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Communicable Diseases Intelligence

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Contents:

. Influenza activity update
. Hepatitis B in a dental practice
. Hepatitis B in household contacts of haemophiliacs.

Editor Dr I.F. Cook

VIRUS REPORTING SCHEME: A total of 1 372 reports were processed for this period.

Eighteen cases of Q fever were reported (8 from New South Wales, 8 from Queensland and 2 from South Australia). Occupational exposure data were only available for:

. the 2 South Australian cases; a 43 year old male abattoir worker and a 33 year old male who frequently visited the local abattoir.

. four Queensland cases; 2 male meatworkers aged 40 (from Inverell) and aged 48 (from Mackay) respectively, a 28 year old male farmer and a 16 year old male trainee teacher.

Polio virus type III was isolated from the cerebrospinal fluid of a 22 year old male presenting with malaise, muscle stiffness and pain in the neck and back, and generalised hyperaesthesia, a couple of weeks after his six month old child has received an oral dose of Sabin vaccine. The case has been reported as vaccine-associated poliomyelitis in an unvaccinated close contact of a recipient of oral poliomyelitis vaccine with the patient experiencing difficulties with micturition and complaining of weakness in his left leg. Unvaccinated or incompletely vaccinated parents of children who are to be given oral poliomyelitis vaccine should be offered a basic course of the same oral vaccine.

INFLUENZA ACTIVITY: AN UPDATE

The occurrence of influenza in the tropics and the Southern Hemisphere between April and September of each year frequently indicates the influenza type that may occur in the Northern Hemisphere during the subsequent winter. The recent increase in circulation of influenza A (H1N1) virus in Southeast Asia suggests that it may appear in the United States during the 1986-1987 influenza season after an almost total absence during the last two seasons.(1)

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Influenza A (H3N2) and Influenza B activity[1]:

- Northern Hemisphere
  During the winter of 1985-1986, influenza outbreaks in the Northern Hemisphere, including the United States, were associated primarily with virus types A (H3N2) and B. Both influenza A (H3N2) and B have been reported from Western Europe (France, the Federal Republic of Germany, Italy, the Netherlands, Switzerland and the United Kingdom) and Scandinavia (Denmark, Finland, Norway and Sweden) and from the Union of Soviet Socialist Republics (USSR), Eastern Europe, and the Balkans (Hungary, Czechoslovakia, Poland and Yugoslavia). Influenza A (H3N2) virus was isolated from Belgium, Bulgaria and Greece. Influenza B was reported from the German Democratic Republic. Elsewhere in the Northern Hemisphere, influenza A (H3N2) virus was isolated in The People's Republic of China (PRC), the Democratic People's Republic of Korea, Hong Kong, and Japan; influenza B was isolated in the PRC, Iran, Israel, the Republic of Korea and Tunisia.

- Southern Hemisphere
  Thus far during 1986, occasional isolations of influenza A (H3N2) and B have been reported from the Southern Hemisphere. A single influenza A (H3N2) isolate was reported from Australia in April. Both influenza A (H3N2) and B viruses were reported from New Guinea in February, and influenza B was reported from Madagascar in March and from Taiwan in April.

Influenza A (H1N1) activity:

During the winter of 1985-86, influenza A (H1N1) viruses were infrequently reported from the United States, Bulgaria, Czechoslovakia, the USSR, the Democratic People's Republic of Korea and the People's Republic of China.

However, since late March, influenza A (H1N1) has been increasingly reported from areas of Southeast Asia. Malaysia and Hong Kong have experienced localised outbreaks, and Singapore and Taiwan have reported regional epidemics of influenza A (H1N1).

Australia, in its report of influenza A (H1N1) isolates for the month of August, drew attention to a new strain of influenza A (H1N1) which appeared in Southeast Asia, New Zealand, and to a limited extent only in Australia. Although a number of isolates still resembled A/Chile/1/83 most closely, antigenic analysis of this new strain of influenza A (H1N1) by the Commonwealth Serum Laboratory (WHO Influenza Reference Centre in Australia) has detected antigenic drift from the previously prevalent strain related to A/Chile/1/83 (H1N1)[2]. The antigenic variation has been evidenced by (i) reciprocal haemagglutination-inhibition tests using animal antisera and (ii) comparisons of antibody levels against the newer variants in persons immunised with vaccine containing A/Chile/1/83 antigen.

Animal antiserum to A/Chile/1/83 is less effective in inhibiting the newer A(H1N1) variants than in inhibiting A/Chile/1/83-like viruses, although the degree or reactivity varies among isolates of the newer A(H1N1) variants (Table 1). In contrast, antisera prepared against representative new isolates (eg. A/Singapore/6/86, A/Taiwan/1/86) react well with all other recent isolates but extremely poorly with A/Chile/1/83. In tests with post infection ferret antisera, the new variants also differed from other variants that have cocirculated with A/Chile/1/83 since 1983-1984 (eg. A/Victoria/7/83, A/Dunedin/27/83).

TABLE 1

Haemagglutination - inhibition reactions * of influenza A (H1N1) viruses with postinfection ferret antisera

	SERA								
Antigen	A/India 6263/80	A/England 333/80	A/Chile 1/83	A/Victoria 7/83	A/Dunedin 27/83	A/Switzerland 79/85	A/Taiwan 1/86	A/Singapore 6/86	A/Hong Kong 7/86
A/India/6263/80	320		80		40			20	
A/England/333/80		320	160	80	160	160		<	<
A/Chile/1/83	160	40	160	40	40	40	<	<	<
A/Victoria/7/83	160	80	320	320	40	160	40	<	<
A/Dunedin/27/83	40	80	80	40	640	160	<	<	<
A/Switzerland/79/85		80	160	40	160	640		<	<
A/Auckland/1/86		<	<	<	40	<		640	
A/Auckland/2/86		<	<	<	80	<		640	
A/Taiwan/1/86	10		40	<	40		2,560	320	
A/Beijing/1/86				<	40		1,280	640	
A/Singapore/6/86		<		<	80	<	1,280	1,280	640
A/Hong Kong/7/86	80	<		<	80	<		1,280	1,280

< = <40

* Titres are the reciprocal of antiserum dilutions; homologous titres are underlined. When comparing reactions of sera with different antigens, fourfold or greater differences are considered experimentally significant.

Through July 1986, influenza A (H1N1) variants had been detected among virus isolates from Kong Kong, Malaysia and New Zealand; from an imported case in England believed to have originated in India; and among viruses from a few outbreaks and sporadic cases of influenza A (H1N1) that occurred in Japan last winter. The presence of an A(H1N1) variant has also been reported from the USSR, where last winter's epidemic was primarily caused by influenza B and influenza A (H3N2) viruses. Recently, influenza A viruses, some confirmed as A(H1N1) strains, have also been reported in the USA, Thailand, American Samoa, and Palau and Micronesia. Influenza outbreaks in Palau and elsewhere in the U.S. Pacific Trust Territories are presently being investigated(3).

EFFECTIVENESS OF THE CURRENT TRIVALENT INFLUENZA VACCINE

The studies (conducted by the Center for Diseases Control Atlanta, Georgia) of antibody response in recipients of last year's influenza vaccine containing A/Chile/1/83 antigen demonstrated the difference of the new variants (Table 2). Approximately 80% of adult vaccinees developed titres to A/Chile/1/83 of 160 or higher and had a post-vaccination geometric mean titre (GMT) of 320. In contrast, only 15%-30% of the vaccinees developed the same titres to the new variants, and the postvaccination GMT was about sixfold lower. However,

since postvaccination titres of 40 or greater have been associated with reduced influenza infection and illness, it is possible that the A/Chile/1/83 antigen in the 1986-1987 trivalent vaccine will provide at least partial protection against the new variants(3).

TABLE 2 Haemagglutination - inhibition serum antibody response to influenza vaccine in immunised adults - United States, Autumn 1985*

	Pre-vaccine sera				Post-vaccine sera			
	Cumulative % with titre				Cumulative % with titre			
Antigen A(H1N1)	⩾ 10	⩾ 40	⩾ 160	GMT	⩾10	⩾40	⩾160	GMT
A/Chile/1/83	82	58	15	33	100	96	80	320
A/Singapore/6/86	87	20	2	18	98	80	15	50
A/Taiwan/1/86	49	15	4	10	93	75	29	56

*Fifty-five adults received trivalent split vaccine containing 15 ug each of A/Phillippines/2/82, A/Chile/1/83, and B/USSR/100/83.

To assess the effectiveness of current vaccines in inducing antibodies against the recent isolates, the World Health Organisation (WHO) have also tested several collections of sera from vaccine trials in which A/Chile/1/83 was used as the H1N1 component. The results in Table 3 show that these vaccines did not reliably induce antibodies against A/Singapore/6/86-like strains to sufficient levels.(4)

TABLE 3

Haemagglutination-inhibiting (HI) antibody responses to vaccination with inactivated vaccines containing A/Chile/1/83(H1N1)

Strain —	Trial	Pre-vaccination - % with HI titre -				Post-vaccination % with HI titre				GMT
		< 10	> 10	> 40	> 160	< 10	> 10	> 40	> 160	
A/Chile/1/83	1-84	68	32	8	—	4	96	93	64	180
	2-84	81	19	6	—	3	97	83	44	90
	1-85	55	45	10	—	—	100	90	45	90
	2-85	73	27	13	7	27	73	53	47	60
A/Singapore/6/86	1-84	94	6	1	—	35	65	30	5	15
	2-84	94	6	1	—	36	64	19	—	10
	1-85	85	15	—	—	20	80	40	5	21
	2-85	93	7	—	—	73	27	20	—	< 10

GMT = Geometric mean titre .

Vaccine studies .

1-84 One dose of disrupted virus vaccine containing 10 or 15 µg A/Chile/1/83 in 96 adults aged 18-60 years ; trial done in 1984.

2-84 One dose of surface antigen vaccine containing 10 µg A/Chile/1/83 in 36 adults aged 18-60 years ; trial done in 1984.

1-85 One dose of disrupted virus vaccine containing 10 µg A/Chile/1/83 in 20 adults aged 18-65 years ; trial done in 1985.

2-85 Two doses of disrupted virus or surface antigen vaccine containing 10 µg A/Chile/1/83 in 15 children 4-13 years ; trial done in 1985.

DISCUSSION

The result of haemagglutination-inhibition (HI) antibody responses elicited by inactivated vaccines containing A/Chile/1/83 (H1N1) reported by WHO (Table 3) appears to be at variance with the results obtained from studies of antibody responses elicited by the same vaccine, conducted by CDC, Atlanta, Georgia (Table 2). The discrepancy is most apparent in postvaccination sera studies where the cumulative % with HI titre was ⩾ 160:

- when challenged with A/Chile/1/83 as Antigen A (H1N1) - WHO reported that 46% of adult vaccinees developed titres of 160 or higher and had a postvaccination geometric mean titre (GMT) of 75, whereas CDC studies reported that 80% of adult vaccinees developed titres to A/Chile/1/83 of 160 or higher and had a postvaccination GMT Of 320.

- when challenged with A/Singapore/6/86 as Antigen A(H1N1) - WHO reported that only 5% of adult vaccinees developed titres of 160 or higher and had a postvacination GMT of 15 or less, whereas CDC studies reported that 15% of adult vaccinees developed titres to A/Singapore/6/86 of 160 or higher and had a post-vaccination GMT of 50.

Although the concensus from both studies appeared to indicate the much reduced effectiveness of the current trivalent vaccine in protecting vaccinees against the new variants, CDC data tended to support the conclusion that since postvaccination titres of 40 or greater have been associated with reduced influenza infection and illness, it is possible that the A/Chile/1/83 antigen component in the current trivalent vaccine will provide at least partial protection against the new variants. WHO data on the other hand, seemed to contradict the above view by indicating that the current trivalent vaccine offers a postvaccination titre to A/Chile/1/83 of 160 or higher in less than 50% of adult vaccinees and appeared to offer no protection against the new variants.

RECOMMENDATIONS FOR 1986-1987 INFLUENZA VACCINE

Based on the currently available reports, WHO has suggested that national health authorities in the Northern Hemisphere consider the addition of the new type A(H1N1) virus to other strains already recommended for incorporation in 1986-1987 influenza vaccines, either as an extra component in current trivalent vaccines or as a monovalent vaccine.

1. WHO recommended vaccine[4]:

WHO recommended that influenza vaccines for use in the 1986-1987 season should be trivalent and contains the following antigens:

- an A/Christchurch/4/85(H3N2) - A/Mississippi/1/85(H3N2) -like antigen
- an A/Chile/1/83(H1N1) - A/Singapore/6/86(H1N1)-like antigen and
- a B/Ann/Arbor/1/86-like antigen.

2. CDC recommended vaccine[5]:

In the United States, the vaccine production schedule permits the manufacture only of a supplemental monovalent vaccine with a new type A (H1N1) virus. Hence vaccination against influenza for the 1986-87 winter season in the U.S.A. consists of the administration of the current trivalent vaccine (containing 15 ug each of A/Chile/1/83(H1N1), A/Mississippi/1/85(H3N2), and B/Ann/Arbor/1/86 haemagglutinin antigens in each 0.5 ml, manufactured by Parks-Davis) and a monovalent vaccine (containing 15 ug A/Taiwan/1/86 (H1N1)-like antigen). The use of antiviral agents including amantadine hydrochloride is also recommended for the prevention and treatment of influenza.

3. CDR recommended influenza vaccine for the U.K. 1986-87 winter season[6]

The antigemic composition of the influenza vaccine for the coming winter is as follows (as published in CDR 86/40 - 3 October 1986):

A/Christchurch/4/85 (H3N2) or a similar strain
A/Chile/1/83 (H1N1)
B/Ann/Arbor/1/86 or a similar strain.

The above composition does not currently reflect the WHO recommendations to include a new type A(H1N1) (eg A/Singapore/6/86-like antigen) in the 1986-87 - influenza vaccine for the Northern Hemisphere. A review of the need for a new separate monovalent vaccine (A/Singapore/6/86 - H1N1) is being considered. A decision is likely to be made by joint committee of vaccination and immunisation in the very near future.

4. AUSTRALIA: Recommendation for the 1987 influenza vaccine

In the 1986 winter season, Australia recorded the lowest number of cases for the past two decades. A total of only 55 cases of influenza A have been reported to the CDI since January 1986 which included 7 isolates of type A(H1N1). The WHO influenza reference centre at CSL (Melbourne) reported that only 20 cases of type A(H1N1) have been isolated in Victoria including 7 isolates of a variant strain of influenza type A(H1N1).

Despite the prevalence of new variant strain of type A (H1N1) (eg A/Singapore/6/86, A/Taiwan/1/86) in European and Asian Countries of the Northern Hemisphere in the 2nd quarter of 1986, no influenza epidemic has been observed in Australia or New Zealand in the winter months of 1986.

Historically, new variants of influenza strain in the Northern Hemisphere did not cause significant epidemics in countries of the Southern Hemisphere until one or two years have elapsed as evidenced by the 1920's and 1950's epidemics in Australia.

Although no evidence of an epidemic caused by new strains of influenza type A(H1N1) (eg A/Singapore/6/86, A/Taiwan/1/86, A/Beijing/1/86, A/Hong Kong/7/86) is yet apparent, it may be prudent for Australia to consider a change in the components making up the antigenic composition of the 1987 influenza vaccine.

The antigenic composition of the current trivalent vaccine available from CSL is as follows:

. an A/Victoria/3/85 (H3N2)-like antigen
. an A/Chile/1/83 (H1N1)-like antigen
. an B/Victoria/3/83-like antigen.

The antigenic composition of a 1987 trivalent vaccine for Australia should consider incorporating the following components:

. an A/Victoria/3/85 (H3N2)-like antigen
. an A/Chile/1/83 (H1N1) - A/Singapore/6/86 (H1N1)-like antigen
. an B/Victoria/3/83-like antigen.

Based on the available reports the above antigenic composition for the 1987 influenza vaccine is only suggested and will not be fully determined until the WHO recommendation for a 1987 influenza vaccine for the Southern Hemisphere (to be published in WER of 29 October 1986) is fully considered.

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2. CSL correspondence to CDI
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4. WER (1986) 61: 237-8
5. MMWR (1986) 35: 517-21
6. CDR (1986) 40: 3-4

HEPATITIS B IN A DENTAL PRACTICE

In September 1984, the health authorities in a small town (population, 16 500) in rural Indiana became aware of four cases of hepatitis B that had occurred in the town since July 1.[1] Such a cluster was unusual for the town and surrounding county (population, 35 000) since there had only been one case reported there in the previous decade.

All of the cases had attended the same dental practice. The dentist concerned became suspicious of his connection with the outbreak and submitted himself for hepatitis B surface antigen (HBsAg) testing; the result was reported positive on September 18 and he immediately discontinued his practice.

An investigation was consequently conducted by the Indiana State Board of Health and the Hepatitis Branch of the Division of Viral Diseases, Centers for Disease Control (the preliminary findings were reported in CDI 85/6). Eight cases were identified by October 1; all had become ill since March 1, 1984. Two patients had died of fulminant hepatitis B and a third was partially paralysed from mononeuritis multiplex due to polyarteritis nodosa, a complication of acute hepatitis B. Two more cases occurred in November and January. Nine of the ten reported cases had been treated by the dentist 2-5 months before the onset of illness.

Using a case definition based on anti-hepatitis B core (anti-HBc) IgM antibody positivity and exposure to the dentist during a defined time period, a serological survey of the dentist's patients identified 15 asymptomatic cases in a total of 1 123 serum specimens. The overall attack rate was 3.2% for the patients treated in the period March 1 to September 18.

Excluding a patient who worked in a physician's office, and a patient who was married to a male bisexual hepatitis B carrier, none of the cases had significant risk factors for hepatitis B. No other case family members were found to be hepatitis B positive. All of the dentist's office staff and his family were negative for hepatitis B serological markers. None of the 24 cases of hepatitis B infection had any other exposure likely to transmit the virus.

In general, the infection risk was related to the amount of trauma involved in the cases' dental procedures. The unusual lethality of the outbreak is unexplained. The patients who died had no other illness that would have contributed to their deaths from fulminant hepatitis. None of the patients, including those who died, were exposed to identifiable hepatoxins. No other agents which may have acted as cofactors, including other viruses, were identified in any of the hepatitis B cases.

Although the dentist involved in the outbreak had never had symptoms of hepatitis, his serum was positive for HBsAg and hepatitis B e antigen and negative for anti-HBc IgM antibody. Such findings are consistent with the dentist being a hepatitis B carrier.

All of the instruments used by the dentist were thoroughly cleaned and then autoclaved. The dentist did not wear gloves when treating patients except when the patient's mouth was purulent. He scrubbed his hands with a surgical brush and iodophor soap for 1-2 minutes before and after seeing each patient (approximately 30-40 times per day). He denied any hand lacerations, abrasions or rashes, although microlesions from vigorous hand scrubbing may have been involved.[2]

The dentist had no risk factors for hepatitis B other than those associated with a dental practice. It is therefore likely that his infection was acquired from a patient. However, the source of infection was not identified among the patients tested in the serological survey.

Interestingly, on April 1 and May 1, 1984, the dentist had received the first and second doses of hepatitis B vaccine. However, he did not undergo serological screening prior to receiving the vaccine, nor had he ever been tested for hepatitis B prior to the outbreak.

CDI EDITORIAL COMMENT

The One Hundred and First Session of the NH&MRC (June 1986) endorsed the document 'Guidelines for the Dental Treatment of Patients with Infectious Diseases' and recommended that it should be made widely available to members of the dental profession. This document describes procedures for the prevention of cross-infection of viral diseases in dental practice.

Hepatitis B vaccine is recommended for susceptible persons who are at substantial risk of hepatitis B infection, including dentists and their clinical staff.

The hepatitis B vaccine currently available in Australia (H-B-Vax; Merck Sharp & Dohme) is an inactivated vaccine prepared from virus particles purified from the plasma of human hepatitis B carriers. Initial fears about the safety of the vaccine, particularly with regard to the potential transmission of hepatitis or AIDS and the possible induction of autoimmune reactions, have been shown to be unfounded.[3-7]

The current adult immunisation regimen consists of three 1 mL (20 µg) doses of vaccine given intramuscularly (three 0.5 mL doses for those aged 6 months to 10 years). The second injection is given 1 month after, and the third injection 6 months after, the initial injection.

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HEPATITIS B IN HOUSEHOLD CONTACTS OF HAEMOPHILIACS

The risks of haemophiliacs contracting hepatitis as a result of the infusions of large numbers of plasma products prepared from multiple blood transfusions have been well defined.[1-4] However, relatively little information is available on the incidence of hepatitis in the household contacts of patients with haemophilia. A recent study in Texas examined the frequency of hepatitis B in close family contacts of patients with congenital blood coagulation disorders.[5]

Family members of 49 patients with severe or moderately severe haemophilia were tested for hepatitis B markers and had their serum levels of alanine aminotransferase (ALT) measured. A total of 78 participating family members, all of whom resided in the immediate household of their relative with haemophilia, were tested. These included 56 parents, 16 siblings, five spouses and one son of the haemophilia patients. Thirty-one of the parents (of 20 patients) participated in a home infusion program. Eight of the 49 patients were chronic hepatitis B virus carriers (HBsAg - positive, anti - HBs - negative) and seven of these were hepatitis e antigen-positive. The other patients were HBsAg - negative and anti - HBs - positive, the antibodies to the virus being the result of either natural infection or active immunisation with hepatitis B vaccine. Six (43%) of 14 household contacts of the eight HBsAg - positive patients had evidence of prior hepatitis B infection. Three of five infusing parents and two of three siblings were anti - HBs - positive; another sibling was also HBsAg - positive.

Two (3%) of the 64 family members (including 26 from home-infusing parents) of the 41 HBsAg - negative patients were found to be positive for hepatitis B markers. Only one of these positive results was likely to be related to their relative's haemophilia; a home-infusing mother of an anti -

HBs - positive teenage patient was anti - HBs - positive as a consequence of an apparent subclinical infection. The second family member with evidence of a prior hepatitis B infection had icteric hepatitis as a child and had been excluded as a blood donor because of hepatitis B antigen positivity.

Of the 78 household contacts of the haemophilia patients, six (8%) had elevated serum ALT levels; only one having a value in excess of 60 IU/L. One patient's mother, not involved in giving home infusions, had an ALT value of 142 IU/L, her husband's ALT activity also being slightly elevated at the same time (48 IU/L). On repeat testing of the mother 6 months later, her ALT activity had returned to normal.

Although hepatitis B was infrequent among family members of HBsAg - negative patients with haemophilia, a much higher rate of hepatitis B (43%) was observed in household contacts of the patients who were HBsAg - positive. This observation is consistent with earlier studies in other patient groups describing a high rate (15-50%) of intrafamilial transmission of hepatitis B, particularly from one young child to another, when a member of the family is HBsAg - positive.[6, 7]

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5. J Pediatr (1986) 108: 937-939

6. Am J Epidemiol (1984); 120: 617-25

7. J Pediatr (1975); 87: 753-56

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 13/10/86 - 26/10/86 Bulletin number 86/22

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	7	3	1	1		1	12	1	26
0101 ADENOVIRUS TYPE 1	1								1
0102 ADENOVIRUS TYPE 2	1			1		1			3
0103 ADENOVIRUS TYPE 3	1					1			2
0105 ADENOVIRUS TYPE 5	1			1		2		1	5
0106 ADENOVIRUS TYPE 6						1			1
0107 ADENOVIRUS TYPE 7	1								1
0108 ADENOVIRUS TYPE 8	1								1
0199 ADENOVIRUS TYPING PENDING					3				3
0201 INFLUENZA A VIRUS	6			1			4		11
0202 INFLUENZA A VIRUS SUBTYPE H3N2					1				1
0203 INFLUENZA B VIRUS	2					1	1		4
0206 INFLUENZA A VIRUS SUBTYPE H1N1				1					1
0301 PARAINFLUENZA VIRUS TYPE 1	1						1		2
0302 PARAINFLUENZA VIRUS TYPE 2	1					1			2
0303 PARAINFLUENZA VIRUS TYPE 3	6	1		1	4	6	2	2	22
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	2		1	8	13	15	30	12	81
0500 RHINOVIRUS (ALL TYPES)				1	11	10			22
0600 MYCOPLASMA PNEUMONIAE	7	1		2			8	7	25
0700 ORNITHOSIS-PSITTACOSIS	3			4		1			8
0809 COXSACKIEVIRUS A9								1	1
0816 COXSACKIEVIRUS A16						1			1
0902 COXSACKIEVIRUS B2				1					1
1003 ECHOVIRUS TYPE 3				1					1
1005 ECHOVIRUS TYPE 5				2					2
1011 ECHOVIRUS TYPE 11				2				6	8
1020 ECHOVIRUS TYPE 20	1								1
1026 ECHOVIRUS TYPE 26						1			1
1102 POLIOVIRUS TYPE 2						2			2
1200 MUMPS VIRUS				1				2	3
1300 HERPES VIRUS GROUP-NOT TYPED	25			3				5	33
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1						1	2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	12	2		4			24	8	50
1303 VARICELLA-ZOSTER VIRUS	2	1		1			2	3	9
1306 HERPES SIMPLEX TYPE 1	11			52		17	37	33	150
1307 HERPES SIMPLEX TYPE 2	56			54		16	70	71	267
1399 HERPES VIRUS TYPING PENDING	4				1				5
1401 COXIELLA BURNETI	8					2	8		18
1502 PICORNA VIRUS-NOT TYPED	6	1	3				11	2	23
1514 MOLLUSCUM CONTAGIOSUM								1	1
1521 MEASLES VIRUS		1		3			2		6
1522 RUBELLA VIRUS	1						35	3	39
1530 HEPATITIS A VIRUS		1							1
1532 HEPATITIS B ANTIGEN	50	3	6	16	1	17	25	15	133
1535 HEPATITIS A ANTIBODY			3			17	3	14	37
1541 CHLAMYDIA A - C TRACHOMATIS	40	1				41	29	61	172
1543 CHLAMYDIA A - LGV TYPE							10		10
1556 CMV - CYTOMEGALOVIRUS	2	1	2	7	4	4	9	9	38
1563 CORONAVIRUS								1	1
1564 ROTAVIRUS	12	6	4		2	16	64		104
1565 CALICI VIRUS	1								1
1571 ENTEROVIRUS TYPE 71 (BRCR)	3	1		1		1			6
1599 ENTEROVIRUS TYPING PENDING		3	6		12				21
9992 ROSS RIVER VIRUS								1	1
9998 ARBO. GROUP B. ...							1		1
Total	275	27	26	169	52	175	388	260	1,372

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 13/10/86 - 26/10/86

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

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir-atory	Enceph-alitis	Mening-itis	Para-lysis	CNS other unspec	GI	Hepa-tic	CVS	Urin-ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1		1									
0102 ADENOVIRUS TYPE 2		1					2				
0103 ADENOVIRUS TYPE 3							1				
0105 ADENOVIRUS TYPE 5	1	2					1				
0106 ADENOVIRUS TYPE 6		1									
0107 ADENOVIRUS TYPE 7		1									
0201 INFLUENZA A VIRUS		6							1		
0202 INFLUENZA A VIRUS SUBTYPE H3N2		1									
0203 INFLUENZA B VIRUS		3									
0206 INFLUENZA A VIRUS SUBTYPE H1N1		1									
0301 PARAINFLUENZA VIRUS TYPE 1		2									
0302 PARAINFLUENZA VIRUS TYPE 2		2									
0303 PARAINFLUENZA VIRUS TYPE 3	1	21									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	2	77					3				1
0500 RHINOVIRUS (ALL TYPES)		1									
0600 MYCOPLASMA PNEUMONIAE	1	15						1			1
0700 ORNITHOSIS-PSITTACOSIS	3	3									
0809 COXSACKIEVIRUS A9											1
0816 COXSACKIEVIRUS A16											1
1003 ECHOVIRUS TYPE 3		1									
1005 ECHOVIRUS TYPE 5					1						
1011 ECHOVIRUS TYPE 11		2			3	1	1				
1020 ECHOVIRUS TYPE 20							1				
1026 ECHOVIRUS TYPE 26					1						
1200 MUMPS VIRUS					1						
1300 HERPES VIRUS GROUP-NOT TYPED											1
1301 HERPES SIMPLEX VIRUS NOT-TYPED								1			1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	8	6			1			2			1
1303 VARICELLA-ZOSTER VIRUS	2										7
1306 HERPES SIMPLEX TYPE 1	4	5	1		1					2	84
1307 HERPES SIMPLEX TYPE 2	7									2	9
1401 COXIELLA BURNETI	4	3						1	2		
1502 PICORNA VIRUS-NOT TYPED	2										
1521 MEASLES VIRUS		1	1			1					2
1522 RUBELLA VIRUS	4	3					1				31
1530 HEPATITIS A VIRUS								1			
1532 HEPATITIS B ANTIGEN	50					1		73		1	
1535 HEPATITIS A ANTIBODY	7							27			
1541 CHLAMYDIA A - C.TRACHOMATIS	23	1					2				
1543 CHLAMYDIA A - LGV TYPE	4										
1556 CMV - CYTOMEGALOVIRUS	2	11				1				3	
1563 CORONAVIRUS		1									
1564 ROTAVIRUS	2	3					96				
1565 CALICI VIRUS							1				
1571 ENTEROVIRUS TYPE 71 (BRCR)	2	1		2						1	1
9992 ROSS RIVER VIRUS											1
9998 ARBO. GROUP B.	1										
Total	130	176	2	10		4	109	106	3	9	227

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 13/10/86 - 26/10/86

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
0103 ADENOVIRUS TYPE 3.....	1									
0105 ADENOVIRUS TYPE 5.....							1			
0108 ADENOVIRUS TYPE 8.....	1									
0201 INFLUENZA A VIRUS.....			1				1	3	1	
0203 INFLUENZA B VIRUS.....								1	1	
0301 PARAINFLUENZA VIRUS TYPE 1....								1		
0303 PARAINFLUENZA VIRUS TYPE 3....								2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	1									
0600 MYCOPLASMA PNEUMONIAE.....	1		1		1			6	3	
0700 ORNITHOSIS-PSITTACOSIS.....								1	1	
0902 COXSACKIEVIRUS B2.....								1		
1005 ECHOVIRUS TYPE 5.....								1		
1011 ECHOVIRUS TYPE 11.....								1		
1102 POLIOVIRUS TYPE 2.....									1	1
1200 MUMPS VIRUS.....			1					1		
1300 HERPES VIRUS GROUP-NOT TYPED..		2								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			17	3	1		1	17	2	
1306 HERPES SIMPLEX TYPE 1.....	1	49							4	
1307 HERPES SIMPLEX TYPE 2.....		166								
1401 COXIELLA BURNETI.....					2		1	6	1	
1521 MEASLES VIRUS.....								1		
1522 RUBELLA VIRUS.....				1	10	1		11	1	
1532 HEPATITIS B ANTIGEN.....								1	8	
1535 HEPATITIS A ANTIBODY.....									3	
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	138							6	
1543 CHLAMYDIA A - LGV TYPE.....	1	5								
1556 CMV - CYTOMEGALOVIRUS.....		2	4	1		4	1	3	8	1
1564 ROTAVIRUS.....								4	1	1
9992 ROSS RIVER VIRUS.....					1					
Total.....	7	362	24	5	15	5	5	61	41	3

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

eriod 5 - 19 April 1986 to 16 May 1986 Bulletin.....86/22.

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	5					6	22
Ankylostomiasis			1	2	1		NN		4	* 9
Anthrax									–	–
Arbovirus infection	74	13	205		13				305	811
Brucellosis	1		2						3	10
Campylobacter infections	55		NN	65	3	NN	6	NN	129	* 923
Chancroid			1	NN					1	4
Cholera									–	–
Congenital rubella syndrome			NN			NN		NN	–	–
Diphtheria							6		6	14
Donovanosis			9	NN	11		4		24	48
Giardiasis	29		NN	59	14	NN	NN	NN	102	487
Genital herpes	57		31	21	NN	NN	2	1	112	525
Gonococcal ophthalmia neonatorum		NN			NN	NN		NN	–	–
Gonorrhoea	139		89	51	180	2	52	7	520	1 978
Hepatitis A (infectious)	11	5	13	39	55				123	* 710
Hepatitis B (serum)	30	19	47	6	31		5	7	145	* 722
Hepatitis – unspecified	2		4	1	NN	NN	2		9	64
Hydatid disease	1			1					2	6
Lassa fever			NN			NN		NN	–	–
Legionnaires disease	1		NN		1	NN		NN	2	34
Leprosy		1			2				3	7
Leptospirosis	1		18						19	84
Lymphogranuloma venereum				NN	NN	NN		NN	–	2
Marburg disease			NN			NN		NN	–	–
Malaria	18	5	24	1	5			4	57	* 288
									–	–
Meningococcal infections		1			1	NN			2	14

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	402		NN	38	NN	NN	NN	NN	440	1 931
Ornithosis									-	* 16
Pertussis (whooping cough)	8	6	NN	3	1	NN	2	NN	20	* 361
Plague									-	-
Poliomyelitis									-	-
Q. fever	1		15	1	1				18	* 100
Rabies				NN		NN		NN	-	-
Salmonella infections	76	18	41	26	24	3	43	3	234	1 233
Shigella infections	14	2	5	5	10	1	29		66	375
Smallpox									-	-
Syphilis	35		58	9	34		105	1	242	885
Tetanus									-	3
Trachoma		NN	1		29	NN	NN		30	35
Tuberculosis (all forms)	21	31	2	6	15	6	3	1	85	361
Typhoid fever					1				1	15
Typhus (all forms)	1								1	* 9
Vibrio parahaemolyticus infections			NN			NN		NN	-	4
Yellow fever									-	-
Yersinia infections	9		NN	1		NN		NN	10	42

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.)

* Adjustments to the Cumulative Total since last report:

Ankylostomiasis	+1	South Australia	Pertussis	-3	South Australia
Campylobacter inf.	-1	Western Australia	Q fever	+2	South Australia
Hepatitis A	+1	South Australia	Typhus (all forms)	+6	New South Wales
Hepatitis B	+1	South Australia			
Malaria	+1	South Australia			
Ornithosis	+1	South Australia			