ISSN 0725-3141

Communicable Diseases Intelligence

Bulletin number 85/10
Issue date: 17 May 1985

Contents:

VIRUS REPORTING SCHEME A total of 798 reports were processed for this period. No reports were received from the State Health Laboratory, Brisbane as Queensland mail has been delayed by the current industrial dispute. Patterns suggested by the reports include the first indications of the seasonal rise in respiratory syncytial virus (61 reports compared with 38, 22 and 23 for the previous three periods). The 17 parainfluenzavirus type 2 cases reported by the State Health Laboratory Services (SHLS), Perth, were predominantly in young children presenting with croup.

- Three arbovirus group B infections were reported by Fairfield Hospital, Melbourne, in patients resident in Nauru. All cases had encephalitic symptoms with serum titres rising from <20 to 320. No information on patient age, sex, or any other clinical details was available.

- Hepatitis B antigen (HBsAg) was reported by the SHLS, Perth, in an Asian businessman who presented with arthritis and gastrointestinal symptoms. The patient had arrived recently from Singapore where he had received hepatitis B vaccine without screening. Hepatitis B screening prior to vaccination is warranted in ethnic groups with a high prevalence of hepatitis B virus markers. However, it should be noted that vaccination in those who are HBsAg positive produces no adverse effects (Am. Int. Med. (1982) 96:575-9).

- Ross River virus infections were reported in Victoria (9 cases, 8 being retrospective reports from January), Western Australia (10) and Northern Territory (1).

Other reports of interest include:-

- An out-of-season influenza type B strain, designated B/Victoria/2/85 which closely resembles B/USSR/100/83, was isolated by the OIC WHO Influenza Reference Centre, Commonwealth Serum Laboratories, Melbourne. The patient was a 22 year old female who presented at a clinic in north Melbourne with mild but typically influenzal symptoms.

The Bulletin is compiled and distributed by the Environmental Health Branch, Department of Health, P.O. Box 100, Woden, A.C.T. 2606, Australia, and is available on request.
Contributions are solicited, and do not preclude later publication elsewhere.

Figures given may be subject to revision.

POLYSACCHARIDE VACCINE FOR PREVENTION OF HAEMOPHILUS INFLUENZAE TYPE B DISEASE

(Based on MMWR (1985) 34: 201-5)

A polysaccharide vaccine against invasive (bacteraemic) disease caused by Haemophilus influenzae type b recently has been licensed in the USA.

Haemophilus influenzae disease

H. influenzae is a leading cause of serious systemic bacterial disease in the United States. It is the most common cause of bacterial meningitis, accounting for an estimated 12,000 cases annually, primarily among children under five years of age. The mortality rate is 5%, and neurological sequelae are observed in as many as 25-35% of survivors. Virtually all cases of H. influenzae meningitis among children are caused by strains of type b (Hib), although this capsular type represents only one of the six types known for this species. In addition to bacterial meningitis, Hib is responsible for other invasive diseases, including epiglottitis, sepsis, cellulitis, septic arthritis, osteomyelitis, pericarditis and pneumonia. Non-typeable (non-capsulated) strains of H. influenzae commonly colonise the human respiratory tract and are a major cause of otitis media and respiratory mucosal infection but rarely result in bacteraemic disease. Hib strains account for only 5-10% of H. influenzae causing otitis media.

Several population-based studies of invasive Hib disease conducted within the last ten years have provided estimates of the incidence of disease among children under five years of age, the major age group at risk. These studies have demonstrated attack rates of meningitis ranging from 51 cases per 100,000 children to 77/100,000 per year and attack rates of other invasive Hib disease varying from 24/100,000 to 75/100,000 per year.(1) Thus, in the United States, approximately one of every 1,000 children under five years of age develops systemic Hib disease each year, and a child's cumulative risk of developing systemic Hib disease at some time during the first five years of life is about one in 200. Attack rates peak between six months and one year of age and decline thereafter. Approximately 35-40% of Hib disease occurs among children 18 months of age or older, and 25% occurs above 24 months of age.

Incidence rates of Hib disease are increased in certain high-risk groups such as Native Americans (both American Indians and Eskimos), blacks, individuals of lower socioeconomic status and patients with asplenia, sickle cell disease, Hodgkin's disease and antibody deficiency syndromes. Recent studies also have suggested that the risk of acquiring primary Hib disease for children under five years of age appears to be greater for those who attend day-care facilities than for those who do not(2,3).

The potential for person-to-person transmission of systemic Hib disease among susceptible individuals has been recognized in the past decade. Studies of secondary spread of Hib disease in household contacts of index patients have shown a substantially increased risk of disease among exposed household contacts under four years of age(4). In addition, numerous clusters of cases in day-care facilities have been reported, and recent studies suggest that secondary attack rates in day-care classroom contacts of a primary case also may be increased(5,6).

Haemophilus b polysaccharide vaccine

The Hib vaccine is composed of the purified, capsular polysaccharide of H. influenzae type b ([→3] ribose-β1→1 ribitol-1 phosphate-5→). Antibodies to this antigen correlate with protection against invasive disease. The Hib vaccine induces an antibody response that is directly related to the age of the recipient; infants respond infrequently and with less antibody than do older children or adults[7]. Improved responses are observed by 18 months of age, although children 18-23 months of age do not respond as well as those two years of age or older. The frequency and magnitude of antibody responses reach adult levels at about six years of age[8,9]. Levels of antibodies to the capsular polysaccharide also decline more rapidly in immunised infants and young children than in adults.

In a manner similar to other polysaccharide antigens, revaccination with Hib vaccine results in a level of antibody comparable to that for a child of the same age receiving a first immunisation[10]. Such polysaccharide antigens have been termed "T-cell independent" because of their failure to induce the T-cell memory responses characteristic of protein antigens.

Limited data are available on the response to Hib vaccine in high-risk groups with underlying disease. By analogy to pneumococcal vaccine, patients with sickle cell disease or asplenia are likely to exhibit an immune response to the Hib vaccine. Patients with malignancies associated with immunosuppression appear to respond less well. Additional data on the immune response to Hib vaccine in these groups are needed.

A precise protective level of antibody has not been established. However, based on evidence from passive protection in the infant rat model and from experience with agammaglobulinaemic children, an antibody concentration of 0.15 µg/ml correlates with protection[7,8,11]. In the Finnish field trial, levels of capsular antibody greater than 1 µg/ml in 3-week postimmunisation sera correlated with clinical protection for a minimum of 1 1/2 years[9,12,13]. Approximately 75% of children 18-23 months of age tested achieved a level greater than 1 µg/ml, as did 90% of 24-35 month old children[9]. Measurement of Hib antibody levels is not routinely available, however, and determination of antibody levels following vaccination is not indicated in the usual clinical setting.

Effectiveness of vaccine

In 1974, a randomised, controlled trial of clinical efficacy was conducted in Finland among children 3-71 months of age[9]. Approximately 98,000 children, half of whom received the Hib vaccine, were enrolled in the field trial and followed for a four-year period for occurrence of Hib disease. Among children 18-71 months of age, 90% protective efficacy (95% confidence limits, 55%-98%) in prevention of all forms of invasive Hib disease was demonstrated for the four-year follow-up period. Although no disease occurred among over 4,000 children 18-23 months of age immunised with Hib vaccine and followed for four years, only two cases occurred in the control vaccine recipients in this age group. As a result, vaccine efficacy in the subgroup of children immunised at

18-23 months of age could not be evaluated statistically. The vaccine was not efficacious in children under 18 months of age.

Revaccination

Limited data regarding the potential need for revaccination are available at present. Current data show that children who have received the Hib vaccine 2-42 months previously have an immune response to the vaccine similar to that in previously unvaccinated children of the same age. No immunological tolerance or impairment of immune response to a subsequent dose of vaccine occurs[10]. As with other polysaccharide vaccines, the shorter persistence of serum antibodies in young children given Hib vaccine, compared with adults, suggests that a second dose of vaccine may be needed to maintain immunity throughout the period of risk, particularly for children in the youngest age group considered for vaccination (those 18-23 months of age). A second injection following the initial dose is likely to increase the protective benefit of vaccination for this high-risk group, because antibody titres 18 months after vaccination, although detectable in most vaccine recipients, are no longer significantly different from those in unvaccinated children of the same age.

Recommendations for vaccine use

Recently published data regarding vaccine efficacy and the risk of Hib disease among young children strongly support the use of Hib vaccine in the United States in high-risk persons for whom efficacy has been established. Specific recommendations are as follows:

1. Immunisation of all children at 24 months of age is recommended. The precise duration of immunity conferred by a single dose of Hib vaccine at 24 months of age is not known, although, based on available data, protection is expected to last 1 1/2-3 1/2 years. Until further data are available to determine whether an additional dose of vaccine may be necessary to ensure long-lasting immunity, routine revaccination is not recommended.

2. Immunisation of children at 18 months of age, particularly those in known high-risk groups, may be considered. Although the precise efficacy of the vaccine among children 18-23 months of age is not known, this age group accounts for approximately 12% of all invasive Hib disease among children under 5 years of age, and Hib vaccine has been shown by serological methods to be immunogenic in most children of this age group. However, physicians and parents should be informed that the vaccine is not likely to be as effective in this age group as in older children. These younger children may need a second dose of vaccine within 18 months following the initial dose to ensure protection. Additional data regarding the duration of the antibody response are needed to define the timing of a second dose more precisely.

   Children who attend day-care facilities are at particular risk of acquiring systemic Hib disease. Initial vaccination at 18 months of age for this high-risk group should be considered.

   Children with chronic conditions known to be associated with increased risk for Hib disease should receive the vaccine, although only limited data on immunogenicity and

clinical efficacy in this group are available. These conditions include anatomical or functional asplenia, such as sickle cell disease or splenectomy[14], and malignancies associated with immunosuppression[15].

3. Immunisation of individuals over 24 months of age who have not yet received Hib vaccine should be based on risk of disease. The risk of invasive Hib disease decreases with increasing age over the age of two years. Because the vaccine is safe and effective, however, physicians may wish to immunise previously unvaccinated healthy children between two years and five years of age to prevent the Hib disease that does occur in this age group. The potential benefit of this strategy in terms of cases prevented declines with increasing age of the child at the time of vaccination. Therefore, children two-three years of age who attend day-care facilities should be given a higher priority than day-care attendees who are four-five years old.

4. Insufficient data are available on which to base a recommendation concerning use of the vaccine in older children and adults with the chronic conditions associated with an increased risk of Hib disease.

5. Vaccine is not recommended for children under 18 months of age.

6. Simultaneous administration of Hib and DTP vaccines at separate sites can be performed, because no impairment of the immune response to the individual antigens occurs under these circumstances.

Side effects and adverse reactions

Polysaccharide vaccines are among the safest of all vaccine products. To date, over 60,000 doses of the Hib polysaccharide vaccine have been administered to infants and children, and several hundred does have been given to adults[9,16]. Only one serious systemic reaction has been reported thus far - a possible anaphylactic reaction that responded promptly to epinephrine. High fever (38.5°C[101.3°F] or higher) has been reported in fewer than 1% of Hib vaccine recipients. Mild local and febrile reactions were common, occurring in as many as half of vaccinated individuals in the Finnish trial. Such reactions appeared within 24 hours and rapidly subsided. Current preparations appear to result in fewer such local reactions. Simultaneous administration with DTP does not result in reaction rates above those expected with separate administration[17].

Precautions and contraindications

The Hib vaccine is unlikely to be of substantial benefit in preventing the occurrence of secondary cases, because children under two years old are at highest risk of secondary disease. Because the vaccine will not protect against nontypeable strains of H. influenzae, recurrent upper respiratory diseases, including otitis media and sinusitis, are not considered indications for vaccination.

New Vaccine Development

New vaccines, such as the Hib polysaccharide-protein conjugate vaccines, are being developed and evaluated and may prove to be efficacious for children under 18 months of age.

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Q FEVER OUTBREAK IN SWITZERLAND

(Based on WER (1985) 60:121-2)

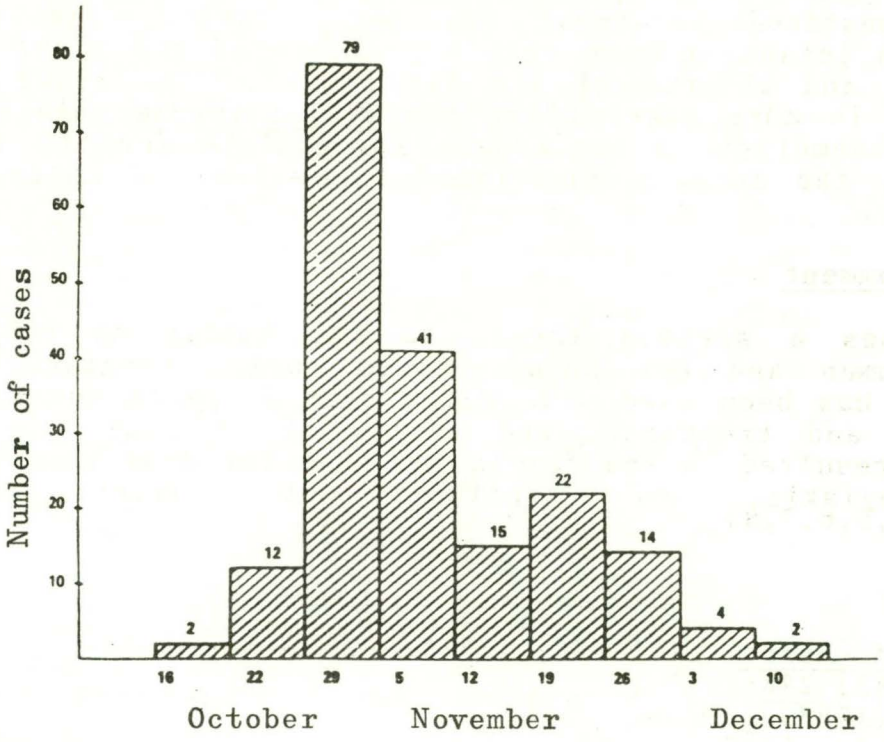
In Autumn 1983, a sizeable outbreak of Q fever in the Valais (val de Bagnes, population 4,652), Switzerland, was probably caused by sheep flocks returning from the mountain pastures on and after 8 October. Three weeks after these animals had passed through the villages, the outbreak suddenly started. Serum analysis of 8 patients admitted to the Martigny Hospital with bronchopneumonia led to the discovery of this outbreak.

Among the 415 cases of Q fever diagnosed on this occasion, 191 (46%) were highly symptomatic and consulted a physician. The date of onset of illness was accurately determined for these 191 patients and is shown in Fig. 1. It may be noted that most of these patients fell ill on or after 29 October. The other 224 cases were persons who had not consulted a physician and were diagnosed on systematic analysis of 3,000 serum samples taken from the population of the valley.

The percentage of positive cases was far higher (21.1%) among the inhabitants of the villages situated directly along the roads on which the sheep had travelled, than among the inhabitants of villages located away from their route (2.9%). In all, 13.7% of the valley population tested suffered from Q fever during this outbreak.

Serum specimens were obtained from 432 sheep; 166 had Coxiella burnetii antibodies.

Fig. 1. Q fever: date of onset of 191 symptomatic cases, Bagnes (Valais), Switzerland, autumn 1983.



Editorial Comment

Q fever is a worldwide zoonosis caused by C. burnetii. This infectious agent may be transmitted to animals by ticks, and is generally passed to other animals and to man by aerosols. Infectious abortion in animals would appear to play an important role in this. No such abortions had been noted among the sheep at the end of spring. It is presumed that the fleece of the sheep was contaminated by C. burnetii and that this was the true cause of the outbreak.

C. burnetii infection in man takes the form of an influenza-like illness, with coughing, fever, headaches, myalgia, asthenia and anorexia. Attenuated forms may pass unnoticed. Complications such as pneumonia and hepatitis may appear in some cases. Myocarditis or glomerulonephritis are noted less frequently. In this particular outbreak 2% of those infected were seriously ill (pneumonia) and had to be admitted to hospital. Among the 191 persons reporting sick, there were also more than 60 cases with radiologically well-documented bronchopneumonia.

Abattoir-associated Q fever became a public health problem in Australia in the late 1970s and early 1980s when abbatoirs started to process feral goats, many of which were pregnant, for export. This situation has prompted the development of an Australian Q fever vaccine which has given encouraging results in early trials (see CDI (1984) 84/12:2).

PLASMODIUM FALCIPARUM MALARIA DURING PREGNANCY - CHLOROQUINE TERATOGENIC EFFECTS?

The safety of malaria prophylaxis with chloroquine during pregnancy has recently been questioned[1]. A 30 year old woman developed Plasmodium falciparum malaria in the first trimester of pregnancy whilst taking chloroquine for malaria

prophylaxis. Her illness was characterized by a haemolytic anaemia, with IgG1 detected on erythrocyte surfaces and IgG3 in the serum. The anaemia resolved early in the third trimester following unstated treatment for the malaria. The mother delivered an infant at term with hypoplasia of the right tibia and fibula, and absence of the fifth ray of the right foot. Although it is more likely that the P. falciparum malaria and associated haemolytic anaemia were responsible for the foetal abnormality, the authors questioned the safety of chloroquine in pregnancy.

Editorial Comment

Malaria poses a serious threat to the health of both the pregnant woman and her unborn child during pregnancy[2,3]. Chloroquine has been used widely for over 30 years for malaria prophylaxis and treatment, and review of its use over this period has resulted in the conclusion that the drug is safe for the prophylaxis and treatment of malaria during pregnancy[4,5,6,7,8].

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PRINTED INFORMATION AVAILABLE ON AIDS

The following publications are being sent to all registered medical practitioners in Australia by the Commonwealth Department of Health:

. "Infection Control Guidelines", revised March 1985.
. "Facts on AIDS", December 1984.
. "Basic Program on AIDS".

Also available are cartoon booklets entitled:

. "AIDS - What everyone should know".
. "AIDS - Information for gay and bisexual men".
. "AIDS - Information for health care workers".

These and the documents cited above may be obtained through the following State and Territory health authorities:-

. New South Wales - All regional offices.

. Victoria - Health Promotion Unit,
555 Collins Street,
Melbourne. VIC. 3000
(Phone 03-616 7185)

. Queensland (except cartoon booklets) - Health Promotion Unit,
9 Costin Street,
Fortitude Valley. QLD. 4006
(Phone 07-854 1144)

. Western Australia - Special Clinic,
69 Moore Street,
Perth. W.A. 6000
(Phone 09-325 6466)

. South Australia - Communicable Diseases Unit,
158 Rundle Mall,
Adelaide. S.A. 5000
(Phone 08-218 3445)

. Tasmania - Health Education,
34 Davey Road,
Hobart. TAS. 7000
(Phone 002-30 3652)

. Northern Territory - Health Department,
P.O. Box 1701
Darwin. N.T. 5794
(Phone 089-80 2911)

. Australian Capital Territory - Health Promotion Unit
A.C.T. Health Authority,
Moore Street,
Canberra City. A.C.T. 2601
(Phone 062-45 4537)

ANNOUNCEMENT - International Union Against Venereal Diseases and Treponematoses (IUVDT) - South East Asia and Western Pacific Region Conference and Fourth Regional Meeting is to be held in Bombay, India, on 18-20 October 1985.

Erratum

In CDI 85/7, page 6, an incorrect titre was given in the Table summarising the immunodiffusion data for Aspergillus species. The "A. fumigatus negative, at least one other species positive" result should read 191/688 (28%).

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 25/4/85 - 8/5/85 BULLETIN NUMBER 85/10

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ WVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR-FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED			2		3	2		1	8
0101 ADENOVIRUS TYPE 1				1		6			7
0102 ADENOVIRUS TYPE 2						3		1	4
0103 ADENOVIRUS TYPE 3				1				1	2
0105 ADENOVIRUS TYPE 5						1		2	3
0108 ADENOVIRUS TYPE 8				2					2
0137 ADENOVIRUS TYPE 37				6					6
0199 ADENOVIRUS TYPING PENDING		1	1		3				5
0201 INFLUENZA A VIRUS	3				1	1		1	6
0202 INFLUENZA A VIRUS SUBTYPE H3N2				1	1				2
0203 INFLUENZA B VIRUS	1		2						3
0301 PARAINFLUENZA VIRUS TYPE 1	1			1		5			7
0302 PARAINFLUENZA VIRUS TYPE 2						1		17	18
0303 PARAINFLUENZA VIRUS TYPE 3					2	1			3
0399 PARAINFLUENZA VIRUS TYPING PENDING					3				3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	24	17	1	2	11	3		3	61
0500 RHINOVIRUS (ALL TYPES)				3	12			2	17
0600 MYCOPLASMA PNEUMONIAE	1							3	4
0700 ORNITHOSIS-PSITTACOSIS				2					2
0800 COXSACKIEVIRUSES GROUP A - NOT TYPED						2			2
0816 COXSACKIEVIRUS A16				1					1
0904 COXSACKIEVIRUS B4						1			1
1002 ECHOVIRUS TYPE 2								1	1
1003 ECHOVIRUS TYPE 3								1	1
1005 ECHOVIRUS TYPE 5								1	1
1007 ECHOVIRUS TYPE 7		1		4	1			1	7
1009 ECHOVIRUS TYPE 9								2	2
1021 ECHOVIRUS TYPE 21				2				1	3
1022 ECHOVIRUS TYPE 22				1					1
1030 ECHOVIRUS TYPE 30				1					1
1099 ECHOVIRUS TYPING PENDING				1					1
1100 POLIOVIRUS NOT TYPED			1		5				6
1101 POLIOVIRUS TYPE 1						1			1
1102 POLIOVIRUS TYPE 2						1			1
1103 POLIOVIRUS TYPE 3						2			2
1200 MUMPS VIRUS	1			7					8
1300 HERPES VIRUS GROUP-NOT TYPED				5		3		5	13
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1		1					2
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	9							8	17
1303 VARICELLA-ZOSTER VIRUS	2	1	1	1				2	7
1306 HERPES SIMPLEX TYPE 1			7	28		5		23	63
1307 HERPES SIMPLEX TYPE 2	1		17	37		10		48	113
1399 HERPES VIRUS TYPING PENDING				1	3	2			6
1401 COXIELLA BURNETI				1					1
1502 PICORNA VIRUS-NOT TYPED	5		10						15
1514 MOLLUSCUM CONTAGIOSUM						1			1
1521 MEASLES VIRUS	1			1					2
1522 RUBELLA VIRUS	6			6		1			13
1532 HEPATITIS B ANTIGEN	53	1	9	18		21		24	126
1535 HEPATITIS A ANTIBODY	1	1		3		8		4	17
1541 CHLAMYDIA A - C TRACHOMATIS	36		7					55	98
1556 CMV - CYTOMEGALOVIRUS	5	4		20	6	5		13	53
1564 ROTAVIRUS			2		1	7			10
1599 ENTEROVIRUS TYPING PENDING			8		7				15
9992 ROSS RIVER VIRUS				8				12	20
9998 ARBO. GROUP B.				3					3
Total	150	27	68	169	59	93		232	798

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 25/4/85 to 8/5/85 ....
Viral Identifications by Clinical Information Table 1.
Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Encephalitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.; 07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0100 ADENOVIRUS NOT TYPED.....		1									
0101 ADENOVIRUS TYPE 1.....		4									
0102 ADENOVIRUS TYPE 2.....		2					1				
0103 ADENOVIRUS TYPE 3.....		1									
0105 ADENOVIRUS TYPE 5.....		2					1				
0201 INFLUENZA A VIRUS.....	1	4									
0202 INFLUENZA A VIRUS SUBTYPE H3N2		2									
0301 PARAINFLUENZA VIRUS TYPE 1....		6					1				
0302 PARAINFLUENZA VIRUS TYPE 2....	1	16									
0303 PARAINFLUENZA VIRUS TYPE 3....		3									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		61									
0500 RHINOVIRUS (ALL TYPES).....		13									
0600 MYCOPLASMA PNEUMONIAE.....		3									1
0700 ORNITHOSIS-PSITTACOSIS.....		2									
0816 COXSACKIEVIRUS A16.....											1
0904 COXSACKIEVIRUS B4.....		1									
1002 ECHOVIRUS TYPE 2.....				1							
1003 ECHOVIRUS TYPE 3.....				1							
1005 ECHOVIRUS TYPE 5.....											1
1007 ECHOVIRUS TYPE 7.....		1		5							
1009 ECHOVIRUS TYPE 9.....							2				
1021 ECHOVIRUS TYPE 21.....				2							
1022 ECHOVIRUS TYPE 22.....		1									
1030 ECHOVIRUS TYPE 30.....				1							
1101 POLIOVIRUS TYPE 1.....							1				
1102 POLIOVIRUS TYPE 2.....							1				
1103 POLIOVIRUS TYPE 3.....	1										
1200 MUMPS VIRUS.....				4							
1300 HERPES VIRUS GROUP-NOT TYPED..											2
1301 HERPES SIMPLEX VIRUS NOT-TYPED											2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	5	1						2			
1303 VARICELLA-ZOSTER VIRUS.....						1					5
1306 HERPES SIMPLEX TYPE 1.....	3	5	1	1				1		1	26
1307 HERPES SIMPLEX TYPE 2.....	4										41
1401 COXIELLA BURNETI.....									1		
1502 PICORNA VIRUS-NOT TYPED.....							1				
1521 MEASLES VIRUS.....		1									2
1522 RUBELLA VIRUS.....											10
1532 HEPATITIS B ANTIGEN.....	71							31			1
1535 HEPATITIS A ANTIBODY.....								16			
1541 CHLAMYDIA A - C.TRACHOMATIS...	1										
1556 CMV - CYTOMEGALOVIRUS.....	3	9	1			1	1	2	1	5	
1564 ROTAVIRUS.....	2	1					7				
1599 ENTEROVIRUS TYPING PENDING....							1				
9992 ROSS RIVER VIRUS.....											6
9998 ARBO. GROUP B.			3								
Total.....	92	140	5	15		2	17	52	2	6	98

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 25/4/85 to 8/5/85 ...
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					1			1
0102 ADENOVIRUS TYPE 2.....							1			
0103 ADENOVIRUS TYPE 3.....								1		
0108 ADENOVIRUS TYPE 8.....	2									
0137 ADENOVIRUS TYPE 37.....	6									
0201 INFLUENZA A VIRUS.....							1			
0202 INFLUENZA A VIRUS SUBTYPE H3N2								1		
0203 INFLUENZA B VIRUS.....					1			1	1	
0301 PARAINFLUENZA VIRUS TYPE 1....								1		
0302 PARAINFLUENZA VIRUS TYPE 2....						1				
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1		
0500 RHINOVIRUS (ALL TYPES).....								2	1	1
0600 MYCOPLASMA PNEUMONIAE.....							1	1		
1007 ECHOVIRUS TYPE 7.....									1	
1021 ECHOVIRUS TYPE 21.....						1				
1103 POLIOVIRUS TYPE 3.....										1
1200 MUMPS VIRUS.....			6							
1300 HERPES VIRUS GROUP-NOT TYPED..									1	
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			2	2				3	2	
1303 VARICELLA-ZOSTER VIRUS.....									1	
1306 HERPES SIMPLEX TYPE 1.....	1	19					2	1	4	
1307 HERPES SIMPLEX TYPE 2.....		68							1	
1514 MOLLUSCUM CONTAGIOSUM.....		1								
1522 RUBELLA VIRUS.....					4	1	1		1	
1532 HEPATITIS B ANTIGEN.....					1				22	
1541 CHLAMYDIA A - C.TRACHOMATIS...	2	95								
1556 CMV - CYTOMEGALOVIRUS.....		6	1	1		3	4	1	14	1
1564 ROTAVIRUS.....									1	
9992 ROSS RIVER VIRUS.....					19					
Total.....	11	190	9	3	25	6	11	14	50	4

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

(1 January 1984 to 31 December 1984)
as at 10 May 1985

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	TOTAL
Amoebiasis	13		4	20	7		1	1	46
Ankylostomiasis	2		5	66	2				75
Anthrax									—
Arbovirus infection	805	161	472	136	2			1	1577
Brucellosis	5	1	7		1		1		15
Campylobacter infections	554	N.N.	N.N.	1226	N.N.	N.N.	19	N.N.	1799
Chancroid	5		2	N.N.	6	N.N.	1		14
Cholera									—
Congenital rubella syndrome	1	N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	1
Diphtheria									—
Donovanosis		N.N.	42	N.N.	105	N.N.	54		201
Giardiasis	279	N.N.	N.N.	746	N.N.	N.N.	N.N.		1025
Genital herpes	790	N.N.	429	87	N.N.	N.N.	18	6	1330
Gonococcal ophthalmia neonatorum		N.N.		1	N.N.	N.N.	8	N.N.	9
Gonorrhoea	2685	1533	1518	726	1434	42	827	129	8894
Hepatitis A (infectious)	131	140	252	68	38	10	22	13	674
Hepatitis B (serum)	522	188	442	199	155	10	20	23	1559
Hepatitis - unspecified	74	10		15	28	N.N.	7		134
Hydatid disease	5		1		1			2	9
Lassa Fever	N.N.		N.N.			N.N.	N.N.	N.N.	—
Legionnaires disease	7	4	N.N.	2	N.N.	N.N.	N.N.	N.N.	13
Leprosy	7	5	5	3	2		4	2	28
Leptospirosis	46	36	118	14	8	5			227
Lymphogranuloma venereum	1	N.N.	N.N.	N.N.	N.N.	N.N.	1		2
Malaria	113	66	330	54	34	9	15	19	640
Marburg Disease	N.N.		N.N.			N.N.	N.N.	N.N.	—
Meningococcal infections	18	7		24	6	N.N.	3	1	59
Non-specific urethritis	3635	N.N.	N.N.	1186	N.N.	N.N.	17	N.N.	4838
Ornithosis		7	2	22	10			1	42
Pertussis (whooping cough)	117	47	N.N.	96	N.N.	N.N.	1	N.N.	261
Plague									—
Poliomyelitis									—
Q. fever	108	9	131	14	N.N.				262
Rabies	N.N.	N.N.	N.N.			N.N.	N.N.	N.N.	—

DISEASE	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	TOTAL
Salmonella infections	659	170	337	346	113	79	355	33	2092
Shigella infections	115	20	64	38	55	2	125	1	420
Smallpox									—
Syphilis	1489	174	358	127	204	2	952	17	3323
Tetanus	4		1	2					7
Trachoma	N.N.	N.N.			N.N.	N.N.	4		4
Tuberculosis (all forms)	510	298	177	82	134	11	65	22	1299
Typhoid fever	28	7	12		2			1	50
Typhus (all forms)	1		6	1					8
Vibrio parahaemolyticus infections	8	N.N.	N.N.	1	N.N.	N.N.	N.N.	N.N.	9
Yellow Fever									—
Yersinia enterocolitica infections	7	N.N.	N.N.	1	N.N.	N.N.	N.N.	N.N.	8

(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.)