/60 min Hg. The axillary and cervical lymph nodes were enlarged. No hepatomegaly, splenomegaly or abnormality of the heart or lungs was observed. WBC (polymorphonuclear leucocytosis) count and ESR were elevated. Chest X ray revealed opacities of the lower parts of both lungs. After admission, liver function tests deteriorated and the patient failed to improve on combined therapy with penicillin and gentamicin or chloramphemicol. A diagnosis of Q fever was then considered; the patient was given doxycycline 200 mg daily p.o. and responded promptly. The patient's serum was checked locally with an ELISA test using pure phase I and phase II antigen against Coxiella burnetti. As the outcome of the test was positive, acute and convalescent sera were sent to the Rocky Mountain Laboratory, National Institute of Allergy and Infectious Diseases. Microimmunofluorescence (MIF) testing confirmed the diagnosis.

Infection by Coxiella burnetti may be asymptomatic or may cause fever, headache, malaise, pneumonitis and, rarely, chronic endocarditis, pericarditis and granulomatous hepatitis. In order to differentiate patients with hepatitis, pnenumonia and endocarditis, a recent study examined both traditional serology and the cellular immune status of these patients.(4) It was felt that the persistent infection in endocarditis patients could be related to a defect in cellular immunity. Cases of documented Q fever hepatitis, endocarditis, and inactive primary Q fever were studied; a control group consisted of patients without exposure to C. burnetii. A complement fixation titre of 1:128 was considered positive. A positive titre combined with positive histopathology confirmed the pneumonia and particularly the hepatitis syndrome.

Cellular immunology was established by testing lymphocytes enriched for helper cells against Q fever antigens as well as standard mitogen stimulation antigens such as PHA. Peripheral blood mononuclear cells were incubated in the presence of at least 3 Coxiella antigens derived from human isolates of Q fever. Cell proliferation was measured by the incorporation of [^{3}H] thymidine into the cellular DNA. Lymphocytes from asymptomatic patients who had convalesced from primary Q fever 2 months-2 years earlier displayed specific proliferation with counts of 5,600-12,300/minute. Lymphocytes from 4 patients with biopsy-documented hepatitis due to Q fever had much higher counts, demonstrating an even more exuberant response. Four patients who had endocarditis showed no proliferative response at all and were comparable to control patients who had never been exposed to C. burnetii. On the other hand, all 4 patients with endocarditis responded to PHA and other antigens in a normal fashion. Additional studies indicated that suppressor cells were partly responsible for this phenomenon but could not account for the majority of inactivity.

This study helps clarify why Q fever presents differently in different people. It is also a useful model for understanding the role of individual immunity in many diseases where cellular immunity plays a major role. The use of cellular immune assays is becoming a natural extension for testing and defining the

presence of specific infections, particularly where serology is either of little use or unpredicatable and where it is difficult to isolate the organism itself. Measuring in vitro cellular immune responses to specific antigens may not only help define the presence of an infection, but it may also aid in following the course and response to therapy of such infections. In addition, assaying the immune system may help define those individuals who are at risk for specific infections, either through inheritance or acquisition of other underlying conditions.(1)

The Institute of Medical and Veterinary Science (IMVS) is currently involved in a trial to examine vaccine prophylaxis of abbatoir-associated Q fever.(5) In the reported stages of the study, 924 nonimmune volunteers at two South Australian abattoirs were inoculated with one dose of a purified, formalin-inactivated, Coxiella burnetii (Henzerling strain) phase 1 vaccine. Some 56% of workers in one abattoir, and 64% in the other, seroconverted after vaccination. In the 18 months after vaccination, no Q fever occurred in fully vaccinated subjects, whereas there were 34 cases in 1349 unvaccinated workers. Transient local reactions were noted in most vaccinated subjects; only a few had mild general reactions. No cases of vaccine-enhanced disease were observed.

The investigators have concluded that the first 18 months to 2 years experience of Ormsbee-type inactivated Q fever vaccine showed that vaccination of seronegative and skin test negative individuals protects against natural infection, and does not produce severe local or systemic reactions, or long term induration at the site.

CDI Editorial Comment

The CDI reports identified cases of Q fever, the patient's history of occupational exposure and whether they are involved in the SA Q fever vaccine trial. It would therefore be appreciated if occupational exposure data and whether the patient was participating in the SA Q fever vaccine trial could be indicated on the virus report forms, if available.

REFERENCES

(1) Infectious Disease Alert (1986) 5:8:30-31

(2) Scand. J. Infect. Dis. (1981) 13:17-21

(3) Acta Med Scand (1985) 218:429-32

(4) J. Inf. Dis (1985) 152:1283

(5) Lancet (1984) ii:1411-1414

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 3/3/86 - 16/3/86 BULLETIN NUMBER 86/6

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	1		5		3	1	7	1	18
0102	ADENOVIRUS TYPE 2				1				1	2
0103	ADENOVIRUS TYPE 3								1	1
0104	ADENOVIRUS TYPE 4				1					1
0105	ADENOVIRUS TYPE 5						2			2
0106	ADENOVIRUS TYPE 6						1			1
0107	ADENOVIRUS TYPE 7	1								1
0108	ADENOVIRUS TYPE 8			3	2		1			6
0119	ADENOVIRUS TYPE 19								1	1
0137	ADENOVIRUS TYPE 37								2	2
0199	ADENOVIRUS TYPING PENDING					2	1			3
0201	INFLUENZA A VIRUS				1					1
0303	PARAINFLUENZA VIRUS TYPE 3				1	1	1	4		7
0400	RESPIRATORY SYNCYTIAL VIRUS (RS)			1		1				2
0500	RHINOVIRUS (ALL TYPES)				3	11	3	4	3	24
0600	MYCOPLASMA PNEUMONIAE	4	1				3	1	2	11
0700	ORNITHOSIS-PSITTACOSIS	2			1					3
0816	COXSACKIEVIRUS A16		1		1					2
0901	COXSACKIEVIRUS B1		1							1
0906	COXSACKIEVIRUS B6				1					1
1007	ECHOVIRUS TYPE 7				1				5	6
1011	ECHOVIRUS TYPE 11	1						1		2
1014	ECHOVIRUS TYPE 14			1						1
1021	ECHOVIRUS TYPE 21				1					1
1022	ECHOVIRUS TYPE 22						2	1		3
1100	POLIOVIRUS NOT TYPED			3						3
1101	POLIOVIRUS TYPE 1	1			1				1	3
1102	POLIOVIRUS TYPE 2	1								1
1104	POLIOVIRUS-VACCINAL STRAIN							2		2
1200	MUMPS VIRUS		1					1		2
1300	HERPES VIRUS GROUP-NOT TYPED	45			4				1	50
1301	HERPES SIMPLEX VIRUS NOT-TYPED		4							4
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	9	2		5				8	24
1303	VARICELLA-ZOSTER VIRUS	1		1	3		1	1	3	10
1306	HERPES SIMPLEX TYPE 1	27			28	13	19	43	11	141
1307	HERPES SIMPLEX TYPE 2	67			53		16	84	34	254
1399	HERPES VIRUS TYPING PENDING			1		4				5
1401	COXIELLA BURNETI	2					1	3		6
1502	PICORNA VIRUS-NOT TYPED	4		5				4	1	14
1521	MEASLES VIRUS			1		1		1		3
1522	RUBELLA VIRUS	1					3		7	11
1532	HEPATITIS B ANTIGEN	79	2	6	17	1	19	29	11	164
1535	HEPATITIS A ANTIBODY	7	1	2	3	1	15	3	19	51
1541	CHLAMYDIA A - C TRACHOMATIS	42		5			45	46	52	190
1556	CMV - CYTOMEGALOVIRUS	2	3			4	10	2	8	29
1564	ROTAVIRUS	4		5		2	3		2	16
1599	ENTEROVIRUS TYPING PENDING		1	8		7				16
9992	ROSS RIVER VIRUS				44		1	10	5	60
9994	SMALL VIRUS (LIKE) PARTICLE	1								1
9995	DENGUE							1		1
Total		302	17	47	172	51	148	248	179	1,164

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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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
0102 ADENOVIRUS TYPE 2.....							1				
0103 ADENOVIRUS TYPE 3.....		1									
0105 ADENOVIRUS TYPE 5.....	1										
0106 ADENOVIRUS TYPE 6.....		1									
0108 ADENOVIRUS TYPE 8.....	1										
0201 INFLUENZA A VIRUS.....		1									
0303 PARAINFLUENZA VIRUS TYPE 3....		5									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		2									
0500 RHINOVIRUS (ALL TYPES).....		7					1				
0600 MYCOPLASMA PNEUMONIAE.....	1	10									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0816 COXSACKIEVIRUS A16.....				1							1
0901 COXSACKIEVIRUS B1.....		1					1				
0906 COXSACKIEVIRUS B6.....				1							
1007 ECHOVIRUS TYPE 7.....		1	1						1		1
1011 ECHOVIRUS TYPE 11.....	1					1					
1014 ECHOVIRUS TYPE 14.....				1							
1022 ECHOVIRUS TYPE 22.....							2	1			
1100 POLIOVIRUS NOT TYPED.....							1				
1101 POLIOVIRUS TYPE 1.....		1									
1102 POLIOVIRUS TYPE 2.....							1				
1104 POLIOVIRUS-VACCINAL STRAIN....							2				
1200 MUMPS VIRUS.....					1	1					
1300 HERPES VIRUS GROUP-NOT TYPED..	1										
1301 HERPES SIMPLEX VIRUS NOT-TYPED											3
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	4	4			1			3			
1303 VARICELLA-ZOSTER VIRUS.....	1										9
1306 HERPES SIMPLEX TYPE 1.....	16	9								1	64
1307 HERPES SIMPLEX TYPE 2.....	28	2									56
1401 COXIELLA BURNETI.....	1	1						1			
1502 PICORNA VIRUS-NOT TYPED.....		2		1			6				
1521 MEASLES VIRUS.....	1					1					1
1522 RUBELLA VIRUS.....										1	9
1532 HEPATITIS B ANTIGEN.....	47							100			
1535 HEPATITIS A ANTIBODY.....	13							27			
1556 CMV - CYTOMEGALOVIRUS.....	8	8				1		6		2	1
1564 ROTAVIRUS.....							16				
9992 ROSS RIVER VIRUS.....	10										11
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
9995 DENGUE.....							1				
Total.....	134	57	1	4	2	4	33	138	1	4	156

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

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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	Con-genital	PUO	Fever/malaise	Other	SIDS
0102 ADENOVIRUS TYPE 2.....								1		
0103 ADENOVIRUS TYPE 3.....										1
0104 ADENOVIRUS TYPE 4.....	1									
0105 ADENOVIRUS TYPE 5.....								1		
0107 ADENOVIRUS TYPE 7.....		1								
0108 ADENOVIRUS TYPE 8.....	5									
0119 ADENOVIRUS TYPE 19.....		1								
0137 ADENOVIRUS TYPE 37.....	1	1								
0303 PARAINFLUENZA VIRUS TYPE 3....								2		
0600 MYCOPLASMA PNEUMONIAE.....								1		
0700 ORNITHOSIS-PSITTACOSIS.....			1					1		
1007 ECHOVIRUS TYPE 7.....								1		
1021 ECHOVIRUS TYPE 21.....								1		
1101 POLIOVIRUS TYPE 1.....									1	
1301 HERPES SIMPLEX VIRUS NOT-TYPED				1						
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			9	2				1		
1303 VARICELLA-ZOSTER VIRUS.....								1		
1306 HERPES SIMPLEX TYPE 1.....	3	46						1	2	
1307 HERPES SIMPLEX TYPE 2.....		171								
1401 COXIELLA BURNETI.....					1			1	4	
1502 PICORNA VIRUS-NOT TYPED.....					1				1	
1521 MEASLES VIRUS.....									1	
1522 RUBELLA VIRUS.....					3				1	1
1532 HEPATITIS B ANTIGEN.....										17
1535 HEPATITIS A ANTIBODY.....										11
1541 CHLAMYDIA A - C.TRACHOMATIS...		190								
1556 CMV - CYTOMEGALOVIRUS.....				1		4		1	2	
9992 ROSS RIVER VIRUS.....				1	48		1	3		
9995 DENGUE.....								1		
Total.....	10	410	10	5	53	4	7	22	32	1