AUSTRALIA

Communicable Diseases Intelligence

Bulletin number 81/4

Issue date: 27 February 1981

Contents:

In an effort to contain postage costs, the CDI mailing list is being updated. We would be grateful to hear of readers who receive more copies than needed, or of readers from institutions who could receive individual copies under a single cover to the institution with internal distribution from that point. Corrections of addresses would also be appreciated; where possible we would prefer to send CDI to a position rather than to present occupant, who might move.

VIRUS REPORTING SCHEME - A total of 793 reports were received this period.

Reports of interest include:

- Influenza - Influenza A virus (H_1N_1) resembling A/USSR/90/77 was isolated from a 24 year old female with typical influenza symptoms by the State Health Laboratory, Brisbane. The strain has been sent to the WHO Reference Centre, Melbourne, for verification. The patient's husband was employed at a food processing plant that was closed down the previous week because 20 of 74 employees had severe respiratory illnesses. No acute specimens suitable for virus isolation were collected from the workers.

  An influenza C virus infection was serologically diagnosed in a 13 year old female by the same laboratory.

- The Institute of Medical and Veterinary Science, Adelaide, reported the first notification of echovirus type 12 since 1978. The virus was isolated from faecal specimens from a two month old male.

- Echovirus type 9 was isolated by Fairfield Hospital, Melbourne, from nasal aspirate specimens from a one year old girl and a four month old boy. The two patients were from approximately 30 children from a residential home who presented with recurrent fever, lethargy and a generalised rash. Meningitis was noted in some cases.

- A disseminated gonococcal infection was diagnosed at Fairfield Hospital, Melbourne, in a 50 year old man presenting with fever and polyarthritis. He had recently returned from Thailand. N. gonorrhoeae (penicillin sensitive) was isolated from purulent material aspirated from his left knee, and a pharyngeal swab. Urethral and rectal cultures were negative.

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.

ENDEMIC NEONATAL ROTAVIRUS INFECTION IN HOSPITAL NURSERIES

(Contributed by G.L. Gilbert, Department of Pathology, Royal Women's Hospital, Carlton, Victoria.)

Investigations by R. Bishop and colleagues in the Department of Gastroenterology, Royal Children's Hospital, Melbourne, and by B. McLean in the Department of Pathology, Royal Women's Hospital, indicate that rotavirus infections have been endemic in the Royal Women's Hospital for at least five years. Electropherotyping performed by S. Rodger at the Department of Microbiology, University of Melbourne, has shown that the same rotavirus variant has been responsible for most infections up to late 1979 when it was replaced by a second variant. Both of these "hospital" variants are different from the epidemic rotavirus strains that have been prevalent in the community since 1976. Because of virus attenuation, most of the hospital infections are now asymptomatic. Infection is particularly common in the premature nursery, where small babies are observed for several weeks before discharge.

Since March 1980, all specimens of faeces received in the laboratory have been tested routinely for the presence of rotavirus antigen by enzyme linked immunosorbent assay (ELISA). Most specimens were from infants with diarrhoea from either the premature nursery, or less commonly, from the intensive care nursery. A few were from full-term infants in the postnatal wards. During the nine month period (March-December 1980, excluding April) a total of 217 faecal specimens from 170 patients with diarrhoea were examined. Rotavirus antigen was detected in 57 specimens (25%) from 50 patients (29%). These figures almost certainly underestimate the true proportion of rotavirus diarrhoea infections, since in many instances, faeces were not sent until diarrhoea had been present for several days, or the patient was recovering.

A more accurate figure for the incidence of endemic rotavirus infection may be derived from the results of two smaller surveys. One survey was conducted in the premature nursery over a two week period in April. Faeces were collected from 19 babies present in the ward at the beginning of the survey, and from 13 babies admitted to the ward on days 2 and 5 after admission. In addition, faeces were collected from any baby in whom diarrhoea developed during the survey period. Rotavirus antigen was detected in six of the 19 babies in the ward at the beginning of the survey. Ten of the 13 new admissions acquired rotavirus by day 5, and four of these developed diarrhoea. Another eight babies already present in the ward developed diarrhoea during the survey period, and rotavirus antigen was detected in seven cases (i.e. there were 12 babies with diarrhoea, of whom 11 had rotavirus antigen in their faeces).

A second survey was conducted in the postnatal ward in October and November. Thirty-eight specimens were collected from 21 babies over a two week period, starting on day 2 or 3 of life. Rotavirus was detected in 16 specimens from 12 babies, none of whom had significant diarrhoea. Therefore, nearly 60% of babies acquired asymptomatic rotavirus infection in the first week of life. This figure could have been higher if more faecal specimens from each baby had been examined.

The method of rotavirus transmission between the infants is unclear. In the premature nursery the majority of babies were in enclosed isolettes to limit aerosol transmission. In the postnatal wards the babies were generally nursed in open cots besides the mother's bed and cared for by her, so that inadvertent transmission through handling by nursing and medical attendants was minimal. Aerosol transmission would be the more plausible route of infection in this environment.

In summary, it appears that the majority of babies born in this hospital become infected with rotavirus in the first five to seven days of life. Most of them, particularly full-term babies, do not have diarrhoea, although rotavirus is apparently responsible for the vast majority of episodes of diarrhoea which do occur, both in premature and full-term infants. Although every effort is made to prevent cross-infection, particularly in the premature nursery, there is little point in strict isolation of babies with diarrhoea in view of the high proportion of asymptomatic infections. An increased number of diarrhoea episodes in June compared with other months suggests a seasonal variation for the rotavirus infections.

PENICILLIN RESISTANCE IN N. GONORRHOEAE:- DETERMINATION OF MINIMUM INHIBITORY CONCENTRATIONS (MIC) BY AGAR PLATE DILUTION TECHNIQUES

(Contributed by J.W. Tapsall, Department of Microbiology, Prince of Wales Hospital, Sydney.)

A knowledge of the penicillin sensitivity of N. gonorrhoeae isolates is of considerable epidemiological value. Increasing gonococcal penicillin resistance has been shown repeatedly to correlate with inadequate control of gonorrhoea in a community. In addition, more rational treatment regimens can be adopted if the antibiotic sensitivities of the isolates are known. However, the detection of β-lactamase producing strains is still of primary importance, since most non-producing strains are still susceptible to the penicillins if the recommended treatment schedules are followed.

An Australian study group comprising of representatives from a number of diagnostic laboratories interested in venereal disease bacteriology have recommended the following standardised technique for gonococcal penicillin sensitivity testing. Acceptance of this technique would facilitate the collection of comparative data on a national basis.

The minimum inhibitory concentration (MIC) is the lowest concentration of antibiotic which completely inhibits bacterial growth under standardised conditions. This figure is most accurately determined by agar-plate dilution since disc diffusion methods are unreliable and are not recommended. The method is detailed below, and particular emphasis is placed upon preparation of antibiotic solution, choice of medium, preparation of inoculum, use of control strains and determination of endpoints.

Media

1. Basal medium:- Isosensitest agar (Oxoid) is prepared according to the manufacturer's directions and dispersed into volumes appropriate to the amount of media required, e.g. 45ml, 90ml,

etc. The Isosensitest agar is allowed to cool to 52°C prior to the addition of the other constituents.

2. Saponin lysed defibrinated horse blood:- Defibrinated horse blood is lysed by the addition of 0.5ml of filter-sterilized 10% saponin solution (British Drug Houses) per 100ml of horse blood. Sufficient lysed blood is added to the cooled, molten agar so as to give a final concentration of 8%.

3. Penicillin solution:- A stock solution of 1000 µg/ml is dissolved in 0.2M phosphate buffer pH 6.0. This solution may be stored frozen for a maximum of four weeks. Serial dilutions are made in phosphate buffer, and aliquots added to the solutions of agar and lysed horse blood to give final penicillin concentrations (in µg/ml) of 2.0, 1.0, 0.5, 0.25, 0.12, 0.06, 0.03, 0.015, 0.008, 0.004 and zero. The medium is then dispersed into Petri dishes, and the plates are either used the same day, or stored at 4°C for use the following day. The plates are dried for one hour at 37°C prior to use.

Inoculum

The inoculum is prepared from an overnight culture grown on lysed blood agar incubated at 37°C in 5% CO_2. The culture is suspended in normal saline with 20% peptone water to approximately 10^9 organisms/ml, and the turbidity checked either by a nephelometer or by standard opacity tubes (Brown, McFarland). The suspension is then serially diluted using standard 40 dropper Pasteur pipettes (one drop in 2.5ml diluent) to give 10^{-2}, 10^{-4} and 10^{-6} dilutions. The original neat suspension and the 10^{-2} and 10^{-4} dilutions are placed in the wells of Steer's-type multiple inoculator which delivers 0.004ml to the surface of the plate.

The plates are allowed to dry for 15 minutes, and then incubated in 5% CO_2 at 37°C for 24 hours. The final 10^{-6} dilution is used to check the inoculum; surface viable counts are assayed by delivering 0.025ml to the surface of a lysed-blood agar plate.

Controls

The Oxford Staphylococcus, WHO strains III, V and VII, and a freshly isolated strain (from the previous MIC run) are included in each assay as controls.

Reading the MIC

The MIC is determined using the 10^{-4} dilution inoculum which contains approximately 10^4 organisms per replicator drop. The end-point is taken as that penicillin concentration where there is complete inhibition of growth. Any haze is ignored. The control plate containing no antibiotic is used for comparative purposes, as growth diminishes as the endpoint is reached.

The Australian study group gratefully acknowledges the interest and cooperation of the National Health Institute of New Zealand, Wellington. The participating laboratories in the study group are listed below, and are available for consultation:

- Laboratory of Microbiology and Pathology (State Health Laboratory), Brisbane, Queensland.
- Institute of Clinical Pathology and Medical Research, Westmead, N.S.W.
- The Prince of Wales Hospital Microbiology Department, Randwick, N.S.W. (J.W. Tapsall, Study group co-ordinator).
- Woden Valley Hospital Microbiology Department, Woden, A.C.T.
- Microbiological Diagnostic Unit, University of Melbourne, Parkville, Victoria.
- Venereal Diseases Unit, Royal Adelaide Hospital, Adelaide, S.A.
- State Health Laboratory Services, Shenton Park, W.A.

SCARLET FEVER - VICTORIA

(Contributed by M. Peel, Microbiological Diagnostic Unit, Melbourne, and M. Anderson and H. Cailes.)

The occurrence of five cases of clinically diagnosed scarlet fever among school children in a Victorian country town led to a survey to determine the incidence of Group A β-haemolytic streptococci in the school children, their teachers and the parents of the cases. Swabs were taken from the nose and throats of a total of 107 persons, none of whom had symptoms of pharyngitis at the time of collection of the specimens. Group A streptococci were isolated from 12 of the nasal swabs, two of the throat swabs and from both sites in six instances.

The isolates were referred to D. Martin of the Streptococcus Reference Laboratory, National Institute of Health, Wellington, New Zealand, for typing by the precipitin method for M protein. This protein is the major virulence antigen of group A streptococci, and over 60 serotypes are recognized on the basis of antigenic differences. All isolates, except two from nasal swabs, were of M type 4, a recognized epidemic strain which has been recently implicated in outbreaks of scarlet fever in New Zealand.

Penicillin 1g orally daily for 10 days was prescribed for all known positive carriers. Household contacts of the scarlet fever cases were prescribed 1g of penicillin daily for 5 days. About two months after the end of treatment, follow-up cultures for Group A streptococci were undertaken. Group A streptococci were re-isolated from the throats of four of the treated children. Although re-infection may have occurred and non-compliance with treatment cannot be excluded, it is also possible that the Group A streptococci may have persisted despite penicillin therapy as such treatment failures have been documented[1]. The four children were given a repeat course of penicillin treatment. Three of the four were available for a swabbing a further two months later, and none grew β-haemolytic streptococci on culture.

Editorial Comment

Scarlet fever results from infection with a streptococcal strain that elaborates an erythrogenic toxin which is dependent on the lysogeny of the infecting streptococcus with a temperate bacteriophage. Chemotherapy is directed toward prevention of acute rheumatic fever and suppurative complications such as otitis media and acute sinusitis. Data on the preventability of post-streptococcal glomerulonephritis is less clear-cut.

Two aspects of therapy still remain subject to debate - the necessity for retreatment of treatment failures, and the management of family contacts of patients with streptococcal sore throat. Approximately 10-15% of streptococcal-infected patients are found to harbour β-haemolytic streptococci in the pharynx following completion of a ten day course of oral penicillin therapy[2]. The causes are multiple and include failure of compliance with oral medication schedules, reinfection with the same or different streptococcal types, or true treatment failure. There appears to be no correlation between the presence of penicillinase production by co-infecting staphylococcus strains[3,4]. Most treatment failures appear to be streptococcal carriers, and not acutely infected individuals, the general impression being it is more difficult to eradicate the organism since they are multiplying less actively, and hence are not as readily killed by penicillin. Streptococcal carriers are not at inordinate risk of developing rheumatic fever nor of spreading their infection to others.

The second unresolved issue relates to the management of family contacts of patients with streptococcal sore throats since streptococcal acquisition rates of 25% or higher have been recorded. In the absence of special epidemiological circumstances such as a family with a member who has documented rheumatic fever, or a community epidemic of rheumatic fever, routine culture of asymptomatic family or close contacts is unnecessary.

References

1. Amer. J. Dis. Child. (1972) 123:457
2. Bisno, A.L., 'Streptococcus pyogenes' In: Mandell, G.L., Gordon-Douglas,Jr.,R. and Bennett, J.E., eds. Principles and Practice of Infectious Diseases. Vol 2, pp 1562-1573 (1979) John Wiley Medical Publications.
3. Paediatrics (1966) 37:467
4. J. Paed. (1967) 71:132

POLIOVIRUS CONTAMINATION - A CAUTIONARY TALE

(Contributed by I. Cook, State Health Laboratory, Brisbane.)

Two highly unusual isolations of poliovirus were made from a 17 year old patient with simultaneous conjunctivitis and vulval lesions. Poliovirus type 1 was isolated from the eye swab and poliovirus type 3 from the vulval swab. Herpes simplex virus was also isolated from the vulval swab. These viruses were again isolated on retesting the original specimens.

In view of the extremely unlikely involvement of these poliovirus isolates in the diseases presented, it was assumed that they were Sabin vaccine contaminants. The specimens were referred to the State Health Laboratory by a private doctor via a pathology laboratory. Enquiries failed to reveal the possible source of the contamination. Repeat specimens from the patient proved negative for virus.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 1

REPORTING PERIOD - 5-2-81 - 18-2-81 BULLETIN NUMBER . 81/4

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	8		1		1	3	1	1	15
0101 ADENOVIRUS TYPE 1						3		1	4
0102 ADENOVIRUS TYPE 2	1	1				2		4	8
0103 ADENOVIRUS TYPE 3				2					2
0104 ADENOVIRUS TYPE 4			2			1			3
0105 ADENOVIRUS TYPE 5	1				1				2
0107 ADENOVIRUS TYPE 7	2	1				1			4
0111 ADENOVIRUS TYPE 11						1			1
0115 ADENOVIRUS TYPE 15					1				1
0119 ADENOVIRUS TYPE 19								7	7
0199 ADENOVIRUS TYPING PENDING			2		4	2			8
0201 INFLUENZA A VIRUS			1	1		1	1	5	9
0203 INFLUENZA B VIRUS			1	2					3
0204 INFLUENZA C VIRUS							1		1
0301 PARAINFLUENZA VIRUS TYPE 1				1					1
0302 PARAINFLUENZA VIRUS TYPE 2								1	1
0303 PARAINFLUENZA VIRUS TYPE 3	2			1	1	1	3	2	10
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)	1	1					2		4
0500 RHINOVIRUS (ALL TYPES)				2	3		1		6
0600 MYCOPLASMA PNEUMONIAE	3	1	5					2	11
0700 ORNITHOSIS-PSITTACOSIS	1			1		3			5
0809 COXSACKIEVIRUS A9					1				1
0902 COXSACKIEVIRUS B2						2			2
1006 ECHOVIRUS TYPE 6								1	1
1009 ECHOVIRUS TYPE 9	1			2					3
1011 ECHOVIRUS TYPE 11			1						1
1012 ECHOVIRUS TYPE 12						1			1
1014 ECHOVIRUS TYPE 14								1	1
1022 ECHOVIRUS TYPE 22	2		3		3				8
1023 ECHOVIRUS TYPE 23			1						1
1030 ECHOVIRUS TYPE 30				2			1		3
1102 POLIOVIRUS TYPE 2	3					2			5

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 2.

REPORTING PERIOD - 5 - 2 - 81 - 18 - 12 - 81 BULLETIN NUMBER . 81/4

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES-CONTINUED

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
1104 POLIOVIRUS-VACCINAL STRAIN.....			4		1				5
1200 MUMPS VIRUS.....				2		3	1	2	8
1300 HERPES VIRUS GROUP-NOT TYPED.....	11		6	2	2	5			26
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....	4	5		9				35	53
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	4					2		3	9
1303 VARICELLA-ZOSTER VIRUS.....	2		2	2					6
1306 HERPES SIMPLEX TYPE 1.....	3			16		23	15		57
1307 HERPES SIMPLEX TYPE 2.....	37			27		25	23		112
1399 HERPES VIRUS TYPING PENDING.....			6			2			8
1401 COXIELLA BURNETI.....	21		1	4		6	8		40
1502 PICORNA VIRUS-NOT TYPED.....								1	1
1521 MEASLES VIRUS.....	1		5						6
1522 RUBELLA VIRUS.....	1			3			2		6
1532 HEPATITIS B ANTIGEN.....	12		9	25		23	5	6	80
1535 HEPATITIS A ANTIBODY.....	3	2	2	13		9	1	6	36
1541 CHLAMYDIA A - TRIC TYPE.....	19		5					71	95
1543 CHLAMYDIA A - LGV TYPE.....	1								1
1556 CMV - CYTOMEGALOVIRUS.....	8		7	16	4	1	1	10	47
1562 REOVIRUS (ALL TYPES).....					1			1	2
1564 ROTAVIRUS.....		2	3		19	7		5	36
1599 ENTEROVIRUS TYPING PENDING.....		1	4		8		1		14
POXVIRUS GROUP NOT TYPED				1					1
ROSS RIVER VIRUS							2	1	3
SMALL VIRUS (LIKE) PARTICLE	6								6
ARBO. GROUP B.						2			2
Total.....	158	14	71	134	50	131	69	166	793

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 3

PERIOD : 5/2/81 - 18/2/81 .... 81/4

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		4					5				1
0101 ADENOVIRUS TYPE 1		2					2				
0102 ADENOVIRUS TYPE 2		3		1		1					
0103 ADENOVIRUS TYPE 3		1									
0104 ADENOVIRUS TYPE 4		2					1				
0105 ADENOVIRUS TYPE 5							2				
0107 ADENOVIRUS TYPE 7		2									
0115 ADENOVIRUS TYPE 15		1									
0201 INFLUENZA A VIRUS		4				1			1	1	
0203 INFLUENZA B VIRUS		1									
0301 PARAINFLUENZA VIRUS TYPE 1		1									
0302 PARAINFLUENZA VIRUS TYPE 2		1									
0303 PARAINFLUENZA VIRUS TYPE 3	2	7									1
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)		3									
0500 RHINOVIRUS (ALL TYPES)		6									1
0600 MYCOPLASMA PNEUMONIAE	2	6			1				1		
0700 ORNITHOSIS-PSITTACOSIS		2									
0809 COXSACKIEVIRUS A9							1				
0902 COXSACKIEVIRUS B2							2				
1009 ECHOVIRUS TYPE 9	1										1
1011 ECHOVIRUS TYPE 11				1							
1012 ECHOVIRUS TYPE 12							1				
1022 ECHOVIRUS TYPE 22	5						3				
1023 ECHOVIRUS TYPE 23							1				
1030 ECHOVIRUS TYPE 30		1		2							
1102 POLIOVIRUS TYPE 2			1	1			3				
1104 POLIOVIRUS-VACCINAL STRAIN							4				

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 4

PERIOD : 5/2/81 - 18/2/81 .... 81/4

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.-CONTINUED

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
1200 MUMPS VIRUS.....		1	2	1							
1300 HERPES VIRUS GROUP-NOT TYPED..	3		1	1	1	2					9
1301 HERPES SIMPLEX VIRUS NOT-TYPED	3	7									33
1302 EPSTEIN-BARR VIRUS (EB VIRUS).	1		2								
1303 VARICELLA-ZOSTER VIRUS.....	1					1					4
1306 HERPES SIMPLEX TYPE 1.....		1				1					31
1307 HERPES SIMPLEX TYPE 2.....											11
1401 COXIELLA BURNETI.....	10	5		1							
1521 MEASLES VIRUS.....	1				1	1					2
1522 RUBELLA VIRUS.....	1										5
1532 HEPATITIS B ANTIGEN.....	41							36			
1535 HEPATITIS A ANTIBODY.....	2							34			
1541 CHLAMYDIA A - TRIC TYPE.....		1									
1556 CMV - CYTOMEGALOVIRUS.....	8	2				1		1	1	6	
1562 REOVIRUS (ALL TYPES).....							1				1
1564 ROTAVIRUS.....	4						31				
POXVIRUS GROUP NOT TYPED											1
SMALL VIRUS (LIKE) PARTICLE		1					5				
TOTAL.....	85	65	6	8	3	8	62	71	3	7	101

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

5

PERIOD : 5/2/81 - 18/2/81 ...

81/4

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
0100 ADENOVIRUS NOT TYPED.....			1				2	1		
0102 ADENOVIRUS TYPE 2.....										3
0103 ADENOVIRUS TYPE 3.....								1		
0107 ADENOVIRUS TYPE 7.....	1						1			
0111 ADENOVIRUS TYPE 11.....	1									
0119 ADENOVIRUS TYPE 19.....	1	6								
0201 INFLUENZA A VIRUS.....								4		
0203 INFLUENZA B VIRUS.....				1				1		
0204 INFLUENZA C VIRUS.....								1		
0303 PARAINFLUENZA VIRUS TYPE 3....								1		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....								1		
0600 MYCOPLASMA PNEUMONIAE.....					1		1		1	
0700 ORNITHOSIS-PSITTACOSIS.....			1				2			
1006 ECHOVIRUS TYPE 6.....							1			
1009 ECHOVIRUS TYPE 9.....								2		
1014 ECHOVIRUS TYPE 14.....								1		
1104 POLIOVIRUS-VACCINAL STRAIN....	1									
1200 MUMPS VIRUS.....			4							
1300 HERPES VIRUS GROUP-NOT TYPED..		2						1	2	
1301 HERPES SIMPLEX VIRUS NOT-TYPED		19						1		
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			2	4				1		
1303 VARICELLA-ZOSTER VIRUS.....								1		
1306 HERPES SIMPLEX TYPE 1.....	6	16		1				1	1	
1307 HERPES SIMPLEX TYPE 2.....		99								
1401 COXIELLA BURNETI.....					1		15	11	1	
1521 MEASLES VIRUS.....									1	
1532 HEPATITIS B ANTIGEN.....									2	

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE 6

PERIOD : 5/2/81 - 18/2/81 ... 81/4

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 ... **-CONTINUED**

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
1541 CHLAMYDIA A - TRIC TYPE.....	1	93								
1543 CHLAMYDIA A - LGV TYPE.....							1			
1556 CMV - CYTOMEGALOVIRUS.....		8	1	2		1	7	-	5	
1562 REOVIRUS (ALL TYPES).....								1		
1564 ROTAVIRUS.....	1									
ROSS RIVER VIRUS					3			1		
SMALL VIRUS (LIKE) PARTICLE									1	
ARBO. GROUP B.								2		
TOTAL.....	12	243	9	8	5	1	30	37	15	3

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

12th4 Weekly Period for 1980...

1.11.80 to 28.11.80 inclusive

Bulletin ..81/4..

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Amoebiasis	N.N.		1		1			2	4	49
Ankylostomiasis	N.N.			N.N.					—	177
Anthrax									—	2
Arbovirus infection		1		N.N.					1	25
Brucellosis	1	1							2	47
Campylobacter infections	N.N.	N.N.	N.N.	75	N.N.	N.N.	N.N.	N.N.	75	425
Chancroid	N.N.		2	N.N.	N.N.	N.N.	N.N.		2	30
Cholera									—	3
Congenital rubella syndrome	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	2	2	3
Diphtheria									—	1+2 CARRIERS
Donovanosis	N.N.	N.N.	21	N.N.	N.N.	N.N.	4		25	82
Giardiasis	N.N.	N.N.	N.N.	82	N.N.	N.N.	N.N.	N.N.	82	509
Genital herpes	N.N.	N.N.	N.N.	40	N.N.	N.N.	N.N.	N.N.	40	364
Gonococcal ophthalmia neonatorum	N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	N.N.	—	—
Gonorrhoea	209	209	197	78	125	12	25	14	869	*10,644
Hepatitis A (infectious)	64	30	8	7	1	2	18	4	134	1,283
Hepatitis B (serum)	10	16	2	5			1		34	618
Hepatitis - unspecified	N.N.	N.N.		N.N.	12	N.N.	N.N.		12	170 +1 CARRIER
Hydatid disease	3			1					4	38
Lassa Fever	N.N.		N.N.	N.N.		N.N.	N.N.	N.N.	—	—
Legionnaires disease	N.N.		N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	—	—
Leprosy			1				1		2	33
Leptospirosis		13	2						15	51
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.			—	1
Malaria	8	5	8	3	6		1	2	33	526
Marburg Disease	N.N.		N.N.	N.N.		N.N.	N.N.	N.N.	—	—
Meningococcal infections	N.N.		1	1		N.N.			2	67
Non-specific urethritis	N.N.	N.N.	N.N.	106	N.N.	N.N.	N.N.	N.N.	106	1205
Ornithosis									—	17
Pertussis (whooping cough)	N.N.	14	N.N.	20	N.N.	N.N.	N.N.	N.N.	34	120
Plague									—	—
Poliomyelitis									—	—
Q. fever	1	7	23	5	N.N.		N.N.		36	561
Rabies	N.N.	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	CUMULATIVE TOTAL TO DATE FOR YEAR
Salmonella infections	24	10	9	59	18	1	18	3	142	2,084
Shigella infections	N.N.		6	1	5		37		49	504
Smallpox									–	–
Syphilis	54	22	60	4	8		42		190	* 2,572
Tetanus			1						1	9
Trachoma	N.N.	N.N.		N.N.	N.N.	N.N.			–	1
Tuberculosis (all forms)	36	33	22	10	11			2	114	1,440
Typhoid fever	1	1							2	19 + 4 CARRIERS
Typhus (all forms)									–	–
Vibrio parahaemolyticus infections	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	N.N.	–	–
Yellow Fever									–	–
Yersinia enterocolitica infections	N.N.	N.N.	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

* Corrections to the Cumulative Total since last report

Gonorrhoea - 6 cases for W.A.

Syphilis - 6 cases for W.A.