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VIRUS REPORTING SCHEME: A total of 1 383 reports were processed for this period.

Five cases of Q fever were reported from New South Wales. No occupational exposure data was provided for any of these cases. None of the five patients had been involved in the Q fever vaccine field trial conducted in South Australia.

Poliovirus type 3 was isolated from the faeces of a 6 month old male who died from Sudden Infant Death Syndrome in Western Australia.

Enterovirus type 71 (BRCR) was isolated from the faeces of a male suffering from meningitis in New South Wales.

Adenovirus type 3 was isolated from the faeces of a 7 year old female suffering from meningitis in Western Australia.

The previously reported outbreak of aseptic (viral) meningitis due to infection with echovirus types 11 and 5 (CDI 86/25;87/6) continued during this reporting period with New South Wales reporting 4 cases of meningitis due to echovirus type 5 and 6 cases due to echovirus type 11.

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Contributions are solicited, and do not preclude later publication elsewhere.

Material appearing in the Bulletin may be quoted provided suitable acknowledgment is made.

Figures given may be subject to revision.

HUMAN SALMONELLA, SHIGELLA, CAMPYLOBACTER, ENTEROPATHOGENIC E. COLI, V. CHOLERAЕ and V. PARAHAEMOLYTICUS INFECTIONS - AUSTRALIA, 1985

(Contributed by J. Taplin, Microbiological Diagnostic Unit, University of Melbourne).

In 1985, the National Salmonella Surveillance Scheme recorded 4922 Salmonella, 1152 Shigella and 2136 Campylobacter isolates from human sources compared with 4397, 707 and 1720 reports respectively in 1984(1). In addition, 62 isolates of enteropathic E. coli were reported, with one isolate of V. cholerae and one of V. parahaemolyticus. The distribution of isolates by State and Territory is presented in Table 1.

Table 2 presents an analysis of the most common salmonella serotypes by State and Territory. Table 3 presents a similar analysis of the most common shigella serotypes. Table 4 analyses the dominant phage types for isolates of Salmonella typhimurium from human sources in 1985.

1. CDI 86/1.

TABLE 1: Salmonella, Shigella, Campylobacter, E. coli, V. cholerae and V. parahaemolyticus isolations - Australia 1985

	STATE/TERRITORY								
	TOTAL	ACT	NSW	VIC	QLD	SA	NT	WA	TAS
Salmonella	4922	139	1241	715	1130	408	455	852	82
Shigella	1152	3	141	105	130	51	281	440	1
Campylobacter	2136	58	824	460	217	16	5	556	0
<u>E. coli</u>	62	4	8	39	11	0	0	0	0
<u>V. cholerae</u>	1	0	0	1	0	0	0	0	0
<u>V. parahaemolyticus</u>	1	0	0	1	0	0	0	0	0

TABLE 2: The ten most common Salmonella serotypes isolated from human sources 1985.

	STATE/TERRITORY								
	TOTAL	ACT	NSW	VIC	QLD	SA	NT	WA	TAS
<u>S. typhimurium*</u>	1996	102	777	353	164	250	37	267	46
<u>S. virchow</u>	271	3	23	16	211	0	7	4	2
<u>S. saintpaul*</u>	238	1	6	9	135	8	0	48	0
<u>S. chester</u>	153	0	23	3	45	6	35	40	1
<u>S. havana</u>	149	0	10	17	23	20	41	37	1
<u>S. muenchen</u>	145	0	22	4	46	5	24	44	0
<u>S. bovismorbificans</u>	141	2	44	10	6	19	9	50	1
<u>S. anatum</u>	106	0	5	6	56	5	12	22	0
<u>S. infantis</u>	87	0	15	16	15	4	6	31	0
<u>S. singapore</u>	84	3	34	12	9	4	6	15	1

* includes all serotypes.

TABLE 3: The most common *Shigella* isolates from human sources - Australia - 1985.

	TOTAL	STATE/TERRITORY							
		ACT	NSW	VIC	QLD	SA	NT	WA	TAS
<i>Sh. sonnei</i> biotype a	442	0	66	35	41	38	168	94	0
<i>Sh. flexneri</i> 6	191	0	8	9	6	1	5	162	0
<i>Sh. flexneri</i> 2a	189	0	12	6	15	8	72	76	0

TABLE 4: The ten most common *S. typhimurium* phage types in isolates from human sources - Australia 1986.

Phage type:	Number isolates
135	501
9	287
12a	95
64	84
4	75
26	54
5	52
101	51
179	43
44	40

SALMONELLA SURVEILLANCE - NON-HUMAN ISOLATES - SUMMARY 1985

(Contributed by J. Taplin, J. Morris, and J. Powling, Microbiological Diagnostic Unit, University of Melbourne).

A total of 7221 salmonella isolates from non-human sources were notified to the National Salmonella Surveillance Scheme (NSSS) in 1985, compared to 9505 in 1984. Cultures were grouped into 5 categories. The previous category 'Animal products' was combined with 'Foodstuffs'. Table 1 presents an analysis of salmonella reports by category and State.

The results generally reflect the intensity and focus of the State sampling and surveillance schemes. Reports from Western Australia reflect that State's monitoring of water supplies, food processing environments, abattoirs and animals; whilst those from Victoria reflect their interest in dairy factory environments and in poultry products.

7192 of the isolates belonged to 124 serotypes and 29 were not typable. 50 phage types of *S. typhimurium* accounted for 772 isolates. 110 isolates of *S. typhimurium* were not phage typed; 42 were untypable and 71 reacted with typing phages but did not conform to a known type. 143 of the 323 isolates of *S. bovis* belonged to 14 different phage types and 2 cultures reacted with typing phages but could not be placed in a recognized type.

TABLE 1: Source of salmonella isolations collected by NSSS-1985
No. of Cultures Isolated From

CATEGORY	ACT	NSW	VIC	QLD	SA	WA	TAS	NT	TOTAL
WATER & ENVIRONMENT	6	336	512	54	22	2590	54	7	3581
FOODSTUFFS	13	147	148	22	4	1052	7	26	1419
ANIMAL	-	105	352	73	87	1155	54	60	1886
EGGS & EGG PRODUCTS	-	25	260	-	-	8	-	-	293
MILK & MILK PRODUCTS	-	1	41	-	-	-	-	-	42
TOTAL ISOLATIONS IN 1985	19	614	1313	149	113	4805	115	93	7221
TOTAL ISOLATIONS IN 1984	42	822	2579	397	135	5288	65	177	9505

The 10 most common salmonella serotypes isolated from animals in 1985 are presented in Table 2. A detailed listing of serotypes is available from the authors on request.

TABLE 2: THE 10 MOST COMMON SALMONELLA SEROTYPES IN ANIMALS, 1985

SEROTYPE	NO.	PERCENTAGE	PREDOMINANT STATE (number)
<u>S.typhimurium</u>	345	18.3	WA (250), VIC (82)
<u>S.muenchen</u>	208	11.0	WA (200)
<u>S.sofia</u> SG II	189	10.0	WA (170)
<u>S.dublin</u>	171	9.1	VIC (146)
<u>S.infantis</u>	139	7.4	WA (129)
<u>S.havana</u>	95	5.0	WA (86)
<u>S.bovismorbificans</u>	79	4.2	WA (56)
<u>S.arizonae</u>	73	3.9	VIC (25), WA (19)
<u>S.anatum</u>	43	2.3	WA (21), VIC (17)
<u>S.wandsbek</u> SG II	35	1.9	WA (33)
TOTAL	1377	73.0	

SALMONELLA TYPHIMURIUM PHAGE-TYPE 12a OUTBREAK IN TASMANIA
(Contributed by J. Taplin and J. Morris, Microbiological Diagnostic Unit, University of Melbourne).

To 17 March 1987, Laboratories in Tasmania have submitted 34 cultures of Salmonella to the Microbiological Diagnostic Unit. 18 cultures have been identified as S. typhimurium phage type 12a, a type not usually isolated in Tasmania (Table 1).

TABLE 1

Isolates of S. typhimurium phage-type 12a for 1986-87

	ACT	NSW	VIC	QLD	SA	WA	TAS	NT	TOTAL
<u>1986</u>									
1st quarter	0	5	2	4	4	0	1	0	16
2nd quarter	0	5	6	4	4	1	0	1	21
3rd quarter	0	5	0	3	0	0	0	0	8
4th quarter	0	9	2	1	1	0	1	0	14
<u>1987</u>									
1 January-17 March	0	1	1	0	0	1	18	0	21
Total	0	25	11	12	9	2	20	1	80

In 1985, of a total of 82 salmonella isolates notified from Tasmania to the National Salmonella Surveillance Scheme (NSSS), 36 (44%) were S. typhimurium, of which only 2 (6%) were phage-type 12a. This compares with a total of 34 isolates for 1987 of which 23 (68%) have been identified as S. typhimurium and of these 18 (78%) are phage-type 12a. Eleven of these isolates came from the Launceston area, 4 from north-west Tasmania and 3 from Hobart.

The age distribution of these isolates is shown in Table 2.

TABLE 2

Age distribution - Tasmanian S. typhimurium phage-type 12a isolates

Age (yrs)	0-2	3-5	6-10	11-15	16-20	NS	Total
	2	3	1	0	8	4	18

In 1986 S. typhimurium phage-type 12a was associated predominantly with chickens in Queensland and Tasmania and cattle in South Australia (Salmonella Reference Laboratory, Adelaide, Surveillance Reports).

ZOONOTIC SALMONELLOSIS, SCOTLAND

[Based on Communicable Diseases Weekly Report, Scotland (1986) 50: 7 - 9]

A recent outbreak of Salmonellosis in Scotland demonstrates how easily Salmonella can be transmitted from animals to humans if personal hygiene is neglected. The extent of zoonotic infection in this episode was determined through the cooperation of medical and veterinary officers.

A farmer's wife from farm A, case 1, presented at hospital a day after developing severe abdominal pain and diarrhoea. Faecal samples cultured Salmonella typhimurium phage type 204.

Investigation of the case revealed that a neighbouring farmer (case 2) from farm B and two sons (cases 3 and 4) had subsequently suffered acute diarrhoea and abdominal pain. They had not sought medical treatment and the episode lasted less than 24 hours. Faecal samples were "positive" for case 2 and 3 but "negative" for case 4.

It appeared that farms A and B worked closely together.

On the Sunday before case 1 took ill, sheep from both farms had been dipped on farm B in a preparation containing 10% phenol.

On Wednesday, case 1 handled a dead sheep which had been scouring profusely. The rest of the flock appeared healthy.

On Thursday, case 1 developed her illness. The neighbouring farmer, assisted by his sons, dosed his sheep for worms "because they were a bit loose".

Friday, the day case 1 was admitted to hospital, 2 sheep died on farm B. By Sunday when cases 2, 3 and 4 became ill, 7 of the 12 affected sheep on farm B had died.

Discussion

Since the incubation period with Salmonella varies from 6-72 hours, usually 12-36 hours, it is likely that cases 2 to 4 were infected while handling their sheep at worming or over the following days. It is likely that case 1 was infected when she handled the dead sheep and this animal must have been infected at dipping three days previously.

It is fair to assume that sheep on farm B had been excreting Salmonella for several days before deaths occurred and the disease only became acute and fatal due to some other factor (such as a sudden change of feeding, pasteurellosis or the stress associated with dipping).

How the sheep became infected was more difficult to establish. Husbandry techniques on the farms appeared adequate. A few months previously, however, a recently purchased cow on farm B had apparently suffered from lead poisoning, the source of which was assumed to be paint in a shed. The cow scoured badly and was destroyed. The shed had not been cleaned since that episode. Subsequently, sheep from farm B were housed before and after dipping in the same building in which the cow was ill. Circumstantial evidence therefore implicates the cow as a carrier of Salmonella organisms.

GONOCOCCAL SURVEILLANCE (AUSTRALIA)

(Contributed by the Australian Gonococcal Surveillance Programme (AGSP). Co-ordinator Dr J.W. Tapsall, The Prince of Wales Hospital, N.S.W.)

This report provides details of penicillin sensitivity of gonococci isolated in participating laboratories and examined by standard techniques (1). The periods covered here are

July - September 1986 when 1073 strains were examined, and
October - December 1986 when 1077 isolates were available
(Table 1)

Penicillin resistance in Neisseria gonorrhoeae occurs either in the form of chromosomally controlled "intrinsic resistance", manifested as incremental increases in the minimal inhibitory concentration (MIC) of penicillin, or else as β -lactamase-mediated resistance appearing when the organism acquires an appropriate plasmid (penicillinase-producing N. gonorrhoeae-PPNG).

July-September - 1986.

There was little change in the percentage of strains fully sensitive (MIC 0.008 mg/L) or less sensitive (MIC 0.12 mg/L) to penicillin in the five major centres in this quarter when these were compared with the levels of resistance observed in the previous quarter (CDI 86/21). In the previous quarter (April-May 1986) there had been a significant increase in levels of intrinsic resistance in Adelaide and this trend was maintained during July-September 1986.

PPNG were isolated in all centres with the exception of Hobart. In Sydney PPNG represented 30% of all isolates and significant numbers of PPNG were also detected in Melbourne, Brisbane and Perth. The majority of infections with PPNG seen in Sydney were acquired locally and local acquisition of PPNG was also reported from most other centres emphasising the increasing importance of these strains.

October-December 1986

The levels of intrinsic resistance recorded in this quarter differed little from those observed in the previous six months with the majority of isolates in all centres falling into the "less sensitive" category.

The numbers of PPNG isolated in this period were again high with increased numbers of these strains being found in Adelaide and Brisbane. Again there was evidence of widespread local acquisition of PPNG in Sydney, Melbourne and Brisbane, and notably on this occasion Adelaide. Further, it was also noted that in some centres PPNG were isolated from an increasing number of patients who had acquired their infection elsewhere in Australia, a phenomenon which will undoubtedly result in the further spread of resistant strains.

REFERENCES

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

TABLE 1: PENICILLIN SENSITIVITY OF ISOLATES OF N. GONORRHOEAE

Reporting Period	Centre	Sensitive(a)	Percentage of isolates Less Sensitive(b)	PPNG
July- September 1986	Brisbane	24.3	56.8	12.6
	Sydney	6.8	49.1	30.0
	Melbourne	17.0	49.8	16.6
	Adelaide	27.2	60.2	5.2
	Perth	23.5	37.8	12.3
October- December 1986	Brisbane	23.7	55.9	16.1
	Sydney	6.2	49.3	23.2
	Melbourne	14.7	56.0	12.9
	Adelaide	20.8	55.8	11.7
	Perth	28.4	32.9	14.8

(a) Sensitive. MIC = 0.008 mg/L + 1 doubling dilution

(b) Less Sensitive. MIC = 0.12 mg/L + 1 doubling dilution

PREVENTION OF MENINGOCOCCAL INFECTION IN A DAY NURSERY - SCOTLAND
(based on CDS 1987, 87/6: 7)

A one year old male presented with meningococcal meningitis at King's Cross Hospital, Dundee. The case details, as per standard procedures, were notified to the Tayside Health Board's Community Medicine Specialist, together with the information that he attended a Day Nursery. Subsequent investigations revealed that the boy was a cousin of a case of meningococcal infection which presented during the previous week, and that he had been at the Day Nursery a few hours prior to the onset of his symptoms.

A two-day prophylactic course of rifampicin (1,2) was arranged for the household and close family contacts. In view of the length and closeness of contact with the case, it was decided that all children attending the Day Nursery should also receive a prophylactic course of rifampicin.

A circular to the parents/guardian of each child was prepared and was distributed by the Officer-in-Charge of the Day Nursery as the children were collected. This letter advised that the child should see their family physician. Meanwhile, the family general practitioner of each child was contacted and informed to expect a visit from the child, alerting him of the circumstances and providing advice on the prophylactic regimen.

No further cases of meningococcal infection have occurred in the community since.

In view of the importance of meningococcal infection and the need to deal with such a situation efficiently, the procedure followed was evaluated by distribution of a follow-up questionnaire to the general practitioner of each child one week later. The questionnaire enquired whether the child had attended the general practitioner; if so, when; whether prophylactic antibiotics were prescribed; and whether the procedure followed was acceptable. Other comments were also solicited.

RESULTS:

A total of 47 children attended the Day Nursery in question, and were registered with 31 different general practitioners.

Forty five (96%) of the 47 questionnaires were returned. Two children (4%) had not seen their doctor, and no date of visit was provided for a further 9 (20%) of the questionnaires. The data on the remaining 34 children is given in Table 1.

TABLE 1: Prevention of Meningococcal Infection in a Dundee Day Nursery. Response Rate to Preventive Treatment Call - Letter.

	<u>Cumulative Total</u>
Attended within one day of letter	12 (27%)
Attended within two days of letter	24 (54%)
Attended within three days of letter	32 (71%)

All of the children who attended their doctors received prophylactic antibiotics. The general practitioners were unanimous in their approval of the procedures followed. The only additional comment received suggested that a follow-up letter to the general practitioner would be useful.

DISCUSSION:

It was apparent from the questionnaire returns that some general practitioners or their health visitors made home visits to deliver prescriptions or to encourage attendance at the surgery.

It was of concern that 4% (possibly 8% if the two questionnaires which were not returned are included) of the children were not given prophylaxis. In future, a follow-up letter to the general practitioner will be sent which will include the suggestion of further action by the general practitioner should the child not present at the surgery.

A second refinement to the system would be to stress, both in contacts with the parents/guardian and the family general practitioner, the need for prompt action, as prophylaxis was initiated in some cases up to five days after the distribution of the letter. This does not accord with the recommendation that prophylaxis should be given within two days (1) and is of concern in view of the fact that 70% of secondary cases occur within seven days of the index case (3).

To prevent the spread of meningococcal infection, action should be immediate, and preferably in accordance with a pre-existing plan.

CDI EDITORIAL COMMENT:

Chemoprophylaxis of meningococcal infection with rifampicin has been discussed previously in CDI (4).

The treatment regimen for case contacts of meningococcal infections should follow that recommended by the NH&MRC for H. influenzae (5). The NH&MRC comments and recommendations are:

"The Council was advised that Haemophilus influenzae type B caused a variety of serious childhood diseases including epiglottitis, meningitis and pneumonia.

If noted that because of the communicable nature of H. influenzae type B infection, the need for effective prophylaxis for contacts had a high priority.

The Council recommended that in any household in which a case of H. influenzae type B infection has occurred and in which another child less than four years of age resides, all members of the household, including adults, should receive rifampicin in a dose of 20mg/kg once daily (maximum dose 600mg/day) for four days; the dose for neonates (less than one month) is 10mg/kg once daily for four days."

1. JAMA (1976) 235:261.
2. Lancet (1984) i:101.
3. BMJ (1981) 282:2134.
4. CDI (1986) 86/4:8.
5. NH&MRC: Report of the Hundredth Session, November 1985. (1986):29.

YERSINIA ENTEROCOLITICA - A CASE REPORT

(Contributed by Dr D. Mitchell and Dr P Collignan, Woden Valley Hospital, Infectious Diseases Department)

Yersinia enterocolitica, biotype 3, serotype 0:5,27 was cultured from the blood of a 67 year old male patient who was transferred to Woden Valley Hospital following treatment failure with intravenous cephalothin and metronidazole at a country hospital.

Following laboratory confirmation that the organism was sensitive to gentamicin, chloramphenicol, cephotaxime and trimethoprim, but resistant to ampicillin, sulphonamide and tetracycline; the patient responded slowly to treatment with intravenous gentamicin 80mg three times a day, but improved more rapidly when oral trimethoprim 300mg daily was added.

Y. enterocolitica could not be isolated from faecal specimens obtained after antibiotic treatment had commenced.

Medical history taken on admission recorded fever, rigors, nausea and profuse watery diarrhoea during the previous five days. Physical examination indicated signs of chronic liver disease, hepatosplenomegaly and hydroperitonia. The patient appeared bronzed and jaundiced, complained of osteoarthritic changes in the second and third metacarpal joints of both hands, and gave a history of glucose intolerance requiring insulin therapy.

Subsequent biochemical studies of iron metabolism and histological studies of liver biopsy indicated results consistent with idiopathic haemochromatosis.

COMMENT

Systemic yersiniosis is associated with iron overload states in patients receiving desferrioxamine which act as a siderophore for Yersinia⁽¹⁾. However, this was not the mechanism in the above case.

In haemachromatosis excessive iron absorbed from the gut saturates transferrin and then non-specifically binds to albumin and other serum proteins which subsequently act as available iron reservoir for dependent micro-organisms⁽²⁾. The mechanism of iron uptake into the organism is not known, however.

Cirrhosis was found to be the commonest underlying condition associated with a mortality rate of 67% in a review of 34 patients with disseminated yersiniosis haemachromatosis, compared to a mortality rate of 38% overall for yersinia septicaemia⁽³⁾.

REFERENCES

1. Cont. Microb. Immun. (1979) 5:277-82
2. Rev. Infect. Dis. (1983) 5:759-73
3. Arch. Intern. Med. (1976) 126:1305-08

LEGIONNAIRES' DISEASE IN VICTORIA - 1983-1985

[Contribution by Dr G.J. Rouch, Dr S. Ng (Health Department Victoria), Dr M. Peel (Melbourne University); Dr B. Dwyer (Fairfield Hospital)].

Twenty-four cases of Legionnaires' Disease were notified to the Health Department Victoria in the period 1983-1985. The source of infection was not identified in 20 of these cases. Three cases were considered nosocomial and one was associated with a cooling tower. Cases were sporadic in nature. Diagnosis was based on:-

1. demonstration of a greater than fourfold rise in antibody titre of Legionella pneumophila in paired sera, or a raised IgM Antibody titre in a single specimen (17/24 cases);
2. detection of the organism from clinical specimens by culture or DFA staining (12/24 cases);
3. combination of the above methods (5/24 cases).

L. pneumophila accounted for the infection in the majority of cases, with serogroup 1 being the most common serogroup implicated (Table 1).

TABLE 1. IDENTIFICATION OF LEGIONELLA SPECIES FROM CASES OF LEGIONNAIRES' DISEASE IN VICTORIA - 1983-1985

Year	Total cases	<u>L. pneumophila</u>	<u>L. pneumophila serogroup 1</u>	<u>Legionella ssp. not L. pneumophila</u>
1983	11	11	7	0
1984	5	4	0	1
1985	8	6	6	2

In 1984 and 1985, species other than L. pneumophila were identified for the first time in Victoria. These were:-

L. micdadei in a 52 year old male.

L. bozemanii serogroup 2 in a 48 year old male.

L. longbeachae serogroup 2 in a 74 year old male.

In these 3 cases, diagnosis was based on the isolation of the organism from clinical specimens alone (post mortem lung tissue in all 3, including bronchial washings and sputum from one other).

Legionnaires' Disease, caused by L. micdadei was diagnosed in a 52 year old man who was immunosuppressed following a cadaver renal transplant for polycystic kidneys. During the post-operative period, he developed an acute attack of dizziness, palpitations and severe dyspnoea. Atrial fibrillation and, subsequently, atypical pneumonia were diagnosed; he then developed septicaemia, mental confusion and finally died from a myocardial infarct. The organism was isolated from a culture of post-mortem lung tissue. Epidemiological investigations suggested the source of his infection to be nosocomial.

Environmental tests of the water from the cooling tower, water distribution system (showers) and nebulizers were negative for Legionella spp. Serology on dialysis patients attending the Renal Unit was also negative for L. micdadei.

L. bozemanii serogroup 2 was diagnosed in a 48 year old man, also immunosuppressed by treatment for hairy-cell leukaemia. He presented with a left lower lobe pneumonia which rapidly involved both lungs; the pneumonia was complicated by an acute myocardial infarct to which he succumbed. The organism was isolated from specimens of sputum, bronchial washings and post-mortem lung tissue. No serology was done. The source of his infection was not determined; domestic water sampled was negative for Legionella spp.

The third non-pneumophila isolation was L. Longbeachae serogroup 2 recovered from post mortem lung tissue of a 74 year old man who died 25 minutes after presenting at a casualty department with a 2 day history of lethargy, nausea, diarrhoea and severe dyspnoea. No serum was available. The source of his infection was undetermined; water samples from his shower at home did not grow the organism.

RISK FACTORS

13 of the 24 cases (54%) smoked and 16 (66.6%) had underlying conditions which included leukaemia, lymphoma, autoimmune disease, chronic lung disease, hypertension and alcoholism.

The most common complication was renal dysfunction. 13 of 24 (54%) had renal involvement with 9 of these requiring dialysis for renal failure.

The youngest patient was 21 and the oldest 92 (mean age: 53 years). The combined fatality rate for the three years was 54% (Table 2).

TABLE 2: Legionnaires' Disease in Victoria 1983-1985.

Year	Total cases	Male	Female	Deaths	Fatality rate (%)
1983	11	11	0	8	72.7
1984	5	3	2	2	40.0
1985	8	7	1	3	37.5
TOTAL	24	21	3	13	54.2

NOTICE TO READERS:

INTERNATIONAL LEPTOSPIROSIS INFORMATION EXCHANGE NEWSLETTER

A biannual International Leptospirosis Information Exchange newsletter (ILIX) has recently commenced publication. The aim of the newsletter is to provide an informal exchange of data, notice of current research and publications, a vehicle for seeking answers to specific problems, and a vehicle for publicising regional meetings, taxonomic subcommittee meetings etc..

The Editor is currently seeking articles for the second issue (closing date is 30 June 1987). Articles should be brief (no longer than half a page of typewritten text) and preferably in English. Readers interested in submitting articles for ILIX, or in being placed on the mailing list, should write to the Editor:

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

REPORTING PERIOD - 6-4-87 to 19-4-87 BULLETIN NUMBER 87/8
 VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES

VIRUS OR VIRAL ANTIGEN	ICPMR (NSW)/ MVH (ACT)	RAHC (NSW)	PHH/ POW (NSW)	FAIR- FIELD (VIC)	RCH (VIC)	IMVS (SA)	STATE LAB (QLD)	STATE LAB (WA)	Total
0100 ADENOVIRUS NOT TYPED.....	4				11	1	13	2	31
0101 ADENOVIRUS TYPE 1.....	2			1					3
0102 ADENOVIRUS TYPE 2.....						4		1	5
0103 ADENOVIRUS TYPE 3.....	2							1	3
0111 ADENOVIRUS TYPE 11.....				1					1
0119 ADENOVIRUS TYPE 19.....				1		1		1	3
0133 ADENOVIRUS TYPE 33.....	1								1
0199 ADENOVIRUS TYPING PENDING.....	1				3				4
0201 INFLUENZA A VIRUS.....	2								2
0203 INFLUENZA B VIRUS.....	2								2
0301 PARAINFLUENZA VIRUS TYPE 1.....						2			2
0302 PARAINFLUENZA VIRUS TYPE 2.....					2		1	1	4
0303 PARAINFLUENZA VIRUS TYPE 3.....		3			3		2		8
0400 RESPIRATORY SYNCYTIAL VIRUS (RS)...	2	1		1	1	2	8	2	17
0500 RHINOVIRUS (ALL TYPES).....				3	17	5			25
0600 MYCOPLASMA PNEUMONIAE.....	11	1			5	1	1	2	21
0700 ORNITHOSIS-PSITTACOSIS.....	2								2
0903 COXSACKIEVIRUS B3.....	1					3			4
0904 COXSACKIEVIRUS B4.....		1							1
1000 ECHOVIRUS NOT TYPED.....							3		3
1003 ECHOVIRUS TYPE 3.....	2	1		1					4
1004 ECHOVIRUS TYPE 4.....	1								1
1005 ECHOVIRUS TYPE 5.....	2	1		5					8
1009 ECHOVIRUS TYPE 9.....	1								1
1011 ECHOVIRUS TYPE 11.....	5			5			1		11
1014 ECHOVIRUS TYPE 14.....	1								1
1100 POLIOVIRUS NOT TYPED.....							1		1
1102 POLIOVIRUS TYPE 2.....		1				1			2
1103 POLIOVIRUS TYPE 3.....	1							1	2
1200 MUMPS VIRUS.....	2							1	3
1300 HERPES VIRUS GROUP-NOT TYPED.....	24				1	4		2	31
1301 HERPES SIMPLEX VIRUS NOT-TYPED.....		1						3	4
1302 EPSTEIN-BARR VIRUS (EB VIRUS).....	14	1				1		11	27
1303 VARICELLA-ZOSTER VIRUS.....	3	2				1	3	1	10
1306 HERPES SIMPLEX TYPE 1.....	43			51	1	22	39	25	181
1307 HERPES SIMPLEX TYPE 2.....	137			65		17	75	67	361
1399 HERPES VIRUS TYPING PENDING.....				6	2				8
1401 COXIELLA BURNETI.....	4					1			5
1402 OTHER RICKETTSIAE.....							1		1
1502 PICORNA VIRUS-NOT TYPED.....	9						9		18
1521 MEASLES VIRUS.....	2	1						2	5
1522 RUBELLA VIRUS.....	3			2		1		2	8
1532 HEPATITIS B ANTIGEN.....	74	3		14		41	30	19	181
1535 HEPATITIS A ANTIBODY.....	6			6		4	1	3	20
1541 CHLAMYDIA A - C TRACHOMATIS.....	43	2		31	1	43	16	20	156
1543 CHLAMYDIA A - LGV TYPE.....	7						25		32
1556 CMV - CYTOMEGALOVIRUS.....	4			45	2	3	4	10	68
1564 ROTAVIRUS.....	12				1	17	12		42
1571 ENTEROVIRUS TYPE 71 (BRCR).....				1					1
1599 ENTEROVIRUS TYPING PENDING.....		3			5	1			9
9992 ROSS RIVER VIRUS.....	4						23	11	38
9994 SMALL VIRUS (LIKE) PARTICLE.....	1								1
Total.....	435	22		239	55	176	268	188	1,383

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 6-4-87 to 19-4-87 BULLETIN NO 87/8

Viral Identifications by Clinical Information Table 1.

Code 00,99 -No ill or data; 01,02,11,12 -Respiratory; E3 -Enceph-

alitis; M3 -Meningitis; 04 -Paralysis; 05,13 -CNS other unspec.;

07,49 -GI; 17,47 -Hepatic; 19 -CVS; 89 -Urinary; 06 -Skin/mucous.

VIRUS OR VIRAL ANTIGEN	No-ill or data	Respir atory	Enceph alitis	Mening -itis	Para- lysis	CNS other unspec	GI	Hepa -tic	CVS	Urin -ary	Skin/ mucs memb
0101 ADENOVIRUS TYPE 1.....	1						1				1
0102 ADENOVIRUS TYPE 2.....		5									
0103 ADENOVIRUS TYPE 3.....				1							
0111 ADENOVIRUS TYPE 11.....										1	
0119 ADENOVIRUS TYPE 19.....		1									
0133 ADENOVIRUS TYPE 33.....							1				
0201 INFLUENZA A VIRUS.....		2									
0301 PARAINFLUENZA VIRUS TYPE 1....		2									
0302 PARAINFLUENZA VIRUS TYPE 2....		4									
0303 PARAINFLUENZA VIRUS TYPE 3....		8									
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....	4	12									
0500 RHINOVIRUS (ALL TYPES).....		17	1				1				
0600 MYCOPLASMA PNEUMONIAE.....	3	17									
0700 ORNITHOSIS-PSITTACOSIS.....		2									
0903 COXSACKIEVIRUS B3.....		1							1		
0904 COXSACKIEVIRUS B4.....					1						
1000 ECHOVIRUS NOT TYPED.....		2				1					
1003 ECHOVIRUS TYPE 3.....	2						1				1
1004 ECHOVIRUS TYPE 4.....		1									
1005 ECHOVIRUS TYPE 5.....	1	1			4		1				
1009 ECHOVIRUS TYPE 9.....	1										
1011 ECHOVIRUS TYPE 11.....	4				6	1					
1102 POLIOVIRUS TYPE 2.....	1										
1103 POLIOVIRUS TYPE 3.....	1										
1200 MUMPS VIRUS.....			2								
1300 HERPES VIRUS GROUP-NOT TYPED..											2
1301 HERPES SIMPLEX VIRUS NOT-TYPED			1								1
1302 EPSTEIN-BARR VIRUS (EB VIRUS)..	6										4
1303 VARICELLA-ZOSTER VIRUS.....	4										6
1306 HERPES SIMPLEX TYPE 1.....	12	11								4	86
1307 HERPES SIMPLEX TYPE 2.....	22										98
1401 COXIELLA BURNETI.....								1			
1402 OTHER RICKETTSIAE.....	1										
1521 MEASLES VIRUS.....		1		1							
1522 RUBELLA VIRUS.....											6
1532 HEPATITIS B ANTIGEN.....	65				1			98			
1535 HEPATITIS A ANTIBODY.....	5							15			
1541 CHLAMYDIA A - C.TRACHOMATIS...	19									1	
1543 CHLAMYDIA A - LGV TYPE.....	25										
1556 CMV - CYTOMEGALOVIRUS.....	6	8	1				1	2		7	1
1564 ROTAVIRUS.....	9						33				
1571 ENTEROVIRUS TYPE 71 (BRCR)....					1						
9992 ROSS RIVER VIRUS.....	9	2									4
9994 SMALL VIRUS (LIKE) PARTICLE...							1				
Total.....	201	97	5	15		2	40	116	1	13	210

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

PERIOD : 6-4-87 to 19-4-87 BULLETIN NO 87/8

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;

68 -Fever/malaise; 09 -Other; A1 -SIDS ...

VIRUS OR VIRAL ANTIGEN	Eye	Genital	Endo/sal gland	RES	Muscle/joint	Congenital	PUO	Fever/malaise	Other	SIDS
0103 ADENOVIRUS TYPE 3.....	2									
0119 ADENOVIRUS TYPE 19.....	3									
0203 INFLUENZA B VIRUS.....								2		
0400 RESPIRATORY SYNCYTIAL VIRUS (RS).....										1
0500 RHINOVIRUS (ALL TYPES).....							1	3		
0600 MYCOPLASMA PNEUMONIAE.....										1
0903 COXSACKIEVIRUS B3.....							1			1
1005 ECHOVIRUS TYPE 5.....								2		
1014 ECHOVIRUS TYPE 14.....										1
1102 POLIOVIRUS TYPE 2.....										1
1103 POLIOVIRUS TYPE 3.....										1
1200 MUMPS VIRUS.....			1							
1301 HERPES SIMPLEX VIRUS NOT-TYPED										2
1302 EPSTEIN-BARR VIRUS (EB VIRUS).			8	2			2	4		2
1306 HERPES SIMPLEX TYPE 1.....	3	65								1
1307 HERPES SIMPLEX TYPE 2.....		241				1				
1399 HERPES VIRUS TYPING PENDING...		5								1
1401 COXIELLA BURNETI.....							4			
1521 MEASLES VIRUS.....					1		1			1
1522 RUBELLA VIRUS.....			1		1					
1532 HEPATITIS B ANTIGEN.....										18
1541 CHLAMYDIA A - C.TRACHOMATIS...	3	131								1
1543 CHLAMYDIA A - LGV TYPE.....		7								
1556 CMV - CYTOMEGALOVIRUS.....				1		3	3	3		35
9992 ROSS RIVER VIRUS.....			2		23			8		
Total.....	11	449	12	3	25	4	12	22	66	1