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Communicable Diseases Intelligence

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Contents:

VIRUS REPORTING SCHEME - A total of 1229 reports were processed this period. There is no evidence of significant respiratory or gastrointestinal virus infections at present.

- Thirteen echovirus type 7 infections were notified during this period, eight in patients with meningitis and six in infants less than one year of age. The virus was also isolated from the faeces of a 30 year old mother whose baby died from a generalised echovirus type 7 infection. Echovirus type 7 has been the most frequently reported enterovirus this summer, with most initial reports coming from Queensland (see CDI 85/2). Ten reports of echovirus type 7 were received in 1984, compared with 110 and 64 reports of echovirus type 9 and 6 respectively. Coxsackievirus B4 infections (9 cases; 7 in young children) were reported during this period by the Institute of Medical and Veterinary Science, Adelaide. By contrast, coxsackievirus B5 has been the prevalent serotype in the past six months.
- Ross River virus infections were reported from Queensland (22), Western Australia (11), New South Wales (4), Northern Territory (3), and Victoria (1). The State Health Laboratory Services, Perth, have recently adapted the fluorescent antibody technique for the detection of Ross River virus IgM antibody to circumvent the problem of excessive numbers of sucrose density centrifugation separations. The new technique involves the fixation with acetone of Vero cell monolayers on slides infected with the T-48 prototype virus, and incubation with the appropriate test antisera preabsorbed with streptococcal suspension and Ig G gel. Following washing, the preparation is exposed to fluorescein-conjugated antihuman immunoglobulin.

ANNOUNCEMENT:- SAFETY PROCEDURES IN MICROBIOLOGY LABORATORIES
(Contributed by M. Kennett, Fairfield Hospital, Melbourne)

Since the development of recombinant DNA techniques and the emergence of previously unknown viruses such as those associated with African Haemorrhagic fevers and AIDS, laboratory workers have become more aware of the need for the safe handling of microbial agents.

The World Health Organization has set up a Programme on Safety Measures in Microbiology and several collaborating centres have

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HTLV-III ANTIBODY TESTS IN BLOOD DONORS - MANY FALSE POSITIVES EXPECTED

(Based on Colorado Disease Bulletin (1985) Vol. No. XIII, Issue No. 6)

What does a positive HTLV-III antibody test in a healthy blood donor mean?

Definitions:

Predictive Value Positive (PVP) = Probability that a person with a positive test truly has antibody or A/(A+B).
Predictive Value Negative (PVN) = Probability that a person with a negative test truly does not have antibody or D/(C+D).

The PVP and PVN are very important in interpreting test results in populations with different prevalences of infection. In populations with a low prevalence of infection, a positive test result is a poorer predictor of true infection than in populations with a high prevalence of infection. As a result, populations with a low prevalence of infection will have a higher rate of false positives.

	Test Positive	Test Negative	Test Positive	Test Negative	
Truly have Antibody	A	C	97	3	100
Don't have Antibody	B	D	999	98,901	99,900
		Total	1,096	98,904	100,000

A hypothetical situation

Consider a hypothetical population of 100,000 persons being screened with the HTLV-III antibody test. We assume the test is 97% sensitive and 99% specific. The true prevalence of antibody in this population is 0.1% (i.e., 0.1% of the population have antibody), similar to that which might be the case in a self-screened healthy donor population.

Of our total 100,000 persons, only 0.1% or 100 persons have antibody and the remaining 99,900 do not. Since the test is 97% sensitive, it will correctly identify 97 of these 100 persons (Cell A) as having antibody (True Positives). The remaining 3% (Cell C) will be missed (False Negatives). Since the test is 99% specific, it will correctly identify 99% of the 99,900 antibody-negative persons (Cell D or True Negatives), but will falsely identify the remaining 1% of antibody-negative persons as being positive (999 persons in Cell B, False Positives).

The PVP then, is A/(A+B) = 97/(97 + 999) = 8.9%. This is much lower than it would be in a high prevalence population. The % of the population with positive test results, (A + B)/ 100,000 is 1.1%, or 11 times higher than the actual prevalence of antibody in the population.

If we look only at the persons who were false positives on the first test, those persons have a 0.0% prevalence of antibody (i.e., none were True Positives). Given a specificity of 99%, the probability of these persons having an initial reactive test is 1% (1 chance in 100 or .01). The probability that the same person will have two reactive tests is .01 x .01 = .0001, or 1 chance in 10,000, if the two tests results are independent

(i.e., if there is no repeatable cause of a false positive, such as a cross-reactive antibody, that influences both tests).

What will happen in Colorado?

There is as yet limited data on potential repeatable causes of false positives. Early data from blood bank trials suggest that 20 to 30% of persons who test positive on the first ELISA will be positive for antibody on a second test. It also seems very likely that there will be "biological false positives" with this test just as there are with the VDRL and FTA for syphilis. With approximately 130,000 persons giving blood each year in Colorado and from 0.2 to 0.6% having two positive ELISA tests, we could expect from 260 to 780 blood donors per year to be notified and need further evaluation; of these, only a minority will truly have antibody to HTLV-III. The risk of AIDS in antibody positive healthy blood donors who are not part of a high-risk group is not known, but is probably low.

Editorial Comment

It is proposed in Australia that persons with a positive ELISA test will have a confirmatory test (Western Blot) performed at a State/Territory reference laboratory. Any blood donation which is ELISA positive will not be used.

EPIDEMIC MENINGOCOCCAL DISEASE: RECOMMENDATIONS FOR TRAVELLERS TO NEPAL

(Based on MMWR (1985) 34 : 119-20)

During the first six months of 1983, an epidemic of serogroup A meningococcal meningitis, resulting in 875 cases and 95 deaths, occurred in the Kathmandu valley of Nepal. The overall annual attack rate was 103 cases per 100,000 population; the case-fatality ratio was 11%. The highest age-specific attack rate (223/100,000) was for children under one year of age; 83% of the cases occurred among persons under 25 years of age. The epidemic peaked in May and ended in June, coincident with the onset of the rainy season. No vaccination efforts were undertaken.

During December 1983 and January 1984, three times as many cases occurred in Kathmandu as in the same period a year previously; a mass vaccination campaign was initiated on 8 February, 1984. The target population included persons 1-24 years of age living in the Kathmandu valley. Three hundred and thirty thousand doses of bivalent A/C meningococcal vaccine were given, achieving approximately 65% coverage of the target population. A dramatic decline in the number of meningitis cases occurred coincident with the initiation of the mass vaccination campaign.

Surveillance in 1985 indicates that meningococcal meningitis is occurring at a much lower rate than in 1984. However, meningococcal disease among hikers is now being recognized. Between January 1984 and January 1985, two culture-confirmed and four clinically suspected cases of meningococcal disease have been documented among tourists from western countries travelling in Nepal. Three of these occurred between January and April 1984, and three occurred between November 1984 and January 1985. Patients' ages ranged from 16 years to 40 years (mean 27 years). Five patients had evidence of meningococcaemia; the other had meningitis alone. Two (33%) died. All became ill during or shortly after hiking outside Kathmandu. The patients' countries of origin were the United States (three patients), Australia (two), and Switzerland (one).

Epidemic meningococcal disease has not been reported previously in Nepal, and, except for Mongolia and Vietnam, has been reported in no other Asian country [1,2]. Large epidemics of group A meningococcal disease have occurred primarily in the "meningitis belt" of Africa, which consists of the semi-arid Sahelian zone south of the Sahara [3]. In Africa, epidemics have been cyclic, occurring every 10-12 years and lasting 2-3 years. The epidemics occur during the dry season and stop when the rains begin. The seasonal pattern of disease seen in Nepal is similar to that observed in Africa, although Nepal's dry season is cold rather than hot.

Meningococcal polysaccharide vaccines have been used to control epidemics in the past [4-6]. In the immunization campaign in the Kathmandu valley, the target population was selected to cover the age group in which 75% of the cases were occurring. Although at high risk, children under one year of age were not vaccinated because the vaccine is poorly immunogenic in that age group.

Based on the 1983 tourist statistics, approximately 105,000 non-Asian tourists visit Nepal each year. Thus, the attack rate for such tourists during the past year was 6/100,000. Based on the number of hiking permits issued (32,298), the attack rate for hikers was 19/100,000. By comparison, the annual incidence of meningococcal disease in this age group in the United States is approximately 0.3/100,000 [7]. The relative increase in risk is even more striking when the average length of stay for tourists in Nepal (11 days) is taken into account, since the attack rate for the disease in the United States for 11 days would be only .009/100,000. Because of the risk of meningococcal disease among hikers, CDC recommends that tourists planning to hike in Nepal receive meningococcal vaccine. Although all cases of meningococcal disease to date have occurred in hikers, it is prudent for other travellers to Nepal to receive the vaccine also.

The serogroup A meningococcal vaccine has a clinical efficacy of 85%-95% for at least 1 year, with protection achieved 1-2 weeks after vaccination. Adverse reactions are limited to local erythema or soreness.

Editorial Comment

A small epidemic of meningococcal meningitis has also been reported in January 1985, in Delhi, India[8]. There were approximately 2 000 cases up to the end of March 1985, with a case-fatality rate of less than 10%. The cases were mostly from low socioeconomic groups and were not concentrated in any one particular area of the city but were spread out. Most cases occurred in the 5-25-year age group, and about 9% of the cases were under 4 years of age. The meningococci were isolated in a number of cases and were found to belong serologically to group A. The epidemic showed a peak in mid-March and is now declining. The case-fatality rate has also dropped to below 4%. The Indian Government has laid great emphasis on health education and chemoprophylaxis for immediate contacts. The carrier rate is being monitored by the Indian Council of Medical Research. A/C vaccine has been provided for high-risk groups. All strains were found to be sensitive to sulphonamides and penicillin.

A bivalent A/C meningococcal vaccine is currently being evaluated by the Australian Commonwealth Department of Health. This vaccine is available only on an Individual Patient Use (IPU) basis as it is an unevaluated product. Application for

IPU approval should be made through Dr H. Arora, Commonwealth Department of Health, P.O. Box 100, Woden, A.C.T. 2606 (telephone - 062-898575).

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KAWASAKI SYNDROME

(Based on MMWR (1985) 34 : 33-35; British Columbia Disease Surveillance (1984) 5 : 277-279; and California Morbidity (1985) No.7)

Kawasaki syndrome (KS), previously known as mucocutaneous lymph node syndrome, is a rare illness first described by a Japanese paediatrician in 1967[1]. Its aetiology remains unknown in spite of many investigations. Transmission from one person to another has not been demonstrated. It primarily affects children under six years of age, being slightly more frequent in males and appearing to occur more frequently in winter and spring.

Ten outbreaks of KS were recently reported in the USA, between 22 August 1984 - 6 January 1985, with 187 cases satisfying the diagnostic criteria for the syndrome and 75 suspected cases being reported to CDC, Atlanta[2].

Diagnostic criteria include fever lasting five or more days without other more reasonable explanation and at least four of the following:

(i) bilateral conjunctival injection;
(ii) at least one of the following mucous-membrane changes - injected or fissured lips, pharyngeal injection and/or "strawberry" tongue;
(iii) at least one of the following extremity changes - erythema of palms or soles, oedema of the hands or feet, and/or generalised periungual desquamation;
(iv) rash;
(v) cervical lymphadenopathy (at least one lymph node 1.5 cm or greater in diameter).

Unfortunately, 17-31% of children with KS develop coronary artery aneurysms with a case fatality ratio of 1-2%[3]. In the above outbreaks sixty-two (33%) of 186 patients had cardiovascular complications. Coronary artery aneurysms were reported in 37 (20%) cases; one resulted in myocardial infarction and two were associated with pericarditis. Because coronary artery aneurysms are often not detected until 2-8 weeks after onset of KS, the number with this complication may increase. Myocarditis was reported in 12 patients, including one associated with a cardiac arrest and another with myocardial infarction and pericarditis. Eight additional patients had pericarditis. One child had recurrent angina episodes with a normal cardiac catheterisation study, two had peripheral vascular complications resulting in gangrene and requiring amputations. One child had a stroke and one had transient hemiparesis. Non-cardiovascular KS complications reported included sterile pyuria/meatitis (14 cases), hydrops of the gallbladder (eight), hepatitis (six), arthritis (six), aseptic meningitis (four), uveitis (two), small bowel

obstruction (one) and profound anaemia requiring transfusion (one).

Laboratory findings associated with this illness are non-specific. Typically, neutrophilic leukocytosis, thrombocytosis and an elevated sedimentation rate can be detected.

Outbreaks of KS have also been described in Canada[4], Japan[5] and Australia[6]. An association with house dust mites (Dermatophagoides),[7] and the application of rug shampoo in the home in the 30 days before onset of the disease[8] has been suggested. However, the significance of these hypotheses is not clear.[9,10] In British Columbia the syndrome seems to have been more prevalent in the dry areas of the province than in the humid areas.[9]

The efficacy of a single therapeutic regimen has not been well established although one study has suggested that the administration of aspirin during the acute phase[11], and another, that the administration of intravenous high-dose gammaglobulin during the acute phase[12] may reduce the frequency of coronary artery aneurysms. A multicentre study to evaluate the potential efficacy of high-dose intravenous gammaglobulin therapy is currently under way in the USA. Corticosteroids are contraindicated in this syndrome as they have been associated with an increased rate of coronary artery aneurysm formation.

It is hoped that with increasing awareness more cases of KS will be recognised, leading to a better understanding of the illness, and the CDI is interested in hearing of any recent Australian cases or outbreaks of the disease.

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(continued from page 1).

already been established. The co-ordinator of this programme has recently visited Australia and it is likely that a local Collaborating Centre for Bio-Safety in Australia will be established within the next year.

As part of this role, it is intended to gauge interest in forming an association in Australia of people involved in safety aspects, particularly in microbiology. The most likely people would be biological safety officers, institutional biosafety committee members, hospital laboratory workers, research scientists and people from other disciplines involved in recombinant DNA work.

In this regard it has been proposed to form a special interest group of the Australian Society of Microbiology (ASM) and arrange meetings in conjunction with the Annual General Meeting of ASM. The organising committee of the 1985 Perth ASM meeting have set aside one hour - 1pm on Tuesday 14th May (venue not yet determined)-for interested members to discuss the formation of such a group.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 11/4/85 — 24/4/85 BULLETIN NUMBER 85/9
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	10	1	1		2		7	3	24
0101 ADENOVIRUS TYPE 1				1		3		1	5
0102 ADENOVIRUS TYPE 2	2			2		2			6
0103 ADENOVIRUS TYPE 3				2				2	4
0105 ADENOVIRUS TYPE 5				1		1			2
0106 ADENOVIRUS TYPE 6	2					1			3
0108 ADENOVIRUS TYPE 8	2								2
0109 ADENOVIRUS TYPE 9				1					1
0111 ADENOVIRUS TYPE 11				1					1
0137 ADENOVIRUS TYPE 37				1					1
0199 ADENOVIRUS TYPING PENDING			2		3	3			8
0201 INFLUENZA A VIRUS	2				1			1	4
0203 INFLUENZA B VIRUS	2		1					1	4
0301 PARAINFLUENZA VIRUS TYPE 1					3	14			17
0302 PARAINFLUENZA VIRUS TYPE 2				1	2	2	1	12	18
0303 PARAINFLUENZA VIRUS TYPE 3					3	1	1	1	6
0399 PARAINFLUENZA VIRUS TYPING PENDING				1			2		3
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	20	10		1	3		3	1	38
0500 RHINOVIRUS (ALL TYPES)	3			1	4	6	3	6	23
0600 MYCOPLASMA PNEUMONIAE	2						1	1	4
0700 ORNITHOSIS-PSITTACOSIS	2								2
0809 COXSACKIEVIRUS A9	1								1
0901 COXSACKIEVIRUS B1	1								1
0904 COXSACKIEVIRUS B4	1					9			10
0905 COXSACKIEVIRUS B5	1		1						2
1005 ECHOVIRUS TYPE 5								3	3
1006 ECHOVIRUS TYPE 6								1	1
1007 ECHOVIRUS TYPE 7	2	4	2	5					13
1014 ECHOVIRUS TYPE 14				1					1
1015 ECHOVIRUS TYPE 15		1							1
1021 ECHOVIRUS TYPE 21				1					1
1022 ECHOVIRUS TYPE 22				1					1
1030 ECHOVIRUS TYPE 30				1				1	2
1033 ECHOVIRUS TYPE 33				1					1
1101 POLIOVIRUS TYPE 1	1								1
1200 MUMPS VIRUS				2			1		3
1300 HERPES VIRUS GROUP-NOT TYPED	25		1	5		1		2	34
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1		3			1		5
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	13	5		4	2	1		4	29
1303 VARICELLA-ZOSTER VIRUS	7			1		1	2	1	12
1306 HERPES SIMPLEX TYPE 1	24		7	23		12	32	19	117
1307 HERPES SIMPLEX TYPE 2	118		13	34		16	61	41	283
1399 HERPES VIRUS TYPING PENDING					4				4
1401 COXIELLA BURNETI	3			2		3	1		9
1502 PICORNA VIRUS-NOT TYPED	8		7				17	1	33
1521 MEASLES VIRUS	4								4
1522 RUBELLA VIRUS	4	1					1		6
1531 HEPATITIS B VIRUS	2								2
1532 HEPATITIS B ANTIGEN	109		11	27	2	17	8	15	189
1533 HEPATITIS B ANTIBODY	10								10
1534 HEPATITIS B ANTIGEN AND ANTIBODY	1								1
1535 HEPATITIS A ANTIBODY	11	1	1	1		2	2	3	21
1541 CHLAMYDIA A - C TRACHOMATIS	41						23	35	99
1556 CMV - CYTOMEGALOVIRUS	19	1		14	6	5		13	58
1564 ROTAVIRUS	17	3			4	2		1	27
1599 ENTEROVIRUS TYPING PENDING		1	17		5				23
9992 ROSS RIVER VIRUS			1	1			26	12	40
9993 ASTROVIRUS	1								1
9994 SMALL VIRUS (LIKE) PARTICLE	3							1	4
Total	474	29	65	140	44	102	193	182	1,229

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 11/4/85 to 24/4/85 ....
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	Respiratory	Encephalitis	Meningitis	Paralysis	CNS other unspec	GI	Hepatic	CVS	Urinary	Skin/mucs memb
0100 ADENOVIRUS NOT TYPED.....		1									
0101 ADENOVIRUS TYPE 1.....	1	3				1					
0102 ADENOVIRUS TYPE 2.....		3					1				1
0103 ADENOVIRUS TYPE 3.....		1									
0105 ADENOVIRUS TYPE 5.....		2									
0106 ADENOVIRUS TYPE 6.....							2				
0201 INFLUENZA A VIRUS.....		3					1		1		
0203 INFLUENZA B VIRUS.....		3									
0301 PARAINFLUENZA VIRUS TYPE 1....		17									
0302 PARAINFLUENZA VIRUS TYPE 2....		15				1					
0303 PARAINFLUENZA VIRUS TYPE 3....		6									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	3	35									
0500 RHINOVIRUS (ALL TYPES).....	1	19						1			
0600 MYCOPLASMA PNEUMONIAE.....		3									
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0809 COXSACKIEVIRUS A9.....					1						
0901 COXSACKIEVIRUS B1.....					1						
0904 COXSACKIEVIRUS B4.....	1	3					5				
0905 COXSACKIEVIRUS B5.....					2						
1005 ECHOVIRUS TYPE 5.....					1		1				
1006 ECHOVIRUS TYPE 6.....		1									
1007 ECHOVIRUS TYPE 7.....	1	1			8		1				
1014 ECHOVIRUS TYPE 14.....		1									1
1015 ECHOVIRUS TYPE 15.....					1						
1021 ECHOVIRUS TYPE 21.....							1				
1022 ECHOVIRUS TYPE 22.....		1									
1030 ECHOVIRUS TYPE 30.....					1						
1033 ECHOVIRUS TYPE 33.....					1						
1101 POLIOVIRUS TYPE 1.....		1									
1200 MUMPS VIRUS.....		1			2						
1301 HERPES SIMPLEX VIRUS NOT-TYPED											4
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	7	1						2			
1303 VARICELLA-ZOSTER VIRUS.....	2				1						10
1306 HERPES SIMPLEX TYPE 1.....	7	8									47
1307 HERPES SIMPLEX TYPE 2.....	10					1					
1401 COXIELLA BURNETI.....	3	2									
1502 PICORNA VIRUS-NOT TYPED.....							1				
1521 MEASLES VIRUS.....	2										2
1522 RUBELLA VIRUS.....											5
1531 HEPATITIS B VIRUS.....	1										
1532 HEPATITIS B ANTIGEN.....	116							55	1		
1533 HEPATITIS B ANTIBODY.....	2							3			
1534 HEPATITIS B ANTIGEN AND ANTIBODY.....								1			
1535 HEPATITIS A ANTIBODY.....	1							19			
1541 CHLAMYDIA A - C.TRACHOMATIS...		1									
1556 CMV - CYTOMEGALOVIRUS.....	6	10				1		3	2	2	2
1564 ROTAVIRUS.....	3	1					21				
9992 ROSS RIVER VIRUS.....	7	2									13
9993 ASTROVIRUS.....							1				
9994 SMALL VIRUS (LIKE) PARTICLE...	1						2				
Total.....	175	146		19		4	37	84	4	2	131

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 11/4/85 to 24/4/85 ...
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		
0103 ADENOVIRUS TYPE 3.....	2						1			
0106 ADENOVIRUS TYPE 6.....	1									
0108 ADENOVIRUS TYPE 8.....	2									
0109 ADENOVIRUS TYPE 9.....	1									
0111 ADENOVIRUS TYPE 11.....									1	
0137 ADENOVIRUS TYPE 37.....	1									
0203 INFLUENZA B VIRUS.....								1		
0302 PARAINFLUENZA VIRUS TYPE 2....			1				1			
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0500 RHINOVIRUS (ALL TYPES).....							3			
0600 MYCOPLASMA PNEUMONIAE.....							1			
0700 ORNITHOSIS-PSITTACOSIS.....							1			
0809 COXSACKIEVIRUS A9.....								1		
0904 COXSACKIEVIRUS B4.....							1			
1005 ECHOVIRUS TYPE 5.....							1			
1007 ECHOVIRUS TYPE 7.....								2	1	
1030 ECHOVIRUS TYPE 30.....							1			
1200 MUMPS VIRUS.....			1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED		1								
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			10	1			3	1	7	
1303 VARICELLA-ZOSTER VIRUS.....		1								
1306 HERPES SIMPLEX TYPE 1.....	5	46		1			1	2	1	
1307 HERPES SIMPLEX TYPE 2.....		228					1	1	1	
1401 COXIELLA BURNETI.....							2	2		
1521 MEASLES VIRUS.....							1			
1522 RUBELLA VIRUS.....					1				1	
1531 HEPATITIS B VIRUS.....					1					
1532 HEPATITIS B ANTIGEN.....		1			1		2		13	
1533 HEPATITIS B ANTIBODY.....								1	4	
1534 HEPATITIS B ANTIGEN AND ANTIBODY.....									1	
1535 HEPATITIS A ANTIBODY.....		1						1		
1541 CHLAMYDIA A - C.TRACHOMATIS...	1	96				1				
1556 CMV - CYTOMEGALOVIRUS.....		9	1			4	3	7	9	1
1564 ROTAVIRUS.....									3	
9992 ROSS RIVER VIRUS.....					30			3	2	
9994 SMALL VIRUS (LIKE) PARTICLE...									1	
Total.....	13	383	13	2	33	5	23	24	45	1

CDI 85/9

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

(Weeks 1 -4)
1 - 25 January 1985

Bulletin ..85/9..

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		1	1	2			1	6	6
Ankylostomiasis				3					3	3
Anthrax									—	—
Arbovirus infection	3								3	3
Brucellosis									—	—
Campylobacter infections	49	N.N.	N.N.	91	N.N.	N.N.	N.N.	N.N.	140	140
Chancroid				N.N.		N.N.			—	—
Cholera									—	—
Congenital rubella syndrome		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—
Diphtheria									—	—
Donovanosis		N.N.		N.N.	3	N.N.	2		5	5
Giardiasis	18	N.N.	N.N.	51	N.N.	N.N.	N.N.	N.N.	69	69
Genital herpes	140	N.N.	44	23	N.N.	N.N.	3	3	213	213
Gonococcal ophthalmia neonatorum		N.N.			N.N.	N.N.		N.N.	—	—
Gonorrhoea	154	145	89	47	217	6	26	20	704	704
Hepatitis A (infectious)	7	7	23	5	6	1			49	49
Hepatitis B (serum)	29	6	16	14	21	1	2	1	90	90
Hepatitis - unspecified	7					N.N.	N.N.		7	7
Hydatid disease									—	—
Lassa Fever			N.N.			N.N.	N.N.	N.N.	—	—
Legionnaires disease			N.N.		N.N.	N.N.	N.N.	N.N.	—	—
Leprosy				1					1	1
Leptospirosis	5	9		1	1	1			17	17
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.			—	—
Malaria	18	6	62	4	1			1	92	92
Marburg Disease			N.N.			N.N.	N.N.	N.N.	—	—
Meningococcal infections			8			N.N.			8	8
Non-specific urethritis	246	N.N.	N.N.	113	N.N.	N.N.		N.N.	359	359
Ornithosis				1					1	1
Pertussis (whooping cough)	27	6	N.N.	17	N.N.	N.N.	N.N.	N.N.	50	50
Plague									—	—
Poliomyelitis									—	—
Q. fever	3			2	N.N.		N.N.		5	5
Rabies		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	216	12	49	37	19	6	12	4	355	355
Shigella infections	20		11	5	2		8		46	46
Smallpox									—	—
Syphilis	48	4	10	5	55		29	2	153	153
Tetanus			1						1	1
Trachoma		N.N.	1		N.N.	N.N.			1	1
Tuberculosis (all forms)	5	13	6	5	11		3	5	48	48
Typhoid fever	2								2	2
Typhus (all forms)									—	—
Vibrio parahaemolyticus infections	2	N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	2	2
Yellow Fever									—	—
Yersinia enterocolitica infections		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	—

(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