ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 86/7
Issue date: 7 April 1986

Contents:

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VIRUS REPORTING SCHEME: A total of 1,173 reports were processed for this period.

Six cases of Q fever were reported, 5 from New South Wales (males aged 42, 34 and 24; and females aged 46 and 16) and a 21 year old male from South Australia. No details of occupational exposure were available for these patients, however none was involved in the Q fever vaccine field trial conducted in South Australia.

In the current reporting period there have been 73 reports of cytomegalovirus compared with 29, 66, 39 and 38 for the four previous periods. The virus was isolated from:-

- the urine of three HTLV-III seropositive males aged 40, 39 and 33 respectively and one male AIDS patient of unknown age with a febrile illness;
- the leucocytes of a 74 year old female with polyneuritis;
- post-mortem tissue from ulcerative lesions of the ascending colon of a 33 year old male patient who died from AIDS.

Twenty two cases of Ross River virus were reported, 3 from Western Australia, 3 from New South Wales and 16 from Victoria.

ANNOUNCEMENT:

The NH & MRC Antibiotics Committee has prepared a document 'CONTROL OF CROSS-INFECTION WITH METHICILLIN (MULTIPLE ANTIBIOTIC) RESISTANT STAPHYLOCOCCUS AUREUS', providing guidelines for health care institutions for the control of cross-infection with MRSA. The document, approved by The Council at its 99th Session in June 1985, is now available from:

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ACQUIRED IMMUNE DEFICIENCY SYNDROME IN SAUDI ARABIA

The first two cases of AIDS have recently been reported from Saudi Arabia.[1]

The syndrome developed in a 42 year old man who received 16 units of whole blood in 1981 for a bleeding duodenal ulcer and in a 5 1/2 year old boy with glucose-6-phosphate dehydrogenase deficiency who received 300 mL of whole blood in 1981 during a haemolytic episode induced by trimethoprin-sulfamethoxazole. These patients developed AIDS 2 1/2 and 3 1/2 years respectively after transfusion with blood imported from The United States of America.

Further cases of transfusion associated AIDS are expected in Saudi Arabia as a significant but as yet undetermined number of Saudi patients have received blood from this source.

The screening of blood donations in Saudi Arabia and the USA for antibodies to HTLV-III and the limiting of imports of blood products from countries with a high incidence of AIDS are seen as the key tactical components in a program to stop further transfusions with HTLV-III infected blood/blood products.

REFERENCE

1. JAMA 1986; 255:383-384

HTLV-III FROM GENITAL SECRETIONS OF SEROPOSITIVE WOMEN.

Two reports of the isolation of HTLV-III from the cervical and/or vaginal secretions from 8/22 women who were seropositive for the virus, have recently been published[1,2].

In the first[1], HTLV-III was cultured from the cervical secretions of 4/14 women who were seropositive for the virus (ELISA). Each of the 4 also had blood cultures positive for HTLV-III. The virus was also isolated from the blood of 3/13 women (one woman not tested) whose cervical secretions were culture negative for the virus. Of the four with positive cervical cultures, 1 had the clinical features of AIDS related complex (ARC), 2 had persistent generalised lymphadenopathy and 1 was apparently healthy. Three were parenteral drug abusers (PDA) and one had had sexual contact with a PDA. One of the women also stated that she had had more than 40 sexual partners in the previous year.

In the second report[2], HTLV-III was cultured from the vaginal/cervical secretions (obtained mid-way through the menstrual cycle) of 4/8 women who were seropositive for the virus. Each of the 4 also had blood cultures positive for HTLV-III. Three of the 4 women with negative vaginal/cervical secretions had positive blood cultures. Of the 4 with positive vaginal/cervical cultures, 2 had a 3-month history of lymphadenopathy, 1 had symptoms of a viral infection atypical of an AIDS related condition and 1 was healthy. All 4 women were either parenteral drug abusers, or had had sexual contact with a male in one of the risk groups, or both.

CDI Editorial comment

Male to female sexual transmission of HTLV-III, has been well documented[3-8]. By contrast, less data exists supporting

emale to male sexual transmission of the virus. It is ossible that either female-to-male sexual transmission of the irus is less efficient than male-to-female transmission, or he number of heterosexually infected males reflects the elatively small size of the pool of HTLV-III infected women.

he demonstration of virions in the vaginal/cervical secretions f a significant proportion (36%) of HTLV-III seropositive omen accords with the fact that female to male heterosexual ransmission of the virus has been reported.

EFERENCES

1. Lancet 1: 525-27 (1986)
2. Lancet I: 527-29 (1986)
3. N. Engl. J. Med 308: 1181-84 (1983)
4. JAMA 253: 1571-73 (1985)
5. Ann Intern Med 102: 63-66 (1985)
6. Ann Intern Med 102: 623-26 (1985)
7. MMWR 34: 561-63 (1985)
8. Asian Pacific J. Allerg. Immun. 3: 195-99 (1986)

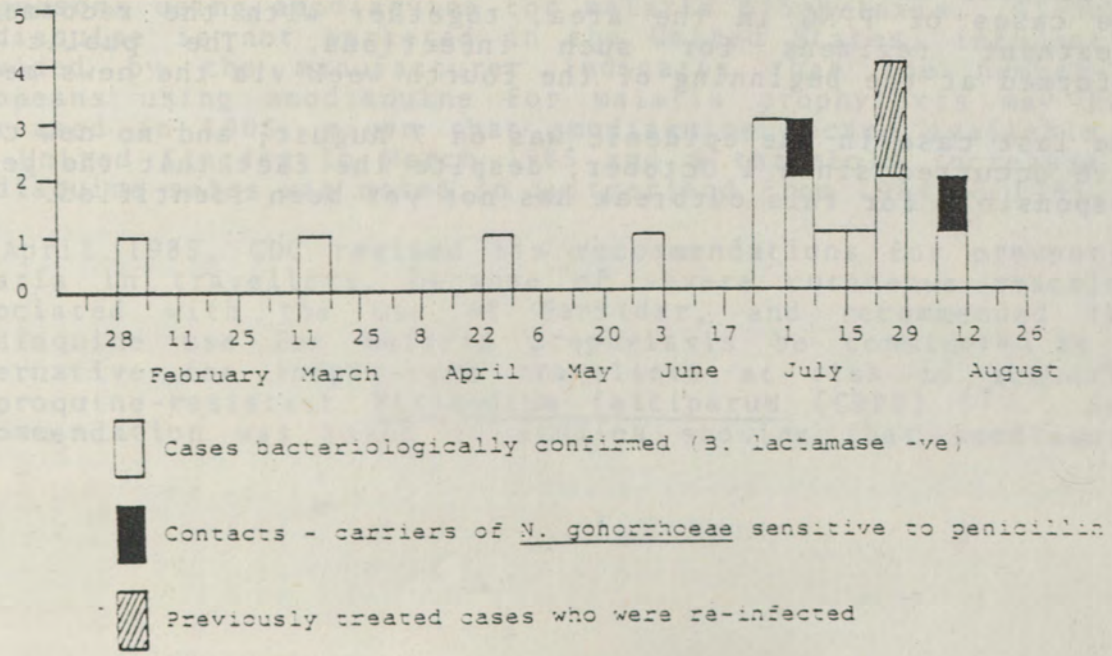
EPIDEMIC OF PENICILLINASE-PRODUCING NEISSERIA GONORRHOEAE - QUEBEC

(Based on CDWR Vol 11-50, 14 December 1985)

rior to 1985, only 1 case of penicillinase-producing Neisseria gonorrhoeae (PPNG) had been diagnosed in the Quebec City and its metropolitan region. However, during the summer of 1985, the Quebec City experienced an epidemic of PPNG (Figure 1):

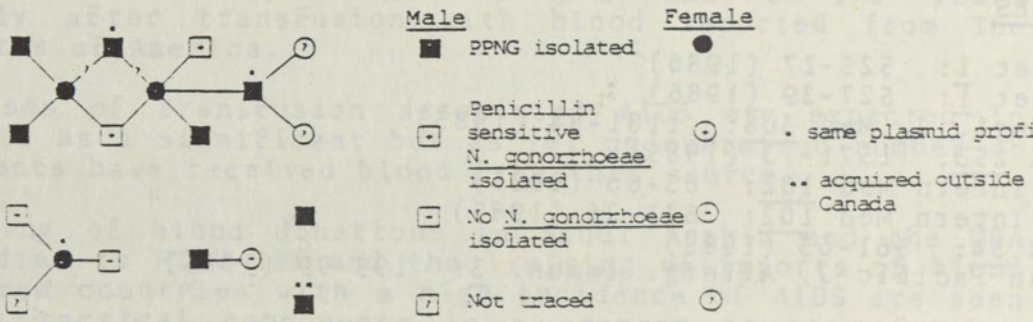
- between February and end of May 1985, 4 unrelated cases, resulting from sexual contact outside the province or with transient foreigners were reported; at that time, no local transmission of the infection had occurred.
- between 24 June and 12 August (a 7-week period), 11 cases of PPNG were bacteriologically confirmed and reported in the 4 Metro-Quebec Departments of Community Health.

IGURE 1. Distribution of PPNG cases according to the date when the culture was done



This was the first time that local transmission of PPNG has been documented in the Quebec city area. Six of the 11 cases (and possibly a seventh) were associated epidemiologically (Figure 2).

FIGURE 2. Schematic representation of transmission of the infection



Medical histories and interviews indicated that all the confirmed cases had resulted from heterosexual contact. Eight of the 11 cases were male, giving a male: female ratio of 2.7:1. Positive cultures were obtained from the urethra in the males and the cervix in the females. Ages ranged from 19 to 60 years, with a median of 25. Ten of the cases were symptomatic (including 2 of the 3 females). Information was not available for the remaining case.

At the beginning of August, the Metro-Quebec Departments of Community Health commenced a regional epidemiological investigation. A systematic search for contacts was conducted to attempt to insure that all carriers received the appropriate treatment. However, at least 3 contacts could not be traced, 2 being unknown and 1 completely untraceable.

By the third week of August, a letter was sent to all physicians and laboratories in the region notifying them about the cases of PPNG in the area, together with the recommended treatment regimens for such infections. The public was informed at the beginning of the fourth week via the news media.

The last case in the epidemic was on 7 August, and no new cases have occurred since 1 October, despite the fact that the person responsible for this outbreak has not yet been identified.

AGRANULOCYTOSIS ASSOCIATED WITH THE USE OF AMODIAQUINE FOR MALARIA PROPHYLAXIS

(Based on MMWR Vol. 35/No 10, 14 March 1986)

A total of 25 cases of agranulocytosis associated with the use of amodiaquine (Camoquine) have been reported: 7 cases among British travellers, 16 cases from Western Europe and 2 US cases. Twenty-three of these 25 cases occurrred in 1985 or 1986, and seven are reported to have been fatal. Among 20 cases for which the duration of amodiaquine prophylaxis is known, usage ranged from 3 weeks to 24 weeks. In 21 of the 25 cases, amodiaquine was used at the appropriate adult dosage of 400 mg base per week for prophlaxis. Concomitant use of another antimalarial drug for prophylaxis has been recorded in 14 patients with five cases using pyrimethamine - sulfadoxine (Fansidar) weekly and nine cases using proguanil (Paludrine) daily.

MMWR EDITORIAL COMMENT

Amodiaquine, a 4-aminoquinoline similar to chloroquine in structure and activity, has been used as both an antimalarial and an anti-inflammatory agent for more than 30 years. Only three of the 13 reports published between 1955 and 1985 indicated an association between agranulocytosis and the use of amodiaquine at recommended dosages for malaria prophylaxis in the absence of the use of other drugs known to have similar toxicity[2, 3].

The reason for these recent experiences with amodiaquine is not clear. While previously used largely for treating malaria in endemic areas, amodiaquine has been increasingly recommended for chemoprophylaxis in nonimmune visitors to endemic areas[4, 5]. It is not known whether bone-marrow toxicity is more likely to occur when the drug is used on a routine weekly basis for prophylaxis or when used in combination with other antimalarials, such as Fansidar or Paludrine. Agranulocytosis has been associated with the use of Fansidar alone[6], but has not been reported when Paludrine has been used alone.

This recent increase in the number of agranulocytosis cases might alternatively be explained by an increase in the number of persons using amodiaquine for malaria prophylaxis. Although amodiaquine is not marketed in the United States, information provided by the manufacturer indicates that the number of Europeans using amodiaquine for malaria prophylaxis may have increased in 1985, given that amodiaquine became available in the United Kingdom in March 1985 and a threefold increaase in amodiaquine sales was noted in Switzerland from 1984 to 1985.

In April 1985, CDC revised its recommendations for preventing malaria in travellers, because of severe cutaneous reactions associated with the use of Fansidar, and recommended that amodiaquine use for malaria prophylaxis be considered as an alternative for longer-term travellers at risk of acquiring chloroquine-resistant Plasmodium falciparum (CRPF) [7]. Such recommendation was based on studies showing that amodiaquine

was somewhat more effective than chloroquine in treating CRPF infections[8] and might therefore provide more protection than chloroquine when used as weekly prophylaxis in areas where CRPF transmission occurs. Similarly, WHO recently suggested the use of amodiaquine as an alternative to chloroquine and recommended it be used in combination with Paludrine or Maloprim (dapsone-pyrimethamine) for travel to certain areas[4].

It is now apparent that any possible prophylactic advantage that amodiaquine may afford is not justified by the risk of agranulocytosis associated with the use of the drug. Subsequently, CDC no longer recommends that amodiaquine be used for prophylaxis. Otherwise, previous recommendations for the prevention of malaria in travellers remain valid[5, 7].

CDI EDITORIAL COMMENT

Amodiaquine is not currently available in Australia but is widely used in many malaria endemic countries, particularly as a 100 mg pleasant tasting, easily quartered, chewable, paediatric formulation.

REFERENCES

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2. Nov Presse Med (1981) 10: 2827-8

3. Br. Med. J. (1967) 3: 32-3

4. WHO (1986) Vaccination certificate requirements and health advice for international travel: 46-8

5. CDC publication no 85-8280: 73-82

6. Lancet (1982) 11: 994

7. MMWR (1985) 34: 185-90

8. Lancet (1984) 1: 357-9

SALMONELLA HEIDELBERG OUTBREAK AT A CONVENTION - NEW MEXICO

(Based on MMWR Vol. 35/No 6, 14 February 1986)

At a convention held between 6 and 8 October 1985 in Santa Fe, New Mexico, 91 of the 1 000 attendees reported a diarrhoeal illness with onset of symptoms between 10 a.m. October 7, and

11 p.m. October 12. The ill attendees reported spending over $11 000 on medical costs and lost 117 days of work. Three persons were hospitalised. Salmonella heidelberg, sensitive to all antibiotics tested, was isolated from the stools of five attendees.

A telephone survey of 76 convention attendees living in New Mexico showed that, of four meals consumed at the convention, only the breakfast of October 7 was significantly associated with illness ($p < 0.002$).

In a subsequent mail survey of the approximately 550 convention attendees who ate the breakfast, the only food significantly associated with illness among the 60% who responded, was eggs. All of the 91 ill attendees ate the eggs, compared with 189 (92%) of the 206 well attendees ($p=0.01$). Eggs served at the meal were not available for culture; other eggs from the same distributor were culture-negative for Salmonella. The eggs had been cracked and stored in tall 2-gallon containers in a walk-in refrigerator the evening before the breakfast. They were then cooked in batches in a steamer in the morning. Several attendees commented that the eggs seemed 'runny'.

Of the staff who worked at the breakfast, three reported illness compatible with Salmonellosis with onset during the same period as the conventioneers, and all three had eaten the eggs. S. heidelberg was isolated from the stools of two staff members who did not handle food but had eaten the eggs.

MMWR EDITORIAL COMMENT

As illustrated by this outbreak, egg-related illness remains an important public health concern. Pathogens may proliferate in eggs or in other food refrigerated in large containers, since the centre of the container may be inadequately cooled [1]. In this outbreak, the fact that many well attendees also ate eggs suggests that only some egg containers were contaminated, that only some eggs were cooked sufficiently to kill the bacteria, or that susceptibility to infection may have varied among the attendees.

In the 1960s, eggs were responsible for a large proportion of salmonellosis outbreaks. With improvements in egg processing and quality control, egg-related outbreaks decreased dramatically in the 1970s [2]. For the 10-year period 1973-1982, 11 outbreaks of salmonellosis due to eggs were reported to CDC's Foodborne Disease Surveillance System. Of the 307 ill people in these outbreaks, 45 (15%) were hospitalised, and nine (3%) died [3]. S. heidelberg has been frequently associated with poultry, accounting for 29% of Salmonella isolates from poultry submitted to the U.S. Department of Agriculture in 1982 [4].

REFERENCES

1. J. Food Protection (1980) 43 : 140-50

2. J. Food Protection (1977) 40 : 798-800

3. CDC (unpublished data)

4. CDC 1982 Salmonella Surveillance.

HUMAN SALMONELLOSIS SURVEILLANCE

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

A total of 820 salmonella (74 serotypes), 193 shigella and 343 campylobacter reports from human cases were collected in Australia during July-September 1985.

Salmonella typhi

- S.typhi D2 was isolated from a 25 year old male Danish traveller.
- S.typhi untypable was isolated from a 51 year old female with rigors and fever following her return from a trip to Bali.

Salmonella paratyphi

- S.paratyphi A
  - phage type 1 was isolated from a 58 year old male with severe diarrhoea after returning from the Philippines
  - phage type 10 was isolated from a 30 year old male with gastroenteritis and bacteraemia.
  - untypable was isolated from a 47 year old male who had just returned from the Philippines.
- S.paratyphi B
  - RDNC was isolated from a 37 year old female whose personal details are unknown.
  - untypable was isolated from a 26 year old female whose history was unavailable.

OTHER SALMONELLA INFECTIONS

A. ISOLATIONS FROM URINE: comprised the following serotypes:

- S.ohio var 14+ was isolated from a 78 year old female.
- S.typhimurium 101 was isolated from a 13 year old female.
- S.typhimurium 9 was isolated from a 4 year old female.
- S.typhimurium RDNC was isolated from a 3 year old female.
- S.typhimurium untypable was isolated from a 2 year old female.

B. ISOLATIONS FROM BLOOD: Cases of septicaemia involved the following serotypes:

- S.dublin was isolated from a 55 year old male with aorta-caval fistula
- S.havana was isolated from a 1 year old child who was also excreting the organism in the faeces (sex unknown)
- S. typhimurium 4 was isolated from a 62 year old male with lymphoma.
- S.typhimurium 135 was isolated from a 69 year old male with infective aortitis.

Shigella septicaemia was reported in 2 cases, one a 2 year old female and the other, a 19 year old female who had recently returned from India. Both cases were infected with Sh.flexneri 2A.

Other isolations of interest included the following serotypes:

- S.infantis was isolated from the pus of a prosthesis, a thigh abscess and faeces in a 58 year old female.
- S.kottbus was isolated from the limbal swab of donor eyes (age and sex unknown)

. S.ohio was isolated from the wound swab of a 71 year old male and also from the bile duct of a 21 year old female.
. S.typhimurium 26 was isolated from the vaginal swab of a 17 year old.
. S.typhimurium RDNC was isolated from antral (unspecified) washings of a 22 year old female.

Serotypes reported for the first time in the surveillance scheme included S.monschaui, S.paratyphi A 10, S.typhimurium 160 and S.wien. The latter was isolated from a 8 month old female who had become ill 3 weeks earlier in Yugoslavia. The culture was resistant to Ampicillin, Streptomycin, Tetracycline, Chloramphenicol, Sulphonamide/Trimethoprim, Kanamicin, Gentamicin and naladixic acid.

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 (number of isolates in brackets).

A. Salmonella species:

. S.agona (6), S.anatum (4), S.blockley (4), S.bredeney (2), S.bovismorbificans (1), S.emek (1), S.hadar (1), S.hvittingfoss (3), S.london (unknown), S.newport (1) and S.weltevreden (4) were acquired in South East Asia.
. S.bareilly (2) were acquired in Sri Lanka.
. S.derby (2) was acquired in China and South East Asia.
. S.duesseldorf (1) and S.typhimurium (92) were acquired in India.
. S.enteritidis (3) were acquired in India and Portugal.
. S.haifa (1) was acquired in the Middle East.
. S.indiana (1) and S.infantis (2) were acquired in the Netherlands.
. S.java (1) and S.montevideo (2) were acquired in Bali.
. S.krefeld (2) was acquired in Thailand and South East Asia.
. S.mbandaka (1) was acquired in the Maldives islands.
. S.senftenberg (1) was acquired in Indonesia.
. S.untypable (3) were acquired in Papua New Guinea (1) and South East Asia (2).
. S.charity (1), S.javiana (1) and S.panama (1) were acquired overseas (country unspecified).

B. Shigella species:

. Sh.dysenteriae 1 was isolated from a 32 year old male who had returned from Sudan.
. Other shigella serotypes included:
  - Sh.flexneri types 1a, 1b, 2a, 3a, 4a and 6
  - Sh.dysenteriae types 7 and 9
  - Sh.sonnei biotypes a and g
  - Sh.boydii 2.

CLUSTERS OF ENTERIC INFECTIONS:

S.typhimurium 9 isolations showed a marked increase during this quarter, in particular in New South Wales and the ACT. The demographic pattern of isolates indicated that over half were recovered from children under 10 with an even distribution in the age groups 0-2, 3-5, 6-10 years. The majority of New South Wales isolates originated from the metropolitan area of Sydney but country cases were reported from Armidale, Tamworth, Bathurst, Goulburn and Batemans Bay. No definite source of the outbreak was identified.

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S.aberdeen	3				3				
S.abony	2			1	1				
S.adelaide	5		1	1			3		
S.agona	18		6	3	4	2	2	1	
S.anatum	16		1	4	6		4		1
S.anatum var 15+	2				1				1
S.arizonae	6				5				1
S.ball	1								1
S.bareilly	2			2					
S.birkenhead	8		4	1	3				
S.blockley	8		4			1	3		
S.bonn	1		1						
S.bovismorbificans	18	2	10	2			4		
S.bovismorbificans 11	1					1			
S.bovismorbificans 15	1		1						
S.bovismorbificans 16	1					1			
S.bovismorbificans 17	1		1						
S.bovismorbificans 23	1		1						
S.bovismorbificans 6	1					1			
S.bredeney	6			3	2		1		
S.charity	1		1						
S.chester	21		2	1	10	3	4		1
S.derby	6		3	1			2		
S.dublin	1			1					
S.duesseldorf	1			1					
S.eastbourne	1				1				
S.emek	2			2					
S.enteritidis	10	2	1		6	1			
S.give	5		1	2	1		1		
S.hadar	1			1					
S.haifa	1			1					
S.havana	15		2		3	3	3		4
S.heidelberg	4		1		2		1		
S.hvittingfoss	5			2					3
S.indiana	1			1					
S.infantis	22		2	5	4	2	9		
S.jangwani	1								1
S.java	2						2		
S.java Dundee	1					1			
S.java RDNC	1					1			
S.java untypable	2			1					1
S.javiana	2			1	1				
S.kottbus	2		1			1			
S.krefeld	2		1	1					
S.lansing	5				5				
S.litchfield	4				1		2		1
S.livingstone	1				1				
S.london	1			1					
S.mbandaka	3		1	2					
S.meleagridis	1					1			
S.monschaui	1				1				
S.montevideo	4		1	2	1				
S.muenchen	20		1		11	1	4		3
S.newport	10		3	2		1	4		
S.ohio	11	1	4	1		3	1	1	
S.ohio var 14+	2		2						
S.ohlstedt	1				1				
S.oranienburg	11		1				8		2
S.orientalis	1		1						

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
S.orion	4						1		3
S.orion var 15+	1					1			
S.panama	1		1						
S.paratyphi A1	7			7					
S.paratyphi A10	1		1						
S.paratyphi A RDNC	3			3					
S.paratyphi A untypable	1			1					
S.paratyphi B RDNC	1					1			
S.paratyphi B untypable	1		1						
S.potsdam	3				2	1			
S.richmond	1			1					
S.rubislaw	4				1	1	1		1
S.saintpaul	31		1	2	19		3		6
S.schwarzengrund	1		1						
S.senftenberg	6		3				2		1
S.singapore	14		5	4		1	4		
S.sofia subgenus II	2			1	1				
S.tennessee	4						2		2
S.thompson	3				3				
S.typhi*	11			5	6				
S.typhimurium*	376	69	121	61	32	39	41	12	1
S.4,12:d:-	6		1		5				
S.untypable -:1v:enz15	1					1			
S.untypable 1,13,23:b:-	1			1					
S.untypable 16:1v:-	1				1				
S.untypable 3,10:r:-	1								1
S.untypable 4,12:-:-	2		2						
S.untypable 4,5,12:-:1,2	2		2						
S.untypable rough:-:-	1					1			
S.untypable rough:1v:enz15	1					1			
S.untypable rough:mt:-	1						1		
S.untypable 6,8:eh:-	1			1					
S.urbana	1						1		
S.virchow	24		2	1	20				1
S.wandsbek subgenus II	1						1		
S.waycross	3				3				
S.welikade	2								2
S.weltevreden	6	1		3					2
S.wien	1		1						
TOTAL	820	75	201	137	167	71	115	14	40

S.typhi*					
S.typhi A	2			2	
S.typhi D2	4			4	
S.typhi E1	3		3		
S.typhi untypable	2		2		
TOTAL	11		5	6	

S.typhimurium*						
S.typhimurium	8	2			6	
S.typhimurium RDNC	29	16	3	2	5	3
S.typhimurium untypable	5	2	2	1		
phage type 2	1			1		
phage type 4	7		4		2	1

Serotype	Total	ACT	NSW	VIC	QLD	SA	WA	TAS	NT
phage type 5	10		2		1	6		1	
phage type 6	5			4				1	
phage type 8	7			5			2		
phage type 9	155	69	68	10	6	2			
phage type 16	2				2				
phage type 21	1						1		
phage type 22	6		1	1	3		1		
phage type 25	3		1	1	1				
phage type 26	12		2	6		4			
phage type 27	3		1		1		1		
phage type 30	1					1			
phage type 31	5			4			1		
phage type 41	1					1			
phage type 44	8			4	3		1		
phage type 46	1				1				
phage type 58	8						8		
phage type 64	12			1			11		
phage type 81	1			1					
phage type 90	3		1			2			
phage type 101	8		1			2		5	
phage type 102	2			1			1		
phage type 108	2			1	1				
phage type 126	1		1						
phage type 12a	8		2	1	2	2		1	
phage type 135	37		14	7	6	7		2	1
phage type 141	2		1				1		
phage type 145	2		1	1					
phage type 155	1							1	
phage type 156	3					3			
phage type 160	1			1					
phage type 170	5		4		1				
phage type 179	6		1	2			3		
phage type 185	2			1			1		
phage type 202	2					2			
TOTAL	376	69	121	61	32	39	41	12	1

UPDATE : CREUTZFELDT-JAKOB DISEASE AND HUMAN GROWTH HORMONE

Creutzfeldt-Jakob Disease (CJD) is a subacute pre-senile dementia which has an incidence of approximately 1 case/million/year. It affects both sexes equally, is almost invariably fatal, and occurs worldwide. Symptoms, which rarely present before the age of 40, generally begin with vagueness and clumsiness, and progress to spasticity and dementia, often with dysarthria, ataxia and myoclonic jerks. Death usually occurs between 6 and 24 months from the onset of symptoms.

CJD produces a characteristic encephalopathy, with a spongiform vacuolation of cortical grey matter, astrocytosis and cerebellar atrophy. The pathology of the disease is similar to that seen with scrapie in sheep and goats, and with kuru in man and the aetiological agents of these diseases are similar. For these reasons, CJD is included in the spongiform encephalopathy group of diseases.

The aetiological agent of CJD is believed to be a prion, a virus-like particle with no demonstrable nucleic acid (DNA or RNA). The method of reproduction and the natural route of transmission to man are unknown. The agent has been found in brain tissue and viscera of patients, and the disease has been transmitted experimentally to primates, guinea pigs and cats by

inoculation with human brain material obtained at post mortem. The incubation period in man is thought to be several years. In experimentally infected chimpanzees, incubation periods of one to several years have been reported.

Iatrogenic transmission of the disease has been described. CJD has been transmitted by a corneal transplant from a live donor who subsequently presented with the disease[1], and the use of a tonometer to measure intraocular pressure has been implicated in the transmission of CJD[2]. The disease has also been transmitted by cerebral insertion of electrodes[3] and by surgical procedures on the head and neck[2].

The use of natural Human Growth Hormone (hGH) has been implicated in the deaths of three patients from neurological disorders in the US (See CDI Bulletin No 85/17, 23 August 1985). Concern over the safety of natural hGH has increased since the recent death of a 23 year old woman from CJD after receiving the hormone and 4 other deaths from neurological disorders in recipients of hGH. Although the last four cases did not present as typical CJD patients, a more detailed examination is being undertaken because of the relatively high incidence of CJD in hGH recipients in the last year.

The organism is remarkably resistant to standard sterilisation and disinfection methods, including ethanol, iodine, hydrochloric acid, formaldehyde, sodium hydroxide, sodium hypochlorite and ultraviolet radiation at 254nm. It is insensitive to proteases and nucleases and retains infectivity after autoclaving at 121°C for 4 hours.

The organism is difficult to detect. Bioassay methods have incubation periods of the order of two years, and there is doubt as to whether currently available animal models of infection are sufficiently sensitive. The problem is exacerbated by "unresolved safety concerns" associated with Genentech's rDNA hGH (See CDI bulletin No 85/17 23 August 1985) which raises questions about the suitability of hGH produced by recombinant DNA methods for human use.

Work by Tateishi et al[4,5], however indicates that suitable procedures for the quantitative separation of hGH from the CJD agent may soon be at hand. The pathogen is retained by a membrane filter with a pore size of 0.25um[4] while the hormone is recovered in the filtrate[5]. The authors suggest that the filtration step could be added to current methods of production of natural hGH.

A second, more recent advance is the production of antibody to the core protein of the agent. Immunological methods can now be used to study CJD ante mortem, and it is thought that epidemiological studies will soon be feasible. Application of the new assay method to natural hGH may prevent future iatrogenic transmission of CJD via this hormone.

REFERENCES

1. N. Engl. J. Med. (1974) 290 692-3

2. N. Engl. J. Med. (1984) 311 1582-3

3. Lancet (1977) 1 478-9

4. Ann. Neurol (1980) 7 390-1

5. Lancet (1985) 2 1299-30

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 17/3/86 - 30/3/86 BULLETIN NUMBER 86/7
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		2	1	8	2	9	1	24
0101 ADENOVIRUS TYPE 1		1	1		5				7
0102 ADENOVIRUS TYPE 2				1	6			1	8
0105 ADENOVIRUS TYPE 5					1	1			2
0106 ADENOVIRUS TYPE 6					2				2
0108 ADENOVIRUS TYPE 8				3					3
0137 ADENOVIRUS TYPE 37				1					1
0199 ADENOVIRUS TYPING PENDING	13				4				17
0201 INFLUENZA A VIRUS			1						1
0203 INFLUENZA B VIRUS	2					1			3
0301 PARAINFLUENZA VIRUS TYPE 1				1	5				6
0303 PARAINFLUENZA VIRUS TYPE 3					2	1			3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1								1
0500 RHINOVIRUS (ALL TYPES)	1			2	9	6	1		19
0600 MYCOPLASMA PNEUMONIAE	12	1	1					2	16
0700 ORNITHOSIS-PSITTACOSIS						1			1
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED							1		1
0816 COXSACKIEVIRUS A16	3	1							4
0904 COXSACKIEVIRUS B4					3				3
1002 ECHOVIRUS TYPE 2				1					1
1007 ECHOVIRUS TYPE 7					3				3
1014 ECHOVIRUS TYPE 14			1						1
1020 ECHOVIRUS TYPE 20	1								1
1021 ECHOVIRUS TYPE 21				1					1
1100 POLIOVIRUS NOT TYPED			5		8				13
1101 POLIOVIRUS TYPE 1	1								1
1300 HERPES VIRUS GROUP-NOT TYPED	19			1		1	1	1	23
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	14	1	2			1		6	24
1303 VARICELLA-ZOSTER VIRUS	2			1		2	1	2	8
1306 HERPES SIMPLEX TYPE 1	15		12	68	6	19	49	22	191
1307 HERPES SIMPLEX TYPE 2	76		25	88		16	89	37	331
1399 HERPES VIRUS TYPING PENDING					3				3
1401 COXIELLA BURNETI	3		2			1			6
1502 PICORNA VIRUS-NOT TYPED	3		5			5		2	15
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS			1						1
1522 RUBELLA VIRUS			1	3				2	6
1532 HEPATITIS B ANTIGEN	28		10		1	21	10	21	91
1535 HEPATITIS A ANTIBODY	4		2	28		20	3	4	61
1541 CHLAMYDIA A - C TRACHOMATIS	37		4			44	19	45	149
1556 CMV - CYTOMEGALOVIRUS	9	2	3	38	3	4	2	4	65
1563 CORONAVIRUS	2								2
1564 ROTAVIRUS	5		2		3	4		1	15
1599 ENTEROVIRUS TYPING PENDING		4	6		6				16
9992 ROSS RIVER VIRUS			3	16				3	22
Total	252	10	89	254	78	150	185	155	1,173

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 17/3/86 - 30/3/86

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0101 ADENOVIRUS TYPE 1.....		6					1	1			
0102 ADENOVIRUS TYPE 2.....		5					2				
0105 ADENOVIRUS TYPE 5.....		2									
0106 ADENOVIRUS TYPE 6.....							2				
?01 INFLUENZA A VIRUS.....		1									
203 INFLUENZA B VIRUS.....		1									1
0301 PARAINFLUENZA VIRUS TYPE 1....		6									
0303 PARAINFLUENZA VIRUS TYPE 3....		3									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		1									
0500 RHINOVIRUS (ALL TYPES).....		10									
0600 MYCOPLASMA PNEUMONIAE.....		13									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED.....											1
0816 COXSACKIEVIRUS A16.....	2										2
0904 COXSACKIEVIRUS B4.....									1		
1007 ECHOVIRUS TYPE 7.....		1									
1014 ECHOVIRUS TYPE 14.....		1					1				
1020 ECHOVIRUS TYPE 20.....		1									
1021 ECHOVIRUS TYPE 21.....				1							
1100 POLIOVIRUS NOT TYPED.....							1				
1300 HERPES VIRUS GROUP-NOT TYPED..	1	2									
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	3	4									1
1303 VARICELLA-ZOSTER VIRUS.....	1										6
1306 HERPES SIMPLEX TYPE 1.....	7	8		1				1		6	100
1307 HERPES SIMPLEX TYPE 2.....	16	1									59
1401 COXIELLA BURNETI.....									1		
1502 PICORNA VIRUS-NOT TYPED.....							6	1	2		
'521 MEASLES VIRUS.....						1					
522 RUBELLA VIRUS.....											3
1532 HEPATITIS B ANTIGEN.....	20	1						53			
1535 HEPATITIS A ANTIBODY.....	13							45			
1556 CMV - CYTOMEGALOVIRUS.....	2	5					1		1	4	1
1563 CORONAVIRUS.....							2				
1564 ROTAVIRUS.....							15				
9992 ROSS RIVER VIRUS.....											2
Total.....	65	73		2		1	31	101	5	10	176

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 17/3/86 - 30/3/86

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		
0102 ADENOVIRUS TYPE 2.....							1	2		
0105 ADENOVIRUS TYPE 5.....	1									
0108 ADENOVIRUS TYPE 8.....	3									
0137 ADENOVIRUS TYPE 37.....	1									
0203 INFLUENZA B VIRUS.....			1					2		
0600 MYCOPLASMA PNEUMONIAE.....			1					4		
0904 COXSACKIEVIRUS B4.....							1			1
1002 ECHOVIRUS TYPE 2.....								1		
1007 ECHOVIRUS TYPE 7.....							1		1	
1100 POLIOVIRUS NOT TYPED.....							1			
1101 POLIOVIRUS TYPE 1.....									1	
1300 HERPES VIRUS GROUP-NOT TYPED..								1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			7	2			1	4	5	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	2	55					2	3	7	
1307 HERPES SIMPLEX TYPE 2.....	1	254							1	
1401 COXIELLA BURNETI.....					1		2	2		
1502 PICORNA VIRUS-NOT TYPED.....			1							
1514 MOLLUSCUM CONTAGIOSUM.....		1								
1522 RUBELLA VIRUS.....									3	
1532 HEPATITIS B ANTIGEN.....									16	
1535 HEPATITIS A ANTIBODY.....									3	
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	146								
1556 CMV - CYTOMEGALOVIRUS.....		5		1	1	3	3	4	34	
9992 ROSS RIVER VIRUS.....					22					
Total.....	11	461	10	3	24	3	12	24	72	1

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

Periods 12 & 13
3 November 1985 to 31 December 1985

Bulletin 86/I..

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		3	2					6	58
Ankylostomiasis			5	1			N.N.		6	40
Anthrax	1								1	1
Arbovirus infection	1		58						59	550
Brucellosis			1						1	16
Campylobacter infections	226	N.N.	N.N.	242	3	N.N.	11	N.N.	482	*2342
Chancroid			1	N.N.		N.N.			1	9
Cholera		1							1	1
Congenital rubella syndrome		N.N.	N.N.			N.N.		N.N.		3
Diphtheria							6		6	23
Donovanosis	1	N.N.		N.N.		N.N.	2		3	* 75
Giardiasis	46	N.N.	N.N.	85	2	N.N.	N.N.	N.N.	135	* 1091
Genital herpes	155	N.N.	122	34	N.N.	N.N.	7		318	1714
Gonococcal ophthalmia neonatorum		N.N.	N.N		N.N.	N.N.		N.N.		5
Gonorrhoea	192	235	266	126	120	5	126	5	1075	7509
Hepatitis A (infectious)	39	8	37	44	30	2	8		168	* 836
Hepatitis B (serum)	60	27	53	13	25		7	1	186	* 1625
Hepatitis - unspecified	16		5		1	N.N	2		24	* 122
Hydatid disease	4								4	15
Lassa Fever		N.N	N.N.			N.N.	N.N.	N.N.		1
Legionnaires' disease	5	2	N.N.	1		N.N.		N.N.	8	* 29
Leprosy	2	1							3	40
Leptospirosis	5	4	4	2	1	6			22	183
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.				7
Malaria	21	21	32	2	3	1	2	1	83	* 606
Marburg Disease		N.N.	N.N.			N.N.	N.N.	N.N.		
Meningococcal infections	2	1		1		N.N.			4	53
Non-specific urethritis	577	N.N.	59	184	N.N.	N.N.	2	N.N.	822	4847
Ornithosis		4		4					8	16
Pertussis (whooping cough)	81	13	N.N.	23	3	N.N.		N.N.	120	* 588
Plague										
Poliomyelitis										
Q. fever	3	2	15	5			N.N.		25	194
Rabies		N.N	N.N.	N.N.		N.N.	N N.	N.N.		

2.

DISEASE	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Salmonella infections	121	25	53	72	12	13	46	6	348	* 2665
Shigella infections	24	3	15	7	7		18		74	720
Smallpox										
Syphilis	60	8	68	56	26		117	5	340	2327
Tetanus										10
Trachoma		N.N.				N.N.	N.N.			63
Tuberculosis (all forms)	74	49	38	20	8		2	2	193	* 1129
Typhoid fever	7	2	1						10	37
Typhus (all forms)		2							2	7
Vibrio parrahaemolyticus infections		N.N.	N.N.			N.N.		N.N.		4
Yellow Fever										1
Yersinia enterocolitica infections	20	N.N.	N.N.	2		N.N.	1	N.N.	23	60

(Note: Data collected under the Notifiable Diseases Returns may bear little or no correlation to that collected under the CDI laboratory scheme. Whilst the latter is a sampling program, the Notifiable Diseases data is dependent upon voluntary reporting by medical practitioners etc.)

*N.N. Not Notifiable

Campylobacter Infections	+1	South Australia
Donovanosis	-1	Addition Error
Giardiasis	+1	South Australia
Hepatitis A	-1	South Australia
Hepatitis B	+4	South Australia
Hepatitis unspecified	-1	Western Australia
Legionnaires' disease	+1	South Australia
Malaria	+2	South Australia
Pertussis (whooping cough)	+26	South Australia
Salmonella infections	+3	South Australia
Tuberculosis	-2	South Australia