



Communicable Diseases Intelligence

Bulletin number

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VIRUS REPORTING SCHEME - A total of 1398 reports were processed this period. The dengue infection reported by the Institute of Medical and Veterinary Science, Adelaide, was in a female who had recently returned from the Philippines. The distribution of the epidemic polyarthrititis reports was Queensland - 26 (20 from the far north of the State); Western Australia - 6; and one each from the Northern Territory, South Australia and Victoria. The ten echovirus type 7 reports from the State Health Laboratory, Brisbane, included the isolation from a 32 year old male with a "red eye" without discharge.

- . On 25 January 1985, a 32 year old Australian woman was admitted for high security isolation at Fairfield Hospital, Melbourne. She had worked in Nigeria as a missionary teacher and had returned recently to Australia. Although perfectly well, she had had a moderately severe attack of Lassa fever three months before in Nigeria. She was admitted for quarantine because of the possibility that she may still be a Lassa fever virus carrier. Her husband and two young children were also quarantined because of the close contact. Specimens from the patient forwarded to Porton Down, Salisbury, UK, and the Centres for Disease Control, Atlanta, USA, were negative for culture. A serum antibody titre of $> 1/320$ was found in the patient, but the 12 sera taken from close contacts were negative. The woman has since been discharged. Lassa fever is endemic in Jos, Nigeria, and antibody prevalence rates reach 20%, with cases occurring all the year round. No secondary cases have occurred with the nine confirmed cases imported in the UK, and more attention is now given to the identification of high-risk hospital contacts and less emphasis given to low-risk community contacts of cases of Lassa fever.
- . To 13 February 1985, 53 cases of AIDS, fulfilling the criteria of case definition, have been reported to the AIDS Task Force.

<u>State</u>	<u>Cases</u>	<u>Deaths</u>
New South Wales	37	7
Victoria	10	7
Western Australia	2	-
Queensland	4	4
Total	53	18

GONOCOCCAL SURVEILLANCE - AUSTRALIA (JULY - SEPTEMBER, 1984)
(Contributed by the Australian Gonococcal Surveillance Program (AGSP). Co-ordinator - J.W. Tapsall, Department of Microbiology, Prince of Wales Hospital, Sydney).

The AGSP reports the sensitivity of gonococci isolated throughout Australia as determined by agreed and standardised methods⁽¹⁾. This report provides details of penicillin sensitivities of 1183 isolates of Neisseria gonorrhoeae for the period July-September 1984. Table 1 gives the percentages of gonococcal isolates classified as either (A) sensitive (minimal inhibitory concentration (MIC) value = 0.008µg/ml) or (B) less sensitive (MIC = 0.12µg/ml) to penicillin by region. Comparison is made with the data obtained over the same period in 1983.

TABLE 1 Penicillin sensitivity of N. gonorrhoeae isolates (July-September 1984)

<u>Centre</u>	<u>Percentage of isolates</u>		
	<u>Sensitive (A)</u>	<u>Less sensitive (B)</u>	<u>PPNG</u>
Brisbane	29.5 (34.7)	61.8 (50.8)	2.0 (11.4)
Sydney	15.0 (16.6)	56.9 (72.8)	13.0 (3.0)
Melbourne	26.0 (32.2)	44.8 (44.4)	6.4 (0.6)
Adelaide	47.9 (32.2)	44.7 (53.9)	1.1 (3.5)
Perth	32.5 (44.0)	42.7 (13.4)	5.7 (8.2)

No. strains examined = 1183 (1053)

In addition to the data contained in Table 1, information was also available from laboratories in Hobart, Canberra and Darwin where smaller numbers of gonococci were isolated. There was little change in the distribution of gonococcal penicillin sensitivity patterns since the previous report (CDI 84/22:2), with the exception of Adelaide where the proportion of fully sensitive strains has increased.

In the previous report reference was made to the significant increasing incidence of penicillinase-producing N. gonorrhoeae (PPNG), particularly in Sydney. In the present period 13% of all gonococci isolated in Sydney were PPNG, compared with 14.4% and 18.1% in the two preceding quarters. Preliminary data for the October-December quarter indicates a further and substantial fall (to about 6%) in the PPNG isolation rate. Similarly, all the other centres, except Melbourne, exhibited a decrease in the PPNG isolation rate. PPNG strains were also isolated in all other centres including Hobart, Canberra and Darwin. However, as before, approximately two-thirds of the Sydney PPNG infections were the result of sustained domestic transmission. For the centres, excluding Sydney, in which the sources of infection were known, 55 (71.4%) of 77 PPNG isolates were acquired in South-East Asia, or were contacts or presumed contacts of patients who acquired their disease in that region.

Reference

1. Br. J. Vener. Dis. (1984) 60 : 226

IMPORTED SHIGELLOSIS WITH BACTERAEMIA - VICTORIA

(Contributed by G.L. Gilbert, Department of Microbiology, Royal Children's Hospital, Melbourne).

An eight month old baby girl developed diarrhoea two weeks

after arriving with her mother on a visit to Lebanon. She was admitted to hospital in Lebanon where she required intravenous therapy and was treated with amoxycillin. She was discharged after initial improvement, but high fever and diarrhoea recurred. She was readmitted to hospital and again given intravenous therapy for approximately 24 hours before leaving, by air, to return to Australia. Her mother was advised to give her plenty of carrots to eat during the flight. However, severe diarrhoea continued at 15-20 minute intervals and she was admitted to the Royal Children's Hospital immediately after arrival at Tullamarine airport.

On examination she was irritable and lethargic. The degree of dehydration was estimated to be approximately 15%. Her temperature was 38.3°C, pulse 140/min and blood pressure 70/30 mm Hg. She was severely hyponatremic (serum sodium 113 mmol/l). Intravenous therapy was commenced immediately. Blood cultures and faeces were sent for culture. The first specimen of faeces consisted mainly of undigested carrot with a fairly small amount of faecal material, and no pathogens were isolated from it.

A Gram-negative bacillus was isolated from two sets of blood cultures taken on the day of admission and the next day. The organism was non-motile, indole and citrate negative; it fermented glucose (with production of gas) and arabinose, but neither lactose nor mannitol (or any other sugar tested). It was not identifiable with the API 20E system but was agglutinated by polyvalent shigella and specific Shigella boydii antiserum. Further investigation at the Microbiological Diagnostic Unit (MDU), University of Melbourne, identified it as S. boydii serotype 14. The same organism was subsequently isolated from specimens of faeces taken on the second and sixth days after admission.

Intravenous fluids were initially given for two days. She improved and was able to tolerate oral fluids. However, frequent diarrhoea persisted. Hyponatremia developed again and intravenous therapy was reinstated.

The S. boydii strain was resistant to sulphonamide and trimethoprim but sensitive to amoxycillin, which was started on the seventh day after admission. S. boydii was not isolated from faeces cultured on the eighth and eleventh days after admission, and the diarrhoea slowly resolved over a period of 5-7 days. She was discharged 17 days after admission and remained well.

S. boydii type 14 is uncommonly encountered in this country. Only three isolates have been identified by the MDU since 1980. It differs from most shigella in that it ferments mannitol and may produce gas from glucose. The fact that it was isolated initially from blood culture but not from faeces was potentially confusing. Bacteraemia associated with shigella infection is unusual. A recent review cited 68 cases reported in the literature during the last 20 years⁽¹⁾. The majority of patients were children under five years of age and the case fatality rate was 46%. The present patient was severely ill on admission to hospital because of dehydration and hyponatremia, but she did not appear, clinically, septicaemic.

Editorial Comment

Most of the isolations of S. boydii type 14 are associated with the Indian sub-continent. During the period 1975-82, only 20

isolates were confirmed in the UK⁽²⁾. As in the above case, it appears that the serotype can, and on occasion does, cause severe symptoms⁽³⁾.

1. Ped. Inf. Dis. (1983) 2 : 21
2. CDR (1983) 83/13 : 3
3. Med. Lab. Sci. (1976) 33 : 309

SALMONELLA AGONA SURVEILLANCE - WESTERN AUSTRALIA

(Based on Enteric Pathogens Report, July-September 1984, State Health Laboratory Services).

Salmonella agona is recognised as a major international epidemic pathogen. However, its epidemiological profile in Australia, although widespread in livestock and humans, has not emerged to date either as a frequent cause of sporadic cases, or a source of local or interstate epidemics.

S. agona was first isolated in Western Australia in 1977 from immigrants arriving from South East Asia, and since that time up to September 1984 a total of 58 cases have been recorded. Two small but significant outbreaks have occurred. In 1979, a nursing sister infected with S. agona was implicated on return from an overseas vacation as the source of a maternity hospital outbreak involving a total of ten contact cases. In October-December 1983, a total of 15 cases occurred in Western Australian residents in which the source of infection was not found. In 1983, the outbreak involved eight different families and was accompanied by a massive statewide increase in S. agona isolations detected by the sentinel sewerage monitoring service.

In the July-September 1984 quarter, the abattoir effluent sentinel monitoring service detected S. agona in abattoir effluents for the first time in Western Australia at a small abattoir and smallgoods producing operation in the South West region of the State. Follow-up investigations revealed widespread S. agona contamination at the abattoir and in offal and sausage casings supplied to the smallgoods trade and animal feed industry.

Three abattoir workers were excreting S. agona and isolations were also recorded from abattoir equipment, pig lairage yards and pig faeces. Cattle and sheep were not implicated as a possible source of contamination at the abattoir, and trace backs of pigs proved negative. Monitoring of other abattoir effluents produced no S. agona isolations and positive findings were confined to one offal rendering plant and a smallgoods manufacturer supplied with sausage casings from the S. agona contaminated abattoir. No human S. agona isolations have been recorded in the wake of remedial measurements at the abattoir or in the animal feed industry including meat meals, raw meats or locally manufactured smallgoods. Representative cultures have been forwarded to the Public Health Enteric Pathogens Reference Laboratory, Colindale, England, for epidemiological study.

BOTULISM DUE TO NON-CANNED, FRESH FOOD - CALIFORNIA

(Based on California Morbidity (1985) No. 4)

Two incidents of botulism in which there were no exposure to canned food were reported recently in California.

OUTBREAK 1 - In August 1984, botulism was reported from Santa Cruz County in two individuals; a 61 year old woman and her 13 year old daughter. The older woman had classic symptoms; bilateral ptosis, diplopia, and facial weakness; the granddaughter was less ill. Trivalent botulinum antitoxin was administered and type A botulinal toxin was confirmed in sera of both patients. Although food histories revealed no recent exposures to home-canned food, improper food handling was identified as a likely cause for this outbreak. Three days before onset, two turkey loaves which included ingredients of cereal, onion and green pepper, were prepared by the grandmother. One loaf was promptly consumed immediately after cooking without incident. The other turkey loaf however, was inadvertently stored in the gas oven with the pilot light on (later measured at 98°F), until the grandmother discovered it the next afternoon. She tasted a small portion, decided it was still alright, reheated it at 300°F for about 20 minutes, and served the turkey loaf to the three other members of her household. Thirty-six hours later she awoke with ptosis, diplopia and facial weakness. Of the others who ate the rewarmed meat loaf, only the granddaughter developed symptoms. She could not recall tasting the turkey loaf with her grandmother prior to reheating, but she did recall eating a portion from the centre of the loaf (which may not have been as hot as other portions). Since both turkey loaves were completely consumed, confirmatory tests were not possible. Both patients recovered completely.

OUTBREAK 2 - In August 1984 in Orange County, a 22 year old man awoke one morning at 2 a.m. with vomiting, difficulty focusing and "thick tongue"; symptoms progressed to total quadriplegia and then respiratory failure requiring mechanical respiratory assistance. Forty hours before onset, he had consumed stew left at room temperature overnight after preparation by his roommate from fresh ingredients that included meat and unpeeled potatoes and carrots. These were cooked in a 7" deep pot that was filled to the top, and simmered for 45 minutes, after which the gas was turned off and the pot left on the range. The cook ate it hot after the initial cooking without incident; but when the patient tasted it without reheating 16 hours later, he complained of a bad taste. The cook confirmed a "sour" taste, immediately spat it out and rinsed his mouth; he remained well. The stew was then discarded and so would not be tested. Type A botulinal toxin was confirmed in the patient's serum; he was treated with botulinal antitoxin and required an extended hospital stay.

Spores of Clostridium botulinum are ubiquitous in soil and so can contaminate fresh foods, particularly those harvested from the ground. Except in rare infants who develop infant botulism, the spores are ingested with impunity. Toxin can be elaborated however, when spores - which can resist hours of boiling-persist and germinate in cooked foods left at ambient temperatures for many hours (particularly deep portions that remain anaerobic). It is suspected that the same mechanism of toxin production accounted for previous episodes of botulism from commercial pot pies that were cooked, allowed to stand at ambient temperatures and consumed later without reheating (CDI (1983) 83/3 : 5). Foods heated for serving should either be eaten hot, or refrigerated and later reheated thoroughly (since the toxin is heat labile) when served again.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE
 REPORTING PERIOD - 31/1/85 - 13/2/85 BULLETIN NUMBER 85/4
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR	RAHC (NSW)	PHH/	FAIR-	RCH	IMVS	STATE	STATE	Total
	(NSW)/ MVH (ACT)		POW (NSW)	FIELD (VIC)			LAB (QLD)	LAB (WA)	
0100 ADENOVIRUS NOT TYPED.....	3	3	8		1	5	20	2	42
0101 ADENOVIRUS TYPE 1.....	1			1		2			4
0102 ADENOVIRUS TYPE 2.....	2			1		1			4
0103 ADENOVIRUS TYPE 3.....	2			3		1			6
0108 ADENOVIRUS TYPE 8.....				2					2
0111 ADENOVIRUS TYPE 11.....				1					1
0119 ADENOVIRUS TYPE 19.....	2								2
0199 ADENOVIRUS TYPING PENDING.....		3	3		1	1			8
0201 INFLUENZA A VIRUS.....	8					1		6	15
0203 INFLUENZA B VIRUS.....			2					2	4
0301 PARAINFLUENZA VIRUS TYPE 1.....						1			1
0302 PARAINFLUENZA VIRUS TYPE 2.....	1						2	1	4
0303 PARAINFLUENZA VIRUS TYPE 3.....	5	2		2	3			2	14
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)...	2		1				1	2	6
0500 RHINOVIRUS (ALL TYPES).....	2				4	2	1		9
0600 MYCOPLASMA PNEUMONIAE.....	6		1				1	3	11
0700 ORNITHOSIS-PSITTACOSIS.....						1			1
0809 COXSACKIEVIRUS A9.....	4		1		1	1	6		13
0904 COXSACKIEVIRUS B4.....				1					1
0905 COXSACKIEVIRUS B5.....		1	1		1	2	2	3	10
1000 ECHOVIRUS NOT TYPED.....							3		3
1007 ECHOVIRUS TYPE 7.....	2						10		12
1009 ECHOVIRUS TYPE 9.....					2			1	3
1011 ECHOVIRUS TYPE 11.....	1	1							2
1014 ECHOVIRUS TYPE 14.....					1				1
1017 ECHOVIRUS TYPE 17.....	1								1
1020 ECHOVIRUS TYPE 20.....				1					1
1030 ECHOVIRUS TYPE 30.....				3	1				4
1100 POLIOVIRUS NOT TYPED.....			1		3		3		7
1101 POLIOVIRUS TYPE 1.....	1			1				1	3
1102 POLIOVIRUS TYPE 2.....						3			3
1200 MUMPS VIRUS.....	6			1	1			1	9
1300 HERPES VIRUS GROUP-NOT TYPED.....	48		1					4	53
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....						1			1
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	5	3			2			12	22
1303 VARICELLA-ZOSTER VIRUS.....	4			2		1			7
1306 HERPES SIMPLEX TYPE 1.....	16		8	39	16	27	40	12	158
1307 HERPES SIMPLEX TYPE 2.....	108		31	63		20	84	68	374
1399 HERPES VIRUS TYPING PENDING.....				3	4				7
1401 COXIELLA BURNETI.....	1					1			2
1502 PICORNA VIRUS-NOT TYPED.....	6		5					1	12
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....								2	2
1521 MEASLES VIRUS.....	12					1		1	14
1522 RUBELLA VIRUS.....	36	1	6	1		7	5	10	66
1532 HEPATITIS B ANTIGEN.....	83		14			19	20	13	149
1535 HEPATITIS A ANTIBODY.....	1		1			1	2	3	8
1541 CHLAMYDIA A - C TRACHOMATIS.....	41		10	34			38	60	183
1556 CMV - CYTOMEGALOVIRUS.....	10	5	2	24	3	3	3	2	52
1564 ROTAVIRUS.....			7	1	2	11		3	24
1599 ENTEROVIRUS TYPING PENDING.....		4	10		5				19
9992 ROSS RIVER VIRUS.....						1	26	7	34
9993 ASTROVIRUS.....				1					1
9994 SMALL VIRUS (LIKE) PARTICLE.....	2								2
9995 DENGUE.....						1			1
Total.....	422	23	113	185	51	115	267	222	1,398

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 31/1/85 to 13/2/85

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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.....		2				1					
0102 ADENOVIRUS TYPE 2.....		3					1				
0103 ADENOVIRUS TYPE 3.....		2									
0201 INFLUENZA A VIRUS.....		14									
0203 INFLUENZA B VIRUS.....		2					2				
0301 PARAINFLUENZA VIRUS TYPE 1....		1									
0302 PARAINFLUENZA VIRUS TYPE 2....		4									
0303 PARAINFLUENZA VIRUS TYPE 3....	1	10									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....		5									
0500 RHINOVIRUS (ALL TYPES).....	1	8									
0600 MYCOPLASMA PNEUMONIAE.....	1	9							1		
0700 ORNITHOSIS-PSITTACOSIS.....		1									
0809 COXSACKIEVIRUS A9.....	2	1		3	1		5				
0904 COXSACKIEVIRUS B4.....				1							
0905 COXSACKIEVIRUS B5.....		3		2			3				
1007 ECHOVIRUS TYPE 7.....		2		7			1				
1009 ECHOVIRUS TYPE 9.....		1					1				
1011 ECHOVIRUS TYPE 11.....				2							1
1014 ECHOVIRUS TYPE 14.....				1							
1017 ECHOVIRUS TYPE 17.....				1							
1020 ECHOVIRUS TYPE 20.....			1								
1030 ECHOVIRUS TYPE 30.....				3							
1101 POLIOVIRUS TYPE 1.....		1									
1102 POLIOVIRUS TYPE 2.....		1					1				
1200 MUMPS VIRUS.....	1			3							
1300 HERPES VIRUS GROUP-NOT TYPED..											1
1301 HERPES SIMPLEX VIRUS NOT-TYPED											1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..	4	6						2			1
1303 VARICELLA-ZOSTER VIRUS.....				2							5
1306 HERPES SIMPLEX TYPE 1.....	5	7	2			1		1	1	1	80
1307 HERPES SIMPLEX TYPE 2.....	18	1								2	61
1502 PICORNA VIRUS-NOT TYPED.....							2				
1515 CONTAGIOUS PUSTULAR DERMATITIS (ORF VIRUS).....											2
1521 MEASLES VIRUS.....	1	1		2			1				5
1522 RUBELLA VIRUS.....	9	2	1								45
1532 HEPATITIS B ANTIGEN.....	54							79	1		
1535 HEPATITIS A ANTIBODY.....	1							6			
1541 CHLAMYDIA A - C.TRACHOMATIS...	3							3			
1556 CMV - CYTOMEGALOVIRUS.....	3	7				2		6		4	
1564 ROTAVIRUS.....							24				
9992 ROSS RIVER VIRUS.....	3	3									12
9993 ASTROVIRUS.....							1				
9994 SMALL VIRUS (LIKE) PARTICLE...							2				
Total.....	107	97	4	27	1	4	44	97	3	7	214

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 31/1/85 to 13/2/85 ...
 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	Gen-ital	Endo/sal gland	RES	Muscle/joint	Con-genital	PUO	Fever/mal-aise	Other	SIDS
0101 ADENOVIRUS TYPE 1.....										1
0103 ADENOVIRUS TYPE 3.....	1						1	3		
0108 ADENOVIRUS TYPE 8.....	2									
0111 ADENOVIRUS TYPE 11.....										1
0119 ADENOVIRUS TYPE 19.....	2									
0201 INFLUENZA A VIRUS.....				1						
0203 INFLUENZA B VIRUS.....								2		
0303 PARAINFLUENZA VIRUS TYPE 3....				1			1			
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....					1			1		
0600 MYCOPLASMA PNEUMONIAE.....							1			
0809 COXSACKIEVIRUS A9.....								1		1
0905 COXSACKIEVIRUS B5.....								3		
1007 ECHOVIRUS TYPE 7.....	1							1		
1009 ECHOVIRUS TYPE 9.....							1			
1017 ECHOVIRUS TYPE 17.....								1		
1030 ECHOVIRUS TYPE 30.....										1
1101 POLIOVIRUS TYPE 1.....										2
1102 POLIOVIRUS TYPE 2.....						1				
1200 MUMPS VIRUS.....			3				1			
1302 EPSTEIN-BARR VIRUS (EB VIRUS).				11				2		
1306 HERPES SIMPLEX TYPE 1.....	7	46					1	4	5	
1307 HERPES SIMPLEX TYPE 2.....	1	298								
1401 COXIELLA BURNETI.....							2			
1521 MEASLES VIRUS.....							4			
1522 RUBELLA VIRUS.....				1				2	5	
1532 HEPATITIS B ANTIGEN.....				2		8		1	12	
1535 HEPATITIS A ANTIBODY.....				1					1	
1541 CHLAMYDIA A - C.TRACHOMATIS...	2	169							6	
1556 CMV - CYTOMEGALOVIRUS.....		3					6	8	14	
9992 ROSS RIVER VIRUS.....						28		7		
9995 DENGUE.....							1			
Total.....	16	517	20	1	36	7	21	28	48	1

NOTIFIABLE DISEASES REPORTED IN AUSTRALIA

(Weeks 41 - 52)
7 October - 31 December 1984)

Bulletin ..85/4.

Disease	N.S.W.	VIC	QLD	S.A.	W.A.	TAS.	N.T.	A.C.T.	Total	CUMULATIVE TOTAL TO DATE FOR YEAR
Amoebiasis	7	1	1	4	3				16	52
Ankylostomiasis	1		2	14	1				18	14
Anthrax									—	—
Arbovirus infection	10			1					11	870
Brucellosis	6	1							7	15
Campylobacter infections	150	N.N.	N.N.	353	N.N.	N.N.	16	N.N.	519	1199
Chancroid	1			N.N.	1	N.N.			2	14
Cholera									—	1
Congenital rubella syndrome		N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	—	1
Diphtheria									—	—
Donovanosis		N.N.	11	N.N.	5	N.N.	7		23	260
Giardiasis	42	N.N.	N.N.	181	N.N.	N.N.	N.N.	N.N.	223	1025
Genital herpes	185	N.N.	122	16	N.N.	N.N.	6	1	330	1331
Gonococcal ophthalmia neonatorum		N.N.			N.N.	N.N.	2	N.N.	2	7
Gonorrhoea	567	257	395	125	236	12	218	16	1829	8753
Hepatitis A (infectious)	36	18	56	19	7	1	6	1	144	676
Hepatitis B (serum)	121	25	97	47	36	2	4	2	334	1556
Hepatitis - unspecified	15			5	5	N.N.	3		28	135
Hydatid disease									—	9
Lassa Fever			N.N.			N.N.	N.N.	N.N.	—	—
Legionnaires disease	1		N.N.		N.N.	N.N.	N.N.	N.N.	1	13
Leprosy	2		1	1	1		1	1	7	27
Leptospirosis	4	14	19	7	1	2			47	217
Lymphogranuloma venereum		N.N.	N.N.	N.N.	N.N.	N.N.			—	2
Malaria	17	6	33	31	6	1	5	7	106	638
Marburg Disease			N.N.			N.N.	N.N.	N.N.	—	—
Meningococcal infections	3		4	8		N.N.			15	96
Non-specific urethritis	158	N.N.	N.N.	275	N.N.	N.N.	3	N.N.	1036	4831
Ornithosis				2	1			1	4	42
Pertussis (whooping cough)	53	10	N.N.	19	N.N.	N.N.	N.N.	N.N.	82	261
Plague									—	—
Poliomyelitis									—	—
Q. fever	57	1	26	5	N.N.		N.N.		89	246
Rabies		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	167	44	59	69	16	19	57	9	440	2075
Shigella infections	23	1	16	4	7	1	25		77	419
Smallpox									—	—
Syphilis	103	33	90	42	26		225	8	531	2244
Tetanus	2			2					4	7
Trachoma		N.N.			N.N.	N.N.			—	1
Tuberculosis (all forms)	116	59	45	28	20	3	26	9	306	1227
Typhoid fever	8	1	4		1			1	15	51
Typhus (all forms)									—	8
Vibrio parahaemolyticus infections	1	N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	1	9
Yellow Fever									—	—
Yersinia enterocolitica infections	1	N.N.	N.N.		N.N.	N.N.	N.N.	N.N.	1	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.)

N.N. Not Notifiable

ADJUSTMENTS

Campylobacter infections	+1	South Australia
Hepatitis A	+1	Queensland
	+1	South Australia
Hepatitis B	-1	Queensland
Leptospirosis	+1	South Australia
Q.fever	+1	Queensland
	+1	South Australia
Salmonella infections	-2	South Australia
	-1	Australian Capital Territory
Tuberculosis	-2	Australian Capital Territory
Typhoid fever	+2	New South Wales