COMMUNICABLE DISEASES INTELLIGENCE

ISSN 0725-3141 VOLUME 15 NUMBER 17 26 August 1991

CONTENTS

Editor Robert Hall

Editorial Staff Jenny Hargreaves, Evon Bowler, Marcus Hodge, Lenore Cupitt and Michelle Jozing.

CDI is produced fortnightly by:

Communicable Diseases Section
Department of Health, Housing and Community Services
GPO Box 9848 ACT 2601.
Fax: (06) 289 7802 Telephone: (06) 289 1555

DEPARTMENT OF HEALTH, HOUSING AND COMMUNITY SERVICES

COMMUNICABLE DISEASES NETWORK-AUSTRALIA
A National Network for Communicable Diseases Surveillance

SALMONELLA SURVEILLANCE, AUSTRALIA, FOURTH QUARTER REPORT 1990

(Reproduced with acknowledgement from the National Salmonella Surveillance Scheme Quarterly Report, (Editor Joan Powling) June 1991)

Summary

There were 1099 Australian acquired cases of salmonella notified during this quarter which was a 24 percent decrease from the total for the same period in 1989 (1433). There were 165 Australian acquired cases of shigella as against 207 for the corresponding period, a decrease of 20 percent.

There was a decrease in the salmonella case rate per 100,000 head of population in all states of Australia by comparison to the same period last year. The biggest percentage decreases in cases rates were recorded in South Australia (49%), the Northern Territory (48%) and Western Australia (38%).

S Typhimurium again headed the top ten salmonella serovars with 382 cases involving 32 phage types and accounting for 35 percent of the total Australian acquired cases of salmonella. **S Typhimurium 9** was the most common phage type with 75 cases, 85 percent of which were from New South Wales and Victoria. The second most common serovar was **S Bovismorbificans** with 60 cases, notified mostly from New South Wales where an outbreak of phage type 21 commenced in Sydney in mid-December.

The top ten salmonella serovars accounted for 69 percent of the Australian acquired cases.

Seven outbreaks were notified during this quarter, four of which were associated with serovars from the top ten. These were **S Havana** (NSW), **S Typhimurium** phage types **2** (Tas) and **135** (Vic), **S Heidelberg** phage type **2** (Qld) and **S Bovismorbificans 21** (NSW).

There were 92 serovars of salmonella isolated from the 1099 cases (86 serovars, Q4/'89). Of these 77 were from subgenus I (10 phage types of S Bovismorbificans and 32 of S Typhimurium), 3 from subgenus II and 2 from subgenus III (S Arizonae).

New and unusual salmonella serovars notified during the quarter included **S Isangi** (F/50 WA ex Bali), **S London** (F/19 SA ex Bali), **S London var 15+** (M/71 NSW), **S Panama** (M/1 NT, M/44 Qld ex Thailand) and **S Sandiego** (M/ SA).

S Isangi was isolated from Sydney sewage during August 1990 and from a traveller returning to Victoria from Thailand in 1989. There were two isolates (NSW & WA) of **S London** in the last quarter and one of **S London var 15+** (NSW). **S Panama** was isolated from Sydney sewage in September. **S Sandiego** was isolated in Western Australia (F/7), and also from a freshwater crocodile at Kununurra, both in the second quarter of 1990.

Uncommon phage types of **S Typhimurium** were **125**, **42** (NSW) and **78** (SA).

Salmonella case rates

The total number of cases acquired in Australia was 1099. There were 100 follow-ups, 7 cases from migrants and refugees and 111 cases acquired overseas (Tables 1 and 2; Figure).

Figure. Case rates per 100,000 for Salmonella infection, 4th Quarters 1989 and 1990.

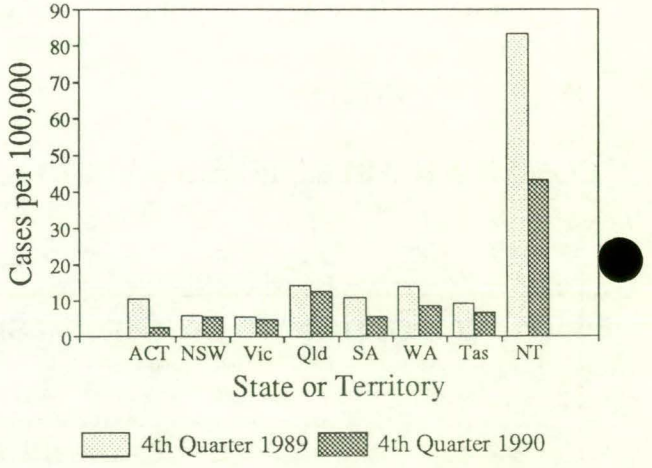


Table 1. Total number of reports received

	ACT	NSW	Vic	Qld	SA	WA	Tas	NT	TOTAL
Salmonella	6	363	233	363	88	153	35	76	1317
Shigella	3	24	21	11	6	59	-	80	204
Vibrio	-	13	2	-	-	-	-	-	15
Yersinia	-	15	4	11	1	-	-	-	31
TOTAL	9	414	260	385	95	212	35	156	1567

Table 2. Case rates per 100,000 for Salmonella infections

	ACT	NSW	Vic	Qld	SA	WA	Tas	NT	TOTAL
4th Quarter '90	2.4	5.5	4.6	12.5	5.5	8.6	6.9	43.3	1099
3rd Quarter ' 90	2.8	4.1	2.9	7.7	5.3	6.7	3.7	41.3	792
4th Quarter '89	10.4	5.8	5.5	14.1	10.7	13.8	8.9	83.3	1429
4th Quarter '88	5.6	5.5	5.5	19.8	5.9	12.0	4.6	56.2	1400

Table 3. Isolations from blood, urine and unusual sites

Bacteraemias excluding enteric fever (14):		
TYPE	SEX/AGE	STATE
S Agona	M/<1	NSW
S Chester	M/5	Qld
S Chester	F/<1	Qld
S Dubin	F/ns	NSW
S Hadar	F/78	Vic
S havana	M/35	NSW
S Heidelberg	M/2	NSW
S Heidelberg	F/2	Vic
S Oranienburg	F/29	NT
S Typhimurium 45	M/42	NSW
S Typhimurium 9	M/ns	NSW
S Typhimurium 9	M/3	NSW
S Virchow	M/18	NT
V cholerae non O1	F/44	Vic

Urines (16):		
TYPE	SEX/AGE	STATE
S Anatum	F/58	Qld
S Anatum	F/77	Vic
S Arizonae 61:1<v:z35	F/80	Vic
S Bovismorbificans 4	M/12	Vic
S Bovismorbificans 7	F/63	Vic
S Derby	M/66	NSW
S Enteritidis	F/26	NSW
S Hadar	F/ns	Vic
S Hadar	F/66	Vic
S Muenchen	F/10	Vic
S Orion	F/23	NSW
s Singapore	F/55	Qld
S Typhimurium 9	F/73	Vic
S Virchow	F/81	NSW
S Waycross	M/70	Qld
Sh flexneri var Y	F/57	NT

Unusual Sites (10):			
TYPE	SEX/AGE	STATE	SITE
S Agona	F/<1	NSW	umbilical cord
S Anatum	M/82	Vic	pus
S Anatum	M/44	NT	testicular cyst
S Birkenhead	M/19	Vic	forearm wound
S Havana	M/35	NSW	unspecified wound*
S Havana	M/63	NSW	pulmonary odoema fluid*
S Oranienburg	F/29	SA	bile
S Saintpaul	M/1	Qld	ear (faecal isolate S Reading)
S Virchow	M/60	NSW	gall bladder
S Virchow	M/50	Qld	ankle wound

* hospital infection, part of outbreak
ns = not stated

Table 4. Outbreaks

TYPE	PLACE	NO.	DATE	NOTES
Sh sonnei biotype a	Darwin	19	Oct-Dec	continuing
S Heidelberg 2	Rockhampton	18	Oct-Dec	suspected chicken
V parahaemolyticus poisoning	Sydney (2)	13	Nov	seafood, two incidents
S Typhimurium 2	Tasmania	3	Nov	no details
S Havana	Sydney	20	Nov-Dec	hospital outbreak
S Bovismorbificans 21	Sydney	16	Dec-	continuing
S Heidelberg	Melbourne	6	Dec-	continuing
S Typhimurium RDNC	Melbourne	3	Dec-	suspected oysters

Table 5. Typhoid and Paratyphoid cases*

S Typhi: 8 cases			
VI-PHAGE TYPE	SEX/AGE	STATE	NOTES
38	F/43	Vic	returned from India
B1	M/32	Vic	returned from Phuket, Thailand
degraded	M/30	NSW	no details
degraded	F/5	NSW	no details
degraded	F/ns	NSW	mother of F/5
degraded	M/8	NSW	recent visit to Indonesia
degraded	F/68	Vic	submammary abscess swab
degraded	F/39	NSW	returned from Philippines

S Paratyphi A: 5 cases			
PHAGE TYPE	SEX/AGE	STATE	NOTES
1	M/32	Vic	no details
2	M/33	Vic	three years teaching in Cambodia
5	M/8	NSW	recent visit to Indonesia
5	F/23	NSW	no details
6	F/18	Vic	ex-Vietnam

S Paratyphi B: 4 cases			
PHAGE TYPE	SEX/AGE	STATE	NOTES
Beccles var 5	F/<1	NT	no details
Dundee	M/11	SA	no details
RDNC	M/24	WA	returned from Bali
RDNC	M/ns	NSW	no details

*ns = not stated

Shigella infections

There were 204 shigella reports received during this quarter. Of these, 11 were follow-up specimens, 6 were from migrants or refugees and 22 were notified from travellers returning from overseas leaving a total of 165 cases assumed to have been acquired in Australia.

However, seven of these 156 cases were of doubtful origin as patient notes on overseas travel did not accompany the reports. These were the one case of *Sh boydii* 8 and the six cases of *Sh sonnei* biotype g, most of whom were adults.

Sh flexneri 2a, *Sh flexneri* 6 and *Sh sonnei* biotype a accounted for 84% of the total cases of shigella acquired in Australia. The most common serotype was *Sh sonnei* biotype a with 62 cases, 28 of which were from the Northern Territory where there was an outbreak in Darwin beginning in mid-November.

Shigella infections acquired overseas include *Sh boydii* 2 and *Sh dysenteriae* 1 (India) *Sh flexneri* 1b, *Sh flexneri* 2a (India, South-east Asia, Vietnam), *Sh flexneri* 6, *Sh flexneri* var y (India), *Sh sonnei* biotype a (Timor, Vietnam, Greece, Fiji), and *Sh sonnei* biotype g (India, Bali, Central America).

Table 6: Shigella Infections Acquired in Australia

ORGANISM	ACT	NSW	Vic	Qld	SA	WA	Tas	NT	TOTAL
Sh boydii 8	-	1	-	-	-	-	-	-	1
Sh flexneri	-	-	-	2	-	-	-	-	2
Sh flexneri 1a	-	-	-	-	-	1	-	-	1
Sh flexneri 1b	-	1	-	-	-	-	-	-	1
Sh flexneri 2a	-	1	2	3	1	27	-	22	56
Sh flexneri 3a	-	2	2	-	-	-	-	-	4
Sh flexneri 3b	1	-	-	-	-	-	-	-	1
Sh flexneri 4a	-	-	-	-	-	-	-	2	2
Sh flexneri 4a mann -ve		-	-	-	-	-	-	1	1
Sh flexneri 6	-	-	-	-	3	4	-	14	21
Sh flexneri var Y	-	1	-	-	-	1	-	5	7
Sh sonnei biotype a	-	7	5	3	2	17	-	28	62
Sh sonnei biotype g	-	2	2	2	-	-	-	-	6
Total	1	15	11	10	6	50	-	72	165

Table 7. Mixed infections

ORGANISMS ISOLATED	SEX/AGE	STATE
S 4,5:1,v:-, *Sh dysenteriae* 1	F/6*	NSW
S Adelaide, S Oranienburg	F/<1	WA
S Anatum, S Mgulani	M/<1	Qld
S Anatum, S Wandsworth	M/<1	NT
S Bahrenfeld, S Oranienburg	F/<1	WA
S Berta, S Enteritidis	F/26	NSW
S Berta, S Potsdam	F/29	WA
S Hadar, *Sh sonnei* biotype g	F/31*	NT
S Hessarek, S Typhimurium 145	F/45	Vic
S Infantis, *Campylobacter* sp.	F/1	NSW
S Litchfield, *Campylobacter* sp., *Giardia* sp., *Y. enterocolitica* 0:3, Bio 4	M/1	SA
S London, S Weltevreden	M/<1*	WA
S Muenchen, S Java	M/1	WA
S Reading, S Saintpaul	M/1	Qld
S Sofia subsp 2, S Typhimurium 12a	F/7	SA
S Typhimurium 9, *C jejuni*	M/<1	Vic
S Typhimurium 26, *Y. enterocolitica*	F/6	Qld
Y enterocolitica, *Campylobacter* sp.	M/1	NSW

* acquired overseas (F/6 India, F/31 South-east Asia, M/<1 Vietnam)

Infections acquired overseas

(including migrants and refugees and excluding enteric fever):

ASIA:- unspecified: S 6,7:r:-, S Enteritidis, S Hadar, *Sh flexneri* 2a, *Sh sonnei* biotype g.

Indonesia: S 9,12:f,g:-, S Cholerae-suis, S Derby, S Emek, S Hadar (2), S Typhimurium RDNC, S Virchow.

Bali: S Berta (5), S Blockley, S Enteritidis (2), S Hadar (11), S Heidelberg, S Isangi, S Litchfield, S London, S Oslo, S Stanley, S Typhimurium RDNC (2), *Sh sonnei* biotype g (3).

Borneo: S Blockley.

Timor: *Sh sonnei* biotype a.

India: S Bareilly, S Derby, S Enteritidis, S Typhimurium 8, *Sh boydii* 8, *Sh dysenteriae* 1, *Sh flexneri* var y, *V parahaemolyticus*.

Malaysia: S Hadar, S Saintpaul, S Stanley, S Virchow.

Singapore: S Poona, S Tennessee, S Typhimurium untypable.

Thailand: S Blockley (3), S Hadar, S Java untypable, S Panama, S Saintpaul, S Stanley, S Virchow.

Hong Kong: S Arizonae 61:l,v:z35, S London var 15+, S Typhimurium 135.

Philippines: S Heidelberg.

Vietnam: S Anatum, S Chailey, S Lexington, S London and S Weltevreden (mixed), S Typhimurium 135, *Sh flexneri* 2a (2), *Sh flexneri* 4a, *Sh flexneri* 5a , *Sh sonnei* biotype a (5).

Kampuchea: S Derby, *Sh flexneri* 6.

AFRICA:- S Muenchen.

Egypt: S Infantis, S Senftenberg.

EUROPE:-

Great Britain: S Hadar.

Greece: *Sh sonnei* biotype a.

Cyprus: S Blockley.

PACIFIC:- Fiji: S Virchow, *Sh sonnei* biotype a.

New Caledonia: S Hadar.

Solomon Islands: S Bovismorbificans 2.

AMERICAS:-

Bermuda: S Mississippi

Central America: *Sh sonnei* biotype g.

USA: S Hadar.

Plus from unspecified countries: S Agona (3), S Anatum, S Berta (2), S Bovismorbificans 7, S Cerro, S Derby, S Enteritidis (2), S Hadar (2), S Montevideo, S Potsdam and S Berta (mixed), S Rissen, S Sofia ssp 2, S Typhimurium phage types 120, 12a, 135, 145, RDNC and untypable, *Sh flexneri* 1b, 2a and 6, *Sh sonnei* biotypes a and g (4).

Top Ten Salmonella Serovars

Of the 1099 Australian acquired cases of salmonella infection, 760 (69%) were isolates from the top ten salmonella serovars (Table 8). Their position in the previous quarter (Q3/'90) is also given where applicable.

In this quarter four of the top ten salmonellas were associated with outbreaks. In November there was an outbreak of **S Havana** in a Sydney hospital, there were two minor outbreaks involving **S Typhimurium** in Tasmania and Victoria, in Rockhampton there was an outbreak of **S Heidelberg** phage type **2** in October and in December an outbreak of **S Bovismorbificans 21** began in Sydney.

S Typhimurium with 382 cases from 32 phage types, was the most common salmonella serovar and accounted for 35 percent of the total Australian acquired cases of salmonella. **S Typhimurium 9** was the most common phage type with 75 cases, mainly from Victoria and New South Wales. The **top five phage types** accounted for 51% of Australian acquired cases of S Typhimurium (Table 9).

Table 4: Top Ten Salmonella Serovars

SEROVAR	POS'N Q3/'90	NO. OF CASES	% OF TOP TEN	ORIGIN/NO. OF CASES
S Typhimurium*	1	382	50.3	NSW 127, Vic 95
S Bovismorbificans*	-	60	7.9	NSW 30, Vic 12
S Heidelberg*	-	51	6.7	Qld 33, Vic 10, NSW 8
S Birkenhead	-	50	6.6	Qld 26, NSW 10
S Vichow	3	50	6.6	Qld 34
S Saintpaul	7	39	5.1	Qld 24, NT 7
S Havana*	10	38	5.0	NSW 24
S Chester	8	32	4.2	Qld 17
S Muenchen	4	32	4.2	Qld 11
S Anatum	5	26	3.4	Qld 11

In: S Bovismorbificans, S Heidelberg, S Birkenhead
Out: S Cerro, S Enteritidis, S Infantis
* associated with outbreaks

Table 9: Top Five Phage Types of S Typhimurium

PHAGE TYPE	NO. OF CASES	% OF TOP FIVE	ORIGIN/NO. OF CASES
9	75	39	Vic 32, NSW 32
135	58	30	NSW 16, Qld 13, Vic 11
145	25	13	Vic 11
12a	18	9	Qld 8, NSW 5
101	18	9	Qld 8, NSW 6

NSSS Update

In the first four months of 1991 there were at least 15 outbreaks of salmonellosis and one of shigellosis. Some were large and spread over wide areas of metropolitan Melbourne and Sydney; others were smaller and more confined.

The larger outbreaks, including the number of cases notified to the NSSS by the end of April, were:

S Typhimurium 135 in the Melbourne metropolitan area (96 cases) late January to late February

S Typhimurium RDNC in Melbourne (55 cases) mid- to late January

S Bovismorbificans 21 in Sydney (59 cases) late December and continuing

S Bovismorbificans 23 in Sydney (53 cases) early January and continuing

S Heidelberg in New South Wales (53 cases) late December to late February

S Heidelberg in Victoria (128 cases), mid-December to late February

S Heidelberg phage type **2** in Rockhampton (20 cases) early November

***Sh sonnei* biotype a** in Darwin (39 cases) mid-February and continuing.

Smaller outbreaks or incidents were:

S Typhimurium 44 in Sydney (13 cases) mid-March

S Typhimurium RDNC in Tasmania (24 cases) late January to mid-February

S Hadar in Tasmania (11 cases) late November to mid-February

S Give in Tasmania (19 cases) mid-January to mid-February

S Anatum in Adelaide (30 cases) late February

S Tennessee in Adelaide (4 cases, all Vietnamese) late February

S Brisbane in Alice Springs (4 cases, children 2 years and under) late February.

HOSPITAL-ACQUIRED INFECTIONS: A CROSS-SECTIONAL PREVALENCE SURVEY

(Reproduced with acknowledgment from Monthly Infectious Diseases Report, Royal Alexandra Hospital for Children, No. 16, February 1991, Editor D Isaacs)

In August 1990 (*Monthly Infectious Diseases Report* No. 10, reproduced in *CDI* **15**:36) we reported the initial results of a cross-sectional survey of hospital-acquired infections (HAI). The aim of this study was to survey all the children in hospital on a single day to determine how many of them had hospital-acquired infections and the risk factors for those infections. This sort of study cannot give information on the incidence of HAI, which would require following a cohort of children prospectively from admission to discharge and also at home to see if they developed HAI. Such longitudinal studies are time-consuming and very difficult to perform because of the problem of adequate follow-up at home. Cross-sectional studies, on the other hand, examine prevalence of HAI at a given point in time. Since HAI often lengthens hospital stay, a cross-sectional prevalence study tends to give a higher proportion of children with HAI than a longitudinal incidence study.

The long-term aim of such studies is to examine whether preventable HAI are occurring, and to evaluate any interventions to try to reduce HAI[1]. It is important to repeat prevalence surveys regularly, to see how the prevalence of HAI varies, both by chance and in association with factors like the season and the number of children in hospital with community-acquired infections.

We repeated the prevalence survey in February 1991. The team who carried out the survey on the wards was Dianne Heywood-Barnes, David Isaacs, David Macintosh, Chris Morgan, Kerry Sherred, Ruth Skelton and Anne Weedon. The information collected was put on computer by Maraia Bale and analysed by Mark Hanlon.

The same definitions were used:

Hospital-acquired infection is an infection acquired during the current hospitalisation or as a result of a past hospital-based procedure, but excluding community-acquired infections. Thus infected central venous cannulas and infected CSF shunts are included as HAI.

Community-acquired infection (CAI) is one present at admission or developing within the incubation period for the organism.

Number of inpatients on the day of study: 174.

Number with hospital-acquired infection: 16 (9.2%).

Number with community-acquired infection: 31 (17.8%).

Various risk factors known or suspected to be associated with HAI were examined. The results of these examinations are presented below.

The greatest proportion of HAI was in Oncology Department patients, 9 out of 18 of whom had HAI (4 central venous catheter infections, the rest had opportunistic infections related to immunosuppression).

DURATION OF HOSPITAL STAY AND PREVALENCE OF HAI			
HOSPITAL STAY (DAYS)	NO. WITH HAI (%)	TOTAL	
0-7	2 (2%)	118	Comparing 0-7 with 8+ days
8-14	4 (15%)	26	$\chi^2 = 24.7$, 1 df. $P < 0.001$
15+	10 (33%)	30	

AGE AND PREVALENCE OF HAI			
AGE (MONTHS)	NO. WITH HAI (%)	TOTAL	
<1	0	18	
1-6	4 (15%)	27	
7-12	2 (14%)	12	
13-24	1 (5%)	19	
25-36	2 (18%)	11	Comparing ≤
37-48	2 (29%)	7	1 year to over
49-60	1 (9%)	11	1 year $\chi^2 = 0.6$, 1df.
60+	4 (6%)	67	Not significant

SEX AND PREVALENCE OF HAI			
SEX	HAI (%)	TOTAL	
Male	6 (6%)	97	$\chi^2 = 2.38$ 1 df.
Female	10 (13%)	77	not significant

INTENSIVE CARE AND PREVALENCE OF HAI			
INTENSIVE CARE	HAI (%)	TOTAL	
Yes	1 (4%)	25	Fisher's exact test:
No	15 (10%)	149	not significant

HOSPITAL SERVICE AND PREVALENCE OF HAI			
SERVICE	HAI (%)	TOTAL	
All medical, excluding neonates	11 (17%)	66	Comparing all medical to all surgical patients (excluding neonates) $\chi^2 = 6.77$, 1 df. $P < 0.01$
All surgical, excluding neonates	4 (4%)	92	
Neonates	1 (6%)	16	Comparing medical to surgical patients (including neonates) $\chi^2 = 4.7$, 1 df. $P < 0.05$

RECENT SURGERY (THIS ADMISSION) AND PREVALENCE OF HAI			
SURGERY	HAI (%)	TOTAL	
Yes	8 (11%)	70	$\chi^2 = 0.7$, 1 df.
No	8 (8%)	104	P >0.5

IMMUNOSUPPRESSION AND PREVALENCE OF HAI			
IMMUNOSUPPRESSED	HAI (%)	TOTAL	
Yes	9 (45%)	20	Fisher's exact test
No	7 (5%)	154	P = 0.00005

ENDOTRACHEAL INTUBATION AND PREVALENCE OF HAI
Only 6 children were intubated at the time of the survey.
None developed HAI.

INTRAVASCULAR CANNULA AND PREVALENCE OF HAI			
CANNULA	HAI (%)	TOTAL	
Yes	13 (20%)	64	$\chi^2 = 15.0$, 1 df.
No	3 (2%)	110	P < 0.001

NATURE OF HOSPITAL-ACQUIRED INFECTION	
Central venous catheter infection	4
Wound infection	4
Septicaemia secondary to immunosuppression	2
Candida oesophagitis	1
CMV pneumonitis	1
Gastroenteritis	1
Otitis media	1
Shunt infection	1
Urinary tract infection	1
TOTAL	16

ORGANISMS CAUSING HAI (some episodes were polymicrobial)	
Staphylococcus epidermidis	5
Candida	3
E. coli	3
MRSA	2
Gram negatives	4
Miscellaneous	5
TOTAL	22

WAS THE HAI CONSIDERED PREVENTABLE?	
Not preventable	6
Possibly preventable	6
Definitely preventable	4

This last category is difficult to evaluate. In general wound infections, gastroenteritis and respiratory virus infections are deemed definitely preventable, central venous catheter infections in immuno-compromised children and shunt infections are possibly preventable, while UTI (unless secondary to urinary catheter), otitis media and opportunist infections secondary to immunosuppression are not preventable.

The risk factors for HAI were similar to the previous survey. Notable differences were that on this occasion, immunosuppression was a major risk factor for HAI whereas intensive care was not. Duration of hospital stay was again a clear risk factor. The oncology patients had a high prevalence of HAI, but the Oncology Department was extremely busy and had a large number of heavily immunosuppressed children with opportunist infections.

Overall the prevalence of HAI was somewhat lower than on the first survey, though not significantly so. However, there were far fewer children with community-acquired infections on the wards, which implies less exposure to infection. It is important that we continue to perform regular surveys and do not over-interpret from one or two fluctuations in prevalence which may be due to chance or confounding variables.

REFERENCE

1. French GL, Cheng AFB, Wong SL, Donnan S. Repeated prevalence surveys for monitoring effectiveness of hospital infection control. *Lancet* 1989;ii:1021-3.

AIR TRAVEL-ASSOCIATED GASTROENTERITIS OUTBREAK, AUGUST 1991.

(Rosemary Lester, Tony Stewart, John Carnie, Sally Ng and Roscoe Taylor, Infectious Diseases Unit, Health Department Victoria)

Extent of the Outbreak

As at 20 August 1991, 3053 individuals had been notified as being affected. This figure incorporates notifications from other States, which were approximately:

SA:	300
NSW :	400
Tasmania :	380
ACT :	40
NT:	40

Individual airlines have also been notified of a large number of cases, although some of these will be duplications of the notifications to Health Department Victoria (HDV). Therefore, the lower estimate of the total at present is around 3,050 plus any notifications to airlines not already documented by HDV.

Further information on the incidence of gastroenteritis in the community at the time of this outbreak is being sought. This information is needed to verify that the attack rates among airline travellers were significantly higher than that of the general community.

Four surveys are presently being organised:

- a survey of sentinel general practices;
- a telephone survey of the general community conducted by a survey company;
- a survey of several AFL teams to compare those which flew during the weekend of 10-11 August to those which did not travel;
- a survey of several flights from the major airlines affected. All the passengers will be questioned, so that attack rates can be calculated.

The vast majority of notified cases have been airline travellers. Other groups have been reported, such as conference and school groups who have not travelled, and these are being followed up more fully.

Other States, the Commonwealth Department of Health, Housing and Community Services, and the Australian National University are co-operating with the investigations. Health Department Victoria has been designated as the lead agency in the investigations.

Clinical Features

Fifty-three per cent of the cases had an incubation period of less than 48 hours, and 84% less than 72 hours. Around 5% had a short incubation period of less than 24 hours.

Average duration (of a small sample) was 38 hours. Unfortunately this information was not collected for many cases.

Most cases described sudden onset of severe vomiting and diarrhoea. Fever and generalised aches and pains were less commonly reported.

Cause of the outbreak

The vast majority of notified cases have travelled ex-Melbourne. All the major domestic airline companies and a number of international flights departing ex-Melbourne are involved. It was established that catering arrangements were separate, apart from a common

(Melbourne) supplier of orange juice. The two large non-travelling groups which were reported (one in South Melbourne, one in Geelong) as being affected were also supplied by the same company. Surveys of sporting and school groups travelling together have revealed attack rates of illness of up to 100% among orange juice drinkers, and 0% among non-orange juice drinkers:

- in a sporting group of 21 travelling together, of the 10 who drank orange juice, 8 became ill, and of the 11 who did not drink orange juice, only 1 became ill (relative risk: 8.8; odds ratio: 40)
- in at least 50 pairs of people travelling together, the one person who drank both travellers' orange juice became ill and the person who did not drink the orange juice did not become ill
- illness was recorded in at least 5 non-travelling children for whom travelling parents had brought the orange juice home.

Many reports are indicative of a dose - response relationship: those who drank more than one cup of the juice have been more severely affected than those who did not complete their serve. Hence the evidence implicates the orange juice as the vehicle of transmission.

Approximately 30 faecal samples and 40 blood specimens were collected from a football team and two basketball teams. Studies on the faecal samples have shown not bacterial pathogens, but the presence of a viral agent known as a Norwalk - like agent, or SRSV (small round structured virus). This is in accord with the clinical picture, which is typical of a viral type of illness. Five of the 10 faecal samples processed so far have been found to be positive for the Norwalk - like agent.

The epidemic curve (Figure) also provides evidence of the curtailing of the epidemic upon the withdrawal of the implicated product.

Bacteriological testing of several batches of the orange juice and other foods collected from two of the airline companies revealed no significant bacterial pathogens. Chemical analysis of the orange juice revealed no significant abnormalities. Virus isolation will be attempted from the juice, but we expect that this will be a difficult task - this type of virus has not been isolated from food or water before, to our knowledge.

Microscopy of samples containing small particles of foreign matter revealed organic matter consistent with immature orange seeds and small masses of mould filaments, which would have been in the juice prior to packaging and are probably incidental to this epidemic.

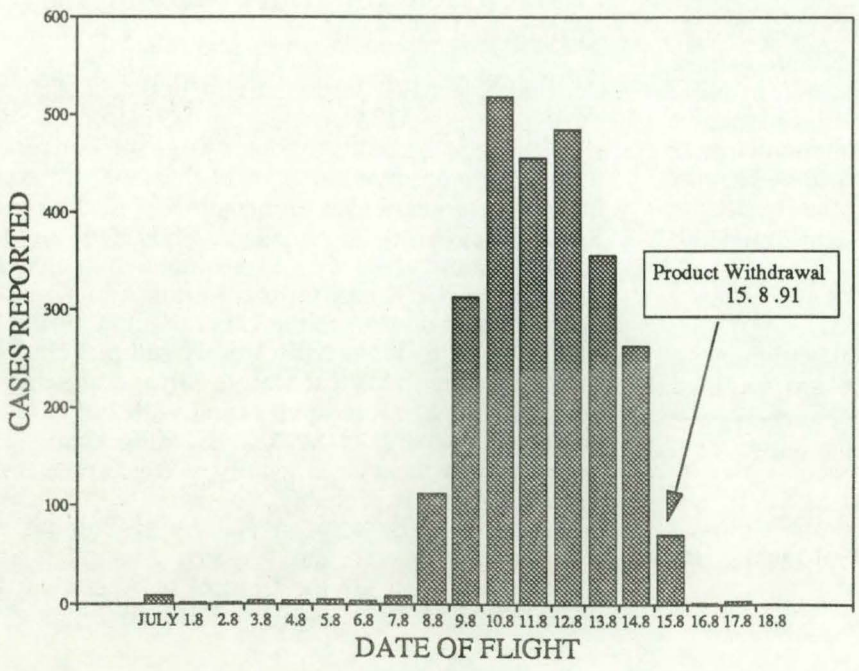
Control Activities

The airlines were contacted during the course of Thursday 15 August when the evidence indicated that the orange juice was responsible. They agreed immediately to withdraw the juice. The company responsible was informed, and agreed to a private recall of their major suppliers and cessation of production of the orange juice. All produce is to be collected by or returned to the company and disposed of in landfill, under the supervision of environmental health officials.

A team from Health Department Victoria, the relevant city council and the Microbiological Diagnostic Unit's food microbiologist visited the factory and identified several problem areas where potential contamination could have occurred. Suspect plumbing connections are being further followed up. A list of requirements to be fulfilled before production re-starts, and less urgent matters to be attended to over a longer time period, is being prepared at the moment.

Staff at the factory have denied any recent illness, and records concur with this. They have agreed to provide blood and faecal specimens to identify any persons actively excreting the virus, or with evidence of a recent infection. This is being conducted in a strictly confidential manner.

Figure. Gastroenteritis cases (with known date of flight) reported by 20 August.



AUSTRALIAN ENCEPHALITIS IN WESTERN AUSTRALIA AND NORTHERN TERRITORY, 1991

(J S Mackenzie[1], K Broom[1], D W Smith[2], J Burrow[3] and P Whelan[4])

Four cases of Australian encephalitis, two from the Kimberley region of Western Australia and two from the Northern Territory, have been diagnosed in 1991. Three of the cases were caused by Murray Valley encephalitis (MVE) virus, and one by Kunjin virus.

Case Histories

Case 1.

A 16 month old Aboriginal girl from Balgo Community (235 km south of Halls Creek) was admitted to Derby Regional Hospital on 9 April with fever, lethargy, diarrhoea, and a history of upper respiratory tract infections. She was transferred to Princess Margaret Hospital, Perth, on 11 April and presented with repeated seizures and respiratory failure requiring ventilatory support. Despite treatment with antibiotics and anticonvulsants, she developed a flaccid paralysis with evidence of brainstem involvement. Both an electroencephalogram and CT scan showed a diffuse encephalopathy. She died 5 days after admission to Princess Margaret Hospital. Sera collected 4 and 2 days prior to death had MVE IgG of 1:80 by haemagglutination-inhibition (HI), and the later serum sample was MVE-IgM positive by immunofluorescence (IF). These results were confirmed by ELISA.

Case 2.

A 74 year old New Zealand woman on holiday near Lake Deane in the Berry Springs area, 55 km south of Darwin, presented to Royal Darwin Hospital on 5 April with a 2 day history of confusion and high fever. She developed brainstem signs and hypopnoea on 8 April, requiring ventilation. This was followed by a descending flaccid paralysis of all limbs over 4 days, despite acyclovir treatment. She was transferred to Royal Adelaide Hospital for plasmaphoresis. She remained paralysed and on ventilation, and returned to New Zealand in that condition one month after presentation. Sera collected on admission and one month later showed a rise in MVE IgG from 1:10 to 1:80 by HI, and a positive MVE IgM on the first specimen by IF. A monoclonal antibody-blocking ELISA confirmed the antibodies were specific to MVE.

Case 3.

A 9 month old Aboriginal boy from an outstation in the Northern Territory, approximately 700 km south of Darwin and 180 km north-east of Balgo Community, presented to Royal Darwin Hospital on 4 May with a week of fever, diarrhoea, vomiting and lethargy. His conscious state decreased rapidly over 24 hours after admission associated with several seizures. Over the next few days he developed flaccid paralysis of all limbs, brainstem signs and required ventilation. Acyclovir was given for possible herpes simplex encephalitis. Five weeks after admission his brainstem functions and respiration had recovered but the limb paralysis persisted. On follow-up 12 weeks after admission, there was partial recovery of upper and lower limb movement. Sera collected on admission and 5 weeks later showed a rise of MVE IgG from 1:40 to 1:640, and a positive MVE IgM on the first specimen by IF. Antibodies specific to MVE were confirmed by a monoclonal antibody-blocking ELISA.

Case 4.

A 33-year-old male Caucasian living 15 km from Kununurra in the Kimberley region of Western Australia presented to the District Medical Officer on 6 May, with weakness, lethargy, headache and fever. Treatment with analgesics did not relieve the symptoms but he was able to continue working. The symptoms were unchanged on review 7 days later, but at 4 weeks after presentation he had only mild lethargy remaining. Sera collected at 7 and 17 days after presentation showed a rise in MVE IgG from 1:20 to 1:160 with positive IgM by IF to MVE and Kunjin. A monoclonal antibody-blocking ELISA and tissue culture neutralisation tests have confirmed the antibodies to be specific to Kunjin virus.

Field epidemiological observations

(a) Kimberley region, Western Australia.

Considerable MVE virus activity has been detected during the period March to May 1991, from sentinel chicken flock seroconversions. Thus seroconversion to MVE was observed in 14 of 19 chickens at Broome, in 7 of 11 chickens at Kununurra, in 5 of 12 chickens at Kalumburu, in 4 of 10 chickens at Wyndham, and in 3 of 11 chickens at Derby. In addition, two chickens seroconverted to Kunjin virus at Kununurra. There has been a spread of virus to the Pilbara region during the period April to June, with 3 of 11 sentinel chickens seroconverting to MVE at Marble Bar, and one chicken seroconverting to Kunjin virus and with 2 of 17 chickens seroconverting to MVE at Harding Dam. This represents the most virus activity in Western Australia since 1981, and is probably associated with a heavier wet season. The seroconversions at Broome and Kununurra triggered the Western Australian State Contingency Plan for the Control of Australian Encephalitis, and as a result, adulticide fogging for

1. Department of Microbiology, The University of Western Australia
2. State Health Laboratory Service, Perth
3. Royal Darwin Hospital
4. Medical Entomology Branch, Department of Health and Community Sevices, Darwin

mosquito control was carried out at Broome on 17 April and at Kununurra on 16, 17 and 19 May. Several MVE virus isolates have been obtained from mosquitoes trapped in Broome in April.

In response to the case of Australian encephalitis at Balgo in early April, an investigation was carried out on 18 April (approximately 2 1/2 weeks after probable exposure) in which Encephalitis Virus Surveillance (EVS)-CO_2 light traps were set at 10 locations. A total of 535 mosquitoes were recovered of which 93% were *Culex annulirostris*, the major vector of MVE and Kunjin viruses, but the mosquitoes have yet to be processed for virus isolation. The only significant mosquito catch was from the sewage lagoons close to the community. Additional evidence of virus activity was obtained from an ongoing surveillance study at Billiluna Community, 80 km north of Balgo, where 6 children have recently seroconverted to MVE. Balgo and Billiluna are situated in an arid area on the edge of the Great Sandy Desert, and it is assumed that virus may be introduced into this area by viraemic waterbirds during periods of flooding or above average rainfall. Over 10,000 mosquitoes were caught during two nights trapping at Kununurra in early April, approximately 2 weeks prior to the estimated exposure in case 4. Trap numbers varied between 50 and 3000, depending on trap site. Although *Cx annulirostris* mosquitoes were the predominant species trapped, identification and processing for virus isolation have yet to be completed.

(b) Northern Territory

A near record wet season occurred in 1990-91 in the Darwin region. Following case 2, an epidemiological investigation was undertaken in the Lake Deane vicinity at Berry Springs, 55 km south-east of Darwin approximately 5 weeks after the probable exposure date. A total of 6 EVS-CO_2 traps were set at or near Lake Deane on 2 May, including traps at Barden's Lakes tourist park which was assumed to be the most significant source of mosquitoes. A total of 2398 mosquitoes were trapped, and *Cx annulirostris* was found to be the predominant species (26%) with 0.5% *Aedes normanensis*. The largest number of *Cx annulirostris* were obtained near the patient's residence. It was considered too late to undertake adulticide fogging for mosquito control. The mosquitoes have been processed but no viruses were isolated.

No epidemiological investigation was carried out at the Northern Territory outstation but mosquito trapping at Lajamanu, approximately 150 km away, at about the same time as the probable transmission, indicated a preponderance of *Cx annulirostris* with no flood water *Aedes* species, and this picture would have probably been similar at the outstation at that time of year. However, like Balgo and Billiluna, the outstation is located in an arid region, and would probably require exceptional environmental conditions for the introduction and dissemination of virus.

The significantly increased incidence of virus activity in northern Australia this year is of concern should increased rainfall or flooding occur in central Australia as this may facilitate eventual virus movement to south-eastern areas of Australia.

Acknowledgement

We would like to thank the following people for their help:

Mr M Lindsay and Miss M D'Ercole from the University of Western Australia, Mr A Wright from the Health Department of Western Australia, Dr M Patel and staff of the Communicable Diseases Centre, Department of Health and Community Services, Darwin, and J Standen, the Health Surveyor from Katherine.

KUNJIN VIRUS - HUMAN INFECTIONS REPORTED IN VICTORIA, 1991.

(Rosemary Lester, Public Health Officer, Environmental Health Section, Health Department Victoria)

Kunjin virus is a flavivirus which is related to the Murray Valley encephalitis (MVE) virus[1]. It is transmitted by mosquitoes and has been shown to infect wild and domestic animals over much of mainland Australia[2]. Inapparent and subclinical infections predominate with most arboviruses; infection with clinical illness is frequently the exception rather than the rule. It appears that the majority of human infections with Kunjin virus follow this pattern[2,3].

The earliest reports of symptomatic human infections with Kunjin virus were published by Allan, Doherty and Whitehead[4]. They described two laboratory workers whose illnesses were proven to be due to Kunjin virus by serology and/or virus isolation. The first case occurred in a 32-year-old female virologist who experienced mild fever, lymphadenopathy and a rash which, at various times, was macular, papular and vesicular. Kunjin virus was isolated from the serum, and a significant rise in antibody titres to several flaviviruses was observed, with a positive neutralisation test for Kunjin and MVE viruses. The second case was a 23-year-old male laboratory assistant, who had a two day febrile illness with nausea, anorexia, lethargy and tremor. Haemagglutination-inhibition (HI) tests showed a rise in serum antibody titres to several flaviviruses, with titres much higher to Kunjin than to the other viruses. Complement fixation titres were also positive to Kunjin virus at 1:32 and to MVE virus at 1:8.

A recent report of a case of Kunjin virus encephalomyelitis was described in Victoria[3]. A 40-year-old male fruit picker suffered a prolonged encephalomyelitis requiring ventilation and was left with a severe motor

disability. HI titres in this patient showed a rise to both MVE virus and Kunjin virus, however, specific IgM was detected only against Kunjin virus.

Serological cross-reactivity, as demonstrated by these cases, is well known in arbovirus infection. This property is exploited by serologists who use one agent to screen for infection caused by several viruses in the group.

Kunjin virus activity was noted in the Mildura area of Victoria by seroconversion in the sentinel chicken flock in January and early February 1991. The virus was also isolated from mosquitoes at Wentworth (south-western NSW) in November and December 1990 by the Westmead Entomology Unit.

In early February 1991, Health Department Victoria received notifications of two human infections which proved to be due to Kunjin virus.

The first was a 51-year-old male shopkeeper from Wentworth whose symptoms first began in November 1990. He experienced extreme lethargy with generalised aches and pains, particularly in the legs. There was no rash and no particular headache or other neurological symptoms. No abnormality was found on examination. Serology performed at the State Health Laboratory, Brisbane, showed negative IgM but positive IgG to Ross River virus, and positive IgM to Kunjin virus. By the end of February 1991, his symptoms had begun to abate.

The second notification was a 48-year-old male fruit grower whose symptoms began in the third week of January 1991. He complained of lethargy, generalised aches and pains and intermittent blurring of vision. There was no significant headache or rash. Examination was unremarkable. Serology revealed IgM positivity to a range of flaviviruses. Since there had been Kunjin virus activity in the area, it was thought that Kunjin virus was the most likely agent of the disease in this case.

In summary, it appears that Kunjin virus may cause a range of human infections from the sub-clinical through to a severe encephalomyelitis. Awareness of this mosquito-borne virus and better methods of recognition will obviously help to delineate further research into the incidence of both inapparent and clinically significant illness.

REFERENCES

1. Doherty, RL. Arboviruses in search of disease. *Med J Aust* 1987;**146**:561-2.
2. Downs, WC. Arboviruses. In: Evans , AS (Ed) *Viral Diseases of Humans: Epidemiology and Control. New* York. 1989.
3. Muller, D, McDonald, M, Stallman, N and King, N. Kunjin virus encephalomyelitis. *Med J Aust* 1986;**144**:41-2.
4. Allan, BC, Doherty, RL and Whitehead, RH. Laboratory infections with arboviruses including reports of two infections with Kunjin virus. *Med J Aust* 1966;**2**:844-7.

CDI Editorial Comment

This year, there has been one other case of Australian encephalitis reported apart from those described in the preceeding two articles. The patient was a 2-year-old girl from Wiepa who showed elevated IgM to Murray Valley encephalitis (MVE) virus (*CDI* **15**:217).

In addition, IgM to MVE has been demonstrated in 3 patients for whom encephalitis was not reported: a 35-year-old male reported from Cairns who had general malaise/mild fever and two patients for whom no clinical information was supplied: a 7-year-old female reported from Cairns and a 71-year-old female reported from Brisbane.

There have been 8 further reports of Kunjin virus infection. Five of the patients suffered joint disease: a 19-year-old male and a 54-year-old female from Rockhampton, and a 32-year-old male, a 35-year-old male and an 81-year-old male from Townsville. There was no clinical information reported for the remaining 3 patients: a 44-year-old female from Toowoomba, a 33-year-old male from Brisbane and a male (age unknown) from Townsville.

HUMAN CUTANEOUS ANTHRAX - A CASE REPORT

(Bin Jalaludin, Research Officer; Elizabeth Sullivan, Acting Director, Western Sector Public Health Unit, Department of Health New South Wales, Reproduced with acknowledgment from New South Wales Public Health Bulletin, June 1991)

This report describes the circumstances and clinical findings of a case of human cutaneous anthrax, and discusses the public health implications.

Anthrax is a zoonosis that was endemic in Europe before the introduction of an effective animal vaccine. Koch, in the late 1800s, demonstrated the bacterial aetiology of anthrax, the first disease to which a bacterial cause was ascribed[1].

Human anthrax is still a problem in many regions of the world[2,3,4,5], although in developed countries it is uncommon. In NSW there have been only two reported cases of human anthrax since 1982.

Anthrax is caused by *Bacillus anthracis*, a spore-forming, gram-positive organism. The spores are extremely resistant to adverse environmental conditions[6], surviving high temperatures and drying, and can remain dormant but potentially infective for more than 20 years[7]. Pathophysiologic effects in infected animals

or humans are due to toxins produced by the organism[8].

The main reservoirs of infection are domestic herbivores such as sheep, cattle and goats. Human infection comes from direct contact with contaminated skins or carcasses, inhalation of spores from contaminated animal products such as wool fibres or bone meal, or from eating infected meat[7].

The index case, a 35-year-old male, initially presented to a general practitioner with a number of vesicular lesions on his hands which the patient himself suspected to be anthrax. The patient was otherwise well and was given oral penicillin. In the following 24 hours, the man became systemically unwell and was admitted to a metropolitan hospital for intravenous penicillin therapy.

The vesicular lesions developed black necrotic lesions typical of cutaneous anthrax. *B. anthracis* was cultured from the swabs taken by the GP. The organism was not found in the patient's blood or in the lesions.

Three days before going to the GP, the patient, with two others, had been slaughtering sheep on a property in western NSW. There was no evidence of anthrax on that property, although it was known to be present in neighbouring properties.

The carcasses were to be used as feed for animals in a Sydney wildlife park. Eight other staff members of the park had direct contact with the carcasses.

Anthrax is common among stock in NSW, but human anthrax is uncommon. It is potentially fatal, especially if accompanied by septicaemia and severe toxaemia. Cutaneous anthrax comprises 90-95 per cent of all human anthrax, and has a case fatality rate of 10-20 per cent if untreated[9].

For public health, it is important that all human contacts be treated appropriately, and that contaminated materials be disposed of in a manner that eliminates further human or animal exposure.

All definite contacts, and many others at the wildlife park who were not at risk, were treated with oral penicillin for at least five days. The treatment of non-contacts was unnecessary but deemed justifiable in view of the high level of anxiety, and the relatively low cost of treatment.

It is recommended that contaminated materials be incinerated or disinfected, and that infected carcasses be covered with anhydrous calcium oxide (quicklime) during deep burial[9]. Some of the potentially contaminated materials were burnt, while others were disinfected with 5 per cent formalin solution.

The contaminated carcasses were buried, without any quicklime, at a local waste depot before involvement of the Public Health Unit. Though not the most appropriate site for disposal, it was the most accessible at the time. Fortunately, the buried carcasses are unlikely to be disturbed, since as normally is the case, the waste depot would be designated 'unhealthy building land' under the Public Health Act.

There was the potential for many more cases of human anthrax and it was fortunate that only one case was confirmed. In this case the diagnosis was confirmed by bacterial culture. If microbiological confirmation is not possible, or in epidemiological investigations, serological diagnosis is both sensitive and specific[4,5].

Eradication of human anthrax depends primarily on the ability to control it in domestic animals by effective surveillance and vaccination programs. Vaccination for humans is not available in Australia, so preventive measures should be emphasised to high-risk groups.

REFERENCES

1. Reidman S R. *The Story of Vaccination.* Bailey Brothers and Swinfen, United Kingdom. 1974.
2. Roberts C J, Chambers PG. An outbreak of anthrax in the Mondoro tribal trust lands. *Cent Af J Med* 1975;**21**:73-76.
3. Heyworth B, Ropp M E, Voos U G, et al. Anthrax in the Gambia: an epidemiological study. *B Med J* 1975;**4**:79-82.
4. Sirisanthana T, Nelson K E, Ezzell J W, et al. Serological studies of patients with cutaneous and oral-oropharyngeal anthrax from Northern Thailand. *Am J Trop Med Hyg* 1988;**39**:475-581.
5. Harrison L H, Ezzell J W, Abshire T G, et al. Evaluation of serologic tests for diagnosis of anthrax after an outbreak of cutaneous anthrax in Paraguay. *J Infect Dis* 1989;**160**:706-710.
6. Ditz W, Kohout-Dutz E. Anthrax. *Int J Derm* 1981;**20**:203-206.
7. Yorston D, Foster A. Cutaneous anthrax leading to corneal scarring from cicatricial ectropion. *B J Opthal.* 1989;**73**:809-811.
8. Dogaray M, Bakir M, Dokmetas I. A case of cutaneous anthrax with toxaemic shock. *B J Derm.* 1987;**117**:659-662.
9. Benenson A S (Ed). *Control of Communicable Diseases in Man,* 15th Edition. American Public Health Association, Washington. 1990.

CDI Editorial Comment

The 2 previous New South Wales cases of anthrax mentioned above are the only two cases notified from anywhere in Australia since 1982; one occurred in 1985 and the other occurred in 1987.

It is thought that the source of *Bacillus anthracis* infection is confined to Victoria and New South Wales. High risk groups are knackery workers, veterinarians performing autopsies and workers in tanneries and fertiliser and wool carpet factories.

Further details of the microbiological investigation of the case described above have been provided by Dr David Mitchell, Institute of Clinical Pathology and Medical Research, Westmead: thick gram-positive rods

were seen on the gram stain from the ulcerated pustular lesion on the back of the patient's hand, and were subsequently isolated on culture. The organism was identified as *B. anthracis* on the basis of characteristic colonial and microscopic appearance, negative haemolysis on horse blood agar, negative motility and citrate utilisation, penicillin sensitivity and typical reactions on AP15OCHB and AP12OE[1]. The lesion responded quickly to the penicillin therapy and swabs taken after 5 days were negative.

REFERENCE

1. Logan NA, Carman JA, Melling J and Berkley RC. Identification of *Bacillus anthracis* by API tests. *J Med Microbiol*. 1985;20:75-85.

OVERSEAS BRIEFS

In the last two weeks, the following information regarding cholera cases and recently infected areas has been supplied by the World Health Organization.

Cholera in Africa Update

The latest outbreak of cholera in the 30-year-old El Tor pandemic of the disease is sweeping through Africa and killing people at a much higher rate than seen during the peak of this year's outbreak in Peru, Ecuador and other Latin American countries. Death rates have ranged from 6% to 10% in some countries, but have been as high as 30% in some areas.

The total number of cases reported from Africa for the first 7 months of 1991 (over 45,000) exceeds the number of cases reported from Africa during all of 1990. Although the number of cases reported from Africa is far lower than the number of cases reported from the Americas (wer 250,000), the number of deaths reported from the Americas (2618) is far lower than from Africa (3488 by mid-July).

Cholera occurs in a seasonal pattern, and is often linked with rainy seasons. The high number of cases and deaths now being reported from Africa can be linked with the rainy season in central and western Africa.

Burkino Faso has recently reported its first cholera cases. The Ministry of Health reported 200 cases with 17 deaths up to 31 July. All cases occurred in Boulgou province.

Sao Tome and Principe has also reported its first cases. The Ministry of Health reported that 3 cases with 1 death occurred in July in Lemba district.

Revised figures for **Niger** have been provided. There were 2076 cases and 251 deaths from May to 21 July.

Nigeria continues to report a large number of cholera cases. From January to 6 August, there was a total of 23,463 cases and 3116 deaths. Bauchi, Benue, Borno, Gongola, Kwara, Ondo, Oyo and Sokoto States have joined Kano and Kaduna States as cholera-infected areas.

Chad has reported a further 1381 cases (205 deaths) for the period 16 to 18 July, and 426 cases (52 deaths) for the period 27 July to 4 August.

Cholera in the Americas Update

The epidemic of cholera which emerged in Peru in January 1991 and spread to several other countries in South America has now stabilised due to seasonal factors and the intervention of health authorities. The Latin American epidemic could, however, worsen later this year, when the winter in the Southern Hemisphere ends, and conditions again become more favourable for the spread of the disease.

Further areas within **Mexico** have been declared infected: Chiapas, Puebla and Veracruz States. There were 192 cases and 2 deaths from 23 to 27 July.

Colombia has reported 777 cases and 21 deaths for the period 27 July to 14 August.

Brazil reported 9 cases (no fatalities) from 16 July to 2 August. Its first cholera death was however reported on 14 August; the patient was an 11-year-old girl from an isolated settlement in the State of Amazonas.

Peru has reported a further 2842 cases and 44 deaths for the period 23 July to 1 August.

The Pan American Health Organization reports that 14 cases of cholera were reported in the **United States of America** from 9 April to 31 May 1991. Two of the cases occurred in travellers who had recently returned from Peru and Ecuador. The other 12 cases occurred in 2 groups of people, who consumed shellfish brought into the country by individuals returning from Ecuador.

Cholera in Asia Update

A gastroenteritis outbreak has been reported from **Nepal**. Nine cases of cholera were diagnosed (during the period 14-30 June); seven of the cases occurred in Kathmandu Valley. The Baitadi District has been declared cholera-infected.

Other recent cholera reports from Asia are:

India - 598 cases with 6 deaths during May and 751 cases with 12 deaths during June

Singapore - 3 cases (1 imported) for the period 14-27 July

Hong Kong - 1 case reported on 2 August.

Dengue Epidemic in Cook Islands

The South Pacific Commission's Epidemiological and Health Information Service has provided information about the dengue epidemic which occurred in the Cook Islands earlier this year.

Cases may have begun to occur as early as December 1990 in Rarotonga, and by February, had begun to increase. A total of 833 cases were reported, with 293 cases in February, and 394 in March. The majority of cases (531) were from Rarotonga and the remainder were from the outer islands. The number of cases may, however have been underestimated as the cases were often misdiagnosed as 'influenza-like' illnesses. There were numerous cases with mild to severe haemorrhagic manifestations and four deaths were reported. Three were associated with acute upper gastrointestinal bleeding.

Public health officers have found no *Aedes aegypti* mosquitoes and it is strongly suspected that the vector is *Aedes polynesiensis*. A mass clean-up of Rarotonga island was performed and insecticide spraying of homes of suspected cases and of mosquito breeding areas was carried out. The causative virus was identified as dengue-1.

Influenza in New Zealand

Influenza activity is now decreasing in New Zealand, according to reports from the New Zealand national network of sentinel general practitioners (Table). The majority of cases is continuing to occur in the South Island, and influenza B remains the predominant subtype isolated from swabs collected through the network.

The WHO National Influenza Centre at the Commonwelath Serum Laboratories (CSL) in Melbourne has further characterised the New Zealand influenza type B isolates. Although there is some variation among the strains, all of those typed to date appear close to B/Yamagata/16/88 or the minor variant B/Panama/45/90. Ferret sera raised against B/Yamagata/16/88 show good titres against the New Zealand isolates, and a preliminary study with post-vaccination sera from a group of Australian vaccine recipients indicates that the current vaccine, which contains B/Yamagata strain, produces satisfactory responses to the New Zealand isolates.

According to CSL, there is now also some mild to moderate influenza A H1N1 activity in New Zealand, particularly in the North Island. Overall, the level of influenza is considered to be lower than in last year's epidemic, however, the picture is confused by a concurrent epidemic of respiratory syncytial virus and infection with a variety of other agents including *Mycoplasma pneumoniae*, adenovirus, *Chlamydia pneumoniae* and parainfluenza viruses types 2 and 3.

Table. Recent influenza in New Zealand: consultations and laboratory confirmations

WEEK	DATES	SENTINEL GP INFLUENZA-LIKE ILLNESS REPORTS	SENTINEL GP SWABS		CASES REPORTED TO AREA HEALTH BOARDS (LABORATORY-CONFIRMED)
			TOTAL TAKEN	INFLUENZA B POSITIVE	
29	13-19 Jul	876	38	6	15
30	20-26 Jul	802	36	4	9
31	27 Jul - 2 Aug	686	32	8	13
32	3-9 Aug	636	35	8*	14

* One Influenza A (H1N1) was also identified, from Taranaki

COMMUNICABLE DISEASES SURVEILLANCE

CDI LABORATORY REPORTING SCHEMES

A total of 2244 reports were processed for the latest reporting period (31 July - 13 August 1991). They included some reports from the State Health Laboratory, Brisbane, on specimens collected over the past few months.

A further 29 reports of **influenza B** were received, bringing the total number of reports of this virus to 95 for the year (Figure 1). Syndromes reported for cases this period were respiratory (21 cases), general malaise/mild fever (3 cases) and encephalitis (one 8-year-old girl). Cases were recorded from Sydney, Adelaide, Alice Springs, Canberra, Melbourne, Brisbane and Perth. In addition, there were 4 reports of influenza A this period.

ASPREN reports of consultations for influenza have also increased in the last 2 reporting weeks (Figure 2; p 303 *CDI*, this issue).

The WHO National Influenza Centre at the Commonwealth Serum Laboratories in Melbourne has received 28 influenza isolates for typing in the last 2 months. Of these, 24 are Type B strains and the majority of those studied to date (14 out of 20) are nost closely related to B/Victoria/2/87. Isolates from South Australia have been exclusively B/Vic/2/87 - like whilst the majority of recent isolates from Victoria are more closely related to the B/Yamagata/16/88 virus.

Figure 1. Influenza B reports 1991, by month of specimen collection.

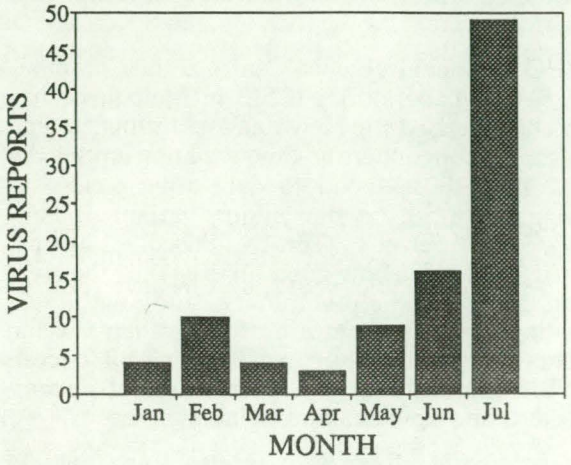
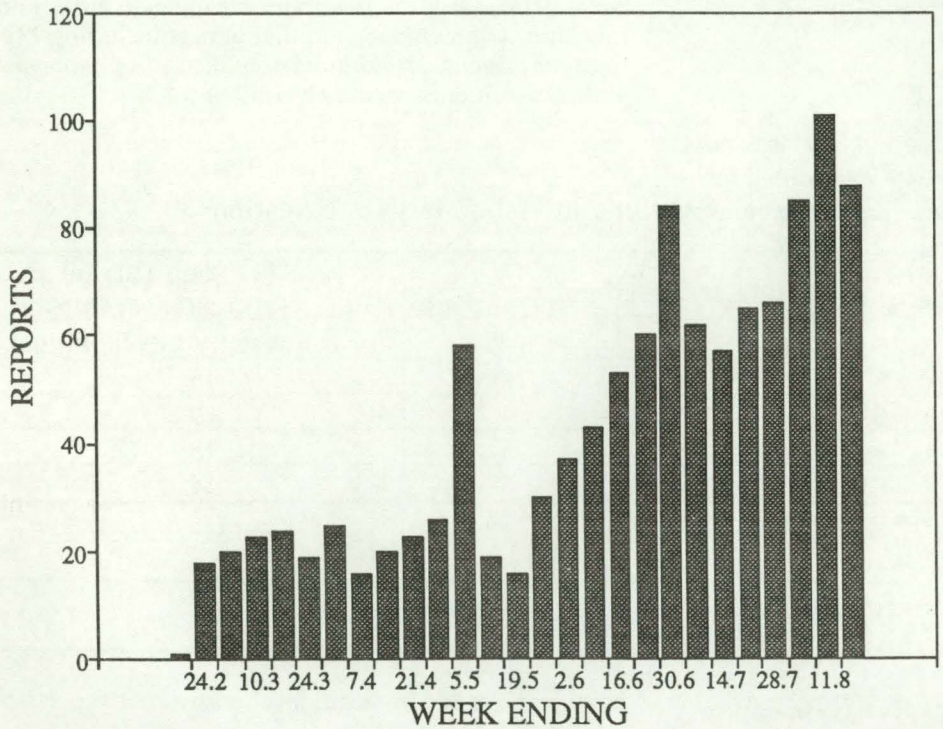


Figure 2. ASPREN reports of influenza, weeks 1-27, 1991*.



*Note: ASPREN data may be subject to revision. The number of practices submitting reports each week can vary.

Nine reports of **rubella** were received. Three were women of child-bearing age (24yrs, 43yrs, 21yrs). Three reported cases were in teenage males (ages 16yrs, 17yrs, 17yrs) from Hobart who had all suffered significant disease. There is no known connection between the three cases.

One case of *Haemophilus influenza e* **type b** meningitis has been reported. It occurred in a female, age group 1-12 months. The organism was isolated from a CSF sample.

Rotavirus reports were received from laboratories in Sydney, Melbourne, Adelaide, Perth, Hobart and Brisbane this period. The total number of reports for the year is 975, which is higher than the average received by this time of year.

Following the introduction of a new code, reports of **parvovirus** have been received for the first time. Four cases were reported, all from a Sydney hospital. The patients were an 11-month-old boy with red cell aplasia, a 25-day-old girl with cardiovascular disease, a 2-year-old girl investigated for viral illness and lowered neutrophils and platelets, and an 11-year-old boy with skin disease. IgM was detected in all cases by the Department of Infectious Diseases, Sydney University.

Human parvovirus infection is caused by parvovirus B19. It is common in children, causing both sporadic cases and epidemics which tend to occur in winter and spring in temperate zones. In the USA, the prevalence of IgG to parvovirus B19 increases from 5-10% in children less than 5 years old to more than 50% in adults. Manifestations of the infection are typically erythema infectiosum in children, arthritis in adults, aplastic anaemia in patients with haemolytic syndromes, and foetal anaemia with hydrops foetalis if contracted in pregnancy[1]. A detailed review of human parvovirus B19 infection was published in *CDI* 89/8 (1989).

Separate codes for the Dengue virus subtypes were introduced in 1990, and Dengue-1, -2, and -3 have since been reported. The first report of **Dengue-4** was received this period; the patient was a 33-year-old female who had specific IgM to the virus, and who had recently returned from Bali. Three reports of dengue -4 had formerly been recorded under the 'Dengue not typed' code. Two of these cases were in 1985 (one had a history of travel to THailand) and the other was recorded in 1988.

The usual winter peak in **respiratory syncytical virus** activity appears to be occurring later than the average for recent years (Figure 3). Most cases reported this year have been in young children (Figure 4), as is usually the case. Syndromes reported have been respiratory (1114 cases), skin disease (2 cases), fever of unknown origin (10 cases) general malaise/mild fever (10 cases), gastrointestinal disease (10 cases), encephalitis (1 case) and other CNS symptoms (1 case).

Figure 3. Respiratory syncytial virus reports 1986-90 average and 1991.

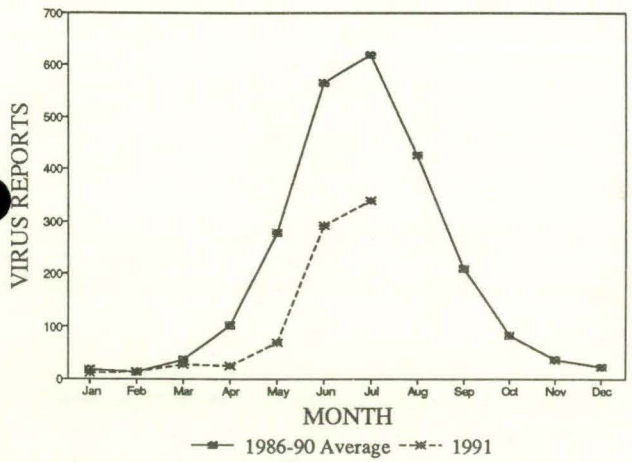
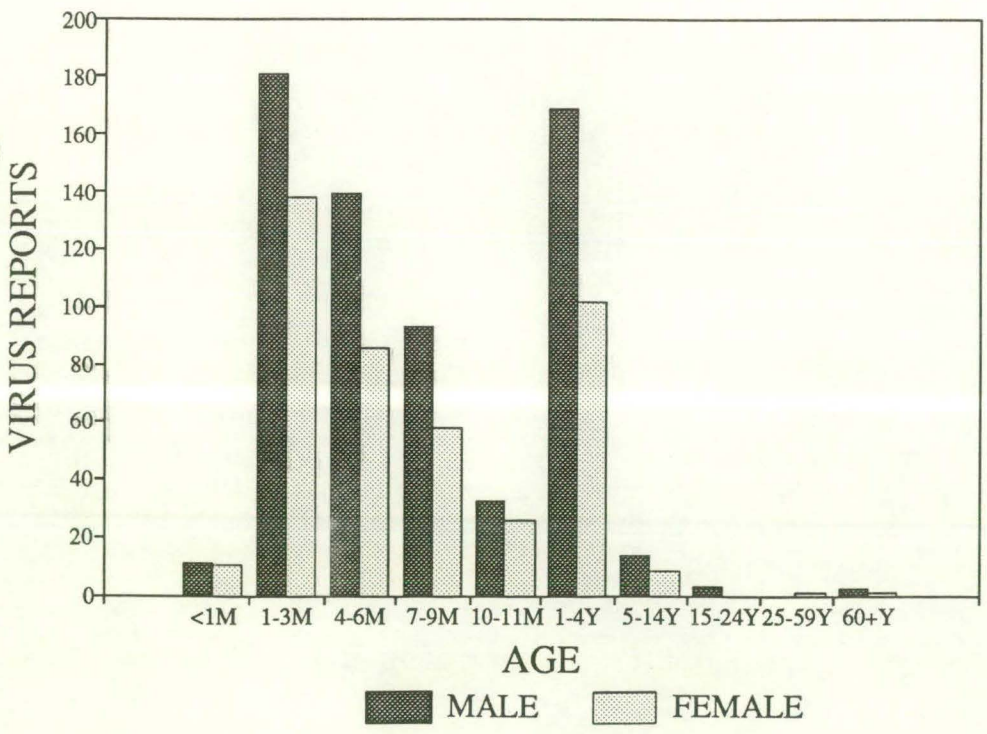


Figure 4. Respiratory syncytial virus reports 1991, by age group.



Twelve reports of **Barmah Forest virus** were received. Three patients (a 5-year-old female, a 37-year-old male and a 44-year-old male) had suffered joint disease, and 2 patients had general malaise/mild fever; no syndrome was reported for the remaining patients. A total of 22 reports of this virus have been received so far this year.

There were 40 reports of **Q fever** this fortnight. Five were meatworkers, one was described as a farmer and one as a grazier.

There were 163 further reports of **hepatitis C**. Two haemophiliacs and a patient with Thalassaemia Major were among those whe were found to have seroconverted to this virus.

There have been a large number of cases of **psittacosis** (*Chlamydia psittaci*) this year, with a total of 74 cases reported so far. This compares with an average of 87.6 cases per year for the period 1986-90. In this reporting period, a report of a male patient, aged 62 years, was accompanied by the comment '4 dead canaries'.

Reports of **hepatitis A** are still being made at a higher rate than usual. There were 34 reports in the last 4 weeks, bringing the total for the year to 239. The majority of cases are still occurring in adult males; 19 of the recent reports have been of males, aged 15 or over. (Further details of recent hepatitis A activity in Australia were in CDI 15:244-253).

There were 84 reports of **Ross River virus** this period, covering sample collection dates from April to July. Most patients were from Queensland, but there were 3 patients from Western Australia, 4 from New South Wales and 4 from Tasmania. We are now well past the peak for Ross River virus activity, which occurred in March this year (197 reports).

Nineteen reports of measles were recieved this period and there have been more than 20 reports each month since March. The total for this year is 141, the highest number reported for a year since 1983, apart from last year, when there were 221 reports. Notifiable disease reports of measles have also been higher than the historical average for the periods covering 6 January to 20 July 1991 (*CDI* **15**:82,126,159,186,220,257 and p303 this issue).

Table. Echovirus type 17 reports, 1991: patient details.

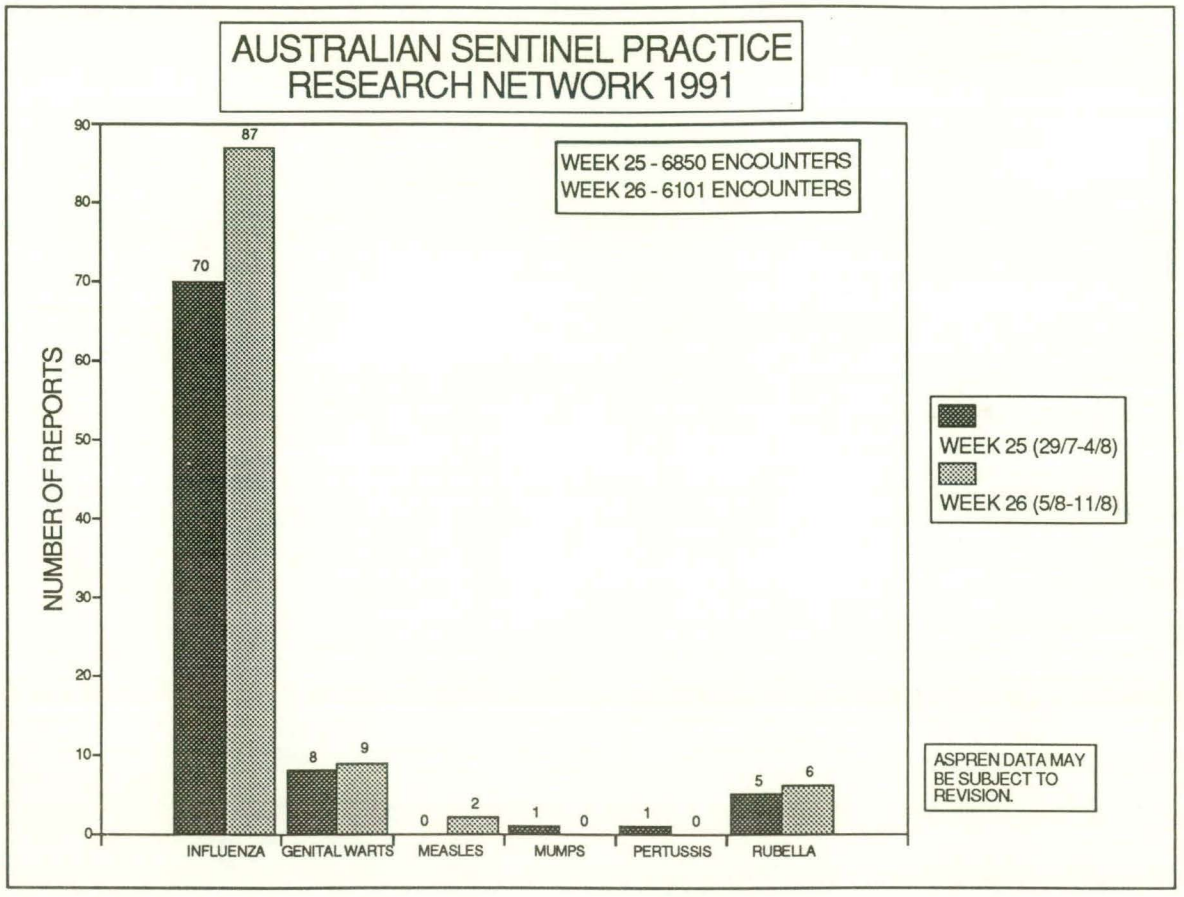
Syndrome	Isolate Source Tissue	Sex and Age
Meningitis	CSF	M/7mths[1], M/<1 mth, F/1mth[1,2], F/1mth[3], M/43yrs, M/18yrs, F/37yrs, M/7yrs, M/5yrs, F/14yrs, M/1yr[1], M/29yrs,
	Faeces	F/<1mth, F/1mth
GeneralMalaise/mild fever	CSF	F/5mths, M/ age unknown, M/3mths
	Faeces	F/1yr[3]
Fever of unknown origin	CSF	F/2mths
Gastrointestinal disease	Faeces	M/1yr, M/6yrs, M/2yrs[4]
Other CNS diseas	Faeces	F/1yr[2]
Lower respiratory tract disease	Nasopharyngeal	M/age unknown
No clinical information provided	CSF	M/7yrs, unknown/2mths, M/8yrs, F/5mths, F/4mths

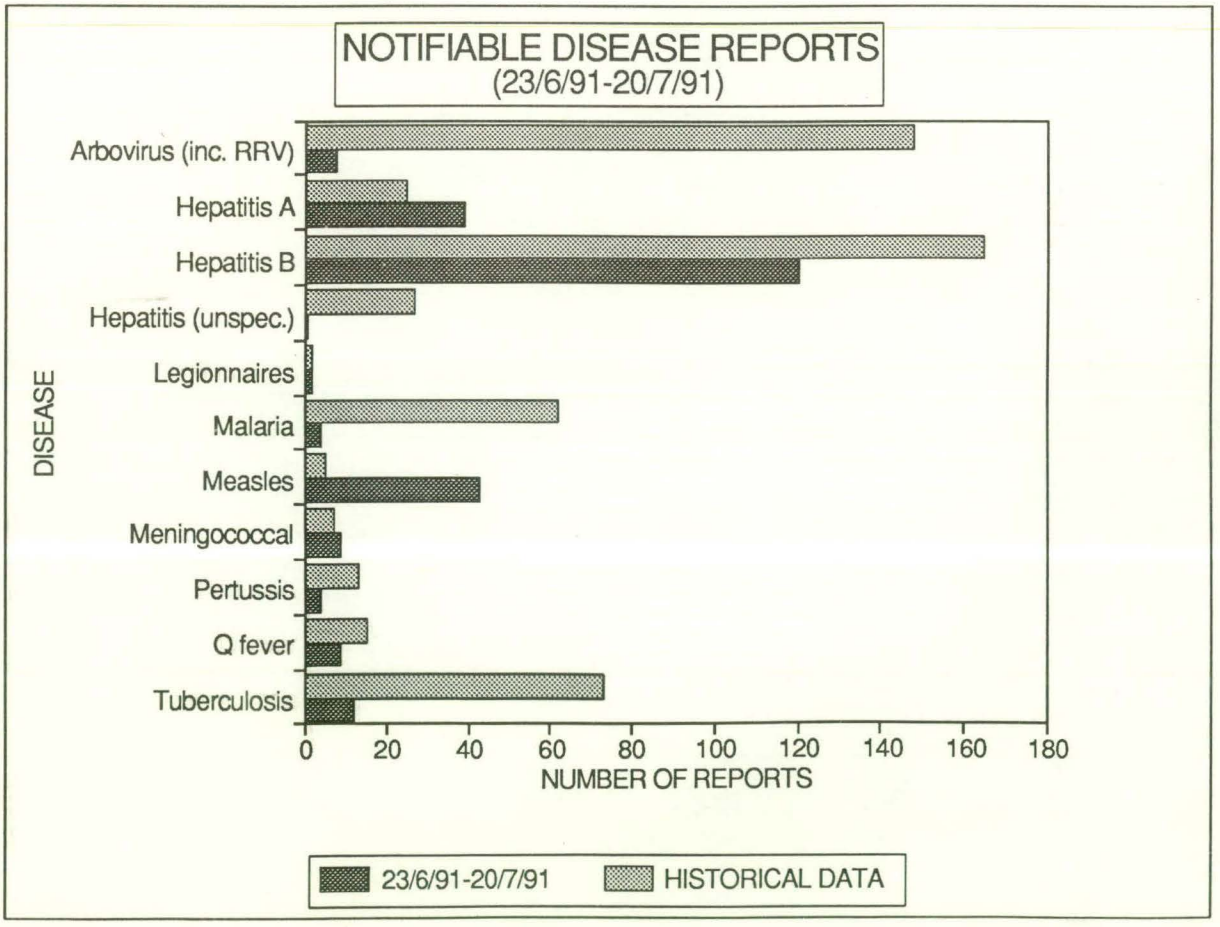
There have been 14 further reports of echovirus type 17, bringing the totals for June to 14, July to 5 and the year to 29, the highest number of reports since 1982. Meningitis was the reported syndrome for 8 cases this period, with skin disease, general malaise/mild fever and fever of unknown origin also reported. Meningitis has been the syndrome most commonly reported for all the cases this year and for 7 of the 12 cases in children under 12 months old (Table).

Most of this period's cases have been reported by Sydney laboratories, but 2 were reported from Canberra.

REFERENCE

1. Benenson AS (Ed). *Control of Communicable Disease in Man.* American Public Health Association. Washington. 1990.


AUSTRALIAN SENTINEL PRACTICE RESEARCH NETWORK 1991
WEEK 25 - 6850 ENCOUNTERS
WEEK 26 - 6101 ENCOUNTERS
NUMBER OF REPORTS
90
80
70
60
50
40
30
20
10
0
87
70
8
9
0
2
1
0
1
0
5
6
INFLUENZA
GENITAL WARTS
MEASLES
MUMPS
PERTUSSIS
RUBELLA
WEEK 25 (29/7-4/8)
WEEK 26 (5/8-11/8)
ASPREN DATA MAY BE SUBJECT TO REVISION.


NOTIFIABLE DISEASE REPORTS
(23/6/91-20/7/91)
DISEASE
Arbovirus (inc. RRV)
Hepatitis A
Hepatitis B
Hepatitis (unspec.)
Legionnaires
Malaria
Measles
Meningococcal
Pertussis
Q fever
Tuberculosis
0
20
40
60
80
100
120
140
160
180
NUMBER OF REPORTS
23/6/91-20/7/91
HISTORICAL DATA

National Notifiable Disease Reports 23/6/90-20/7/91

DISEASES	ACT	NSW*	NT	QLD	SA	TAS	VIC	WA	TOTAL
Arbovirus Infections (unspecified)	0	0	0	0	0	2	0	0	2
Ross River Virus	0	0	0	6	0	0	0	0	6
Dengue fever	0	NN	0	0	0	0	0	0	0
Brucellosis	0	0	0	0	0	0	0	0	0
Campylobacter	4	0	21	16	135	52	28	0	256
Chancroid	0	NN	0	0	NN	NN	NN	0	0
Chlamydia	2	0	23	13	95**	24	NN	0	157
Cholera	0	0	0	0	0	0	0	0	0
Diphtheria	0	0	0	0	0	0	0	0	0
Donovanosis	0	NN	1	0	NN	NN	NN	0	1
Gonococcal diseases	1	0	24	2	19**	1	0	0	47
Haemophilus influenzae b	0	0	0	0	0	0	6	0	6
HIV infections	1	0	0	0	1	0	0	0	2
Hydatid disease	0	0	0	0	0	0	0	0	0
Legionnaires disease	NN	0	0	0	2	0	0	0	2
Leprosy	0	0	0	0	0	0	2	0	2
Leptospirosis	0	0	0	0	0	0	1	0	1
Listeriosis	0	NN	0	0	0	0	0	0	0
Lymphogranuloma venereum	0	NN	NN	0	NN	NN	NN	NN	0
Malaria	0	0	0	0	2	1	1	0	4
Measles	1	0	1	0	12	3	26	0	43
Meningococcal infections	0	0	0	0	2	2	5	0	9
Ornithosis	0	NN	0	0	0	0	7	0	7
Pertussis	0	0	0	0	3	0	1	0	4
Plague	0	NN	0	0	0	0	0	0	0
Poliomyelitis	0	0	0	0	0	0	0	0	0
Q fever	1	0	0	3	4	0	1	0	9
Rabies	NN	NN	0	0	0	0	0	0	0
Rubella	0	0	0	0	6	0	3	0	9
Salmonella	0	0	16	12	29	23	9	0	89
Shigella	0	0	14	2	3	0	2	0	21
Syphilis	1	0	13	0	9**	0	0	0	23
Tetanus	0	0	0	0	0	0	0	0	0
Tuberculosis	0	0	0	0	8	1	3	0	12
Typhoid	0	0	0	0	0	0	2	0	2
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Viral hepatitis (unspecified)	0	0	0	0	1	0	0	NN	1
Hepatitis A	1	0	1	1	16	0	20	0	39
Hepatitis B	0	0	2	9	2	2	105	0	120
Hepatitis C	1	0	0	1	0	1	6	0	9
Yellow fever	0	0	0	0	0	0	0	0	0
Yersiniosis	0	0	0	2	12	0	0	0	14

* data for June 1991

** data to June 29 1991

*** typhoid and paratyphoid

NN not notifiable

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 31/07/91 TO 13/08/91

CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 065 - STATE HEALTH LABORATORY SERVICES, PERTH (WA)
CODE 066 - PRINCESS MARGARET HOSPITAL, PERTH (WA)
CODE 110 - INSTITUTE OF MEDICAL & VETERINARY SCIENCE, ADELAIDE (SA)
CODE 111 - ROYAL CHIDLDRENS HOSPITAL, MELBOURNE (VIC)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 113 - PRINCE HENRY/PRINCE OF WALES HOSPITALS, SYDNEY (NSW)
CODE 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)
CODE 270 - TAMWOTH LAB, NEW ENGLAND PATHOLOGY (NSW)
CODE 400 - DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON (QLD)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

	019	065	066	110	111	112	113	114	115	116	270	400	RHH	TPL	TOTAL
0100 ADENOVIRUS NOT TYPED	0	1	4	10	20	3	4	1	11	0	0	0	0	0	54
0101 ADENOVIRUS TYPE 1	0	0	0	0	0	1	1	0	0	0	0	0	1	0	3
0102 ADENOVIRUS TYPE 2	4	0	0	0	0	4	1	0	0	0	0	0	0	0	9
0103 ADENOVIRUS TYPE 3	5	0	0	1	0	3	0	0	0	0	0	0	0	0	9
0104 ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1
0105 ADENOVIRUS TYPE 5	0	0	0	0	0	1	0	2	0	0	0	0	0	0	3
0106 ADENOVIRUS TYPE 6	0	0	0	3	0	0	0	0	0	0	0	0	0	0	3
0108 ADENOVIRUS TYPE 8	1	0	0	0	0	1	0	0	0	0	0	0	0	0	2
0111 ADENOVIRUS TYPE 11	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
0127 ADENOVIRUS TYPE 27	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
0129 ADENOVIRUS TYPE 29	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1
0131 ADENOVIRUS TYPE 31	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1
0199 ADENOVIRUS TYPING PENDING	0	0	0	1	8	0	0	5	0	0	0	0	0	0	14
0201 INFLUENZA A VIRUS	0	1	0	0	0	0	1	0	2	0	0	0	0	0	4
0203 INFLUENZA B VIRUS	8	1	0	13	2	2	0	1	1	1	0	0	0	0	29
0302 PARAINFLUENZA VIRUS TYPE 2	0	0	1	2	1	0	0	0	0	0	0	0	0	0	4
0303 PARAINFLUENZA VIRUS TYPE 3	0	1	0	5	8	4	0	1	7	0	0	0	0	0	26
0399 PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	5	0	0	0	0	0	0	0	0	0	5
0400 RESPIRATORY SYNCYTIAL VIRUS (R	39	1	43	24	118	42	28	37	27	20	8	1	0	2	390
0500 RHINOVIRUS (ALL TYPES)	7	2	0	0	17	1	2	11	9	0	0	0	2	0	51
0600 MYCOPLASMA PNEUMONIAE	5	2	0	0	3	4	4	1	3	0	0	0	0	0	22
0700 ORNITHOSIS-PSITTACOSIS	3	0	0	0	0	0	1	0	0	0	0	0	0	0	4
0809 COXSACKIEVIRUS A9	1	0	0	0	0	1	3	0	0	0	0	0	0	0	5
0902 COXSACKIEVIRUS B2	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
0904 COXSACKIEVIRUS B4	1	0	0	0	0	3	0	0	0	0	0	0	1	0	5
0905 COXSACKIEVIRUS B5	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
1017 ECHOVIRUS TYPE 17	0	0	0	0	0	5	4	3	0	2	0	0	0	0	14
1022 ECHOVIRUS TYPE 22	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
1100 POLIOVIRUS NOT TYPED	0	0	0	0	0	0	8	0	0	0	0	0	0	0	8
1101 POLIOVIRUS TYPE 1	0	0	0	0	0	2	0	0	0	0	0	0	0	0	2
1102 POLIOVIRUS TYPE 2	3	0	0	0	0	2	0	2	0	0	0	0	0	0	7
1103 POLIOVIRUS TYPE 3	0	0	0	2	0	3	0	0	0	0	0	0	0	0	5
1300 HERPES VIRUS GROUP - NOT TYPED	3	0	0	0	0	0	0	0	1	0	0	0	0	0	4
1301 HERPES SIMPLEX VIRUS - NOT TYP	1	1	3	0	0	24	1	3	0	7	1	1	0	0	42
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	3	15	0	9	7	5	1	3	27	0	0	23	1	0	94
1303 VARICELLA-ZOSTER VIRUS	3	7	0	1	0	7	1	0	4	1	0	0	0	0	24
1306 HERPES SIMPLEX TYPE 1	32	46	1	21	4	5	16	0	40	0	1	1	0	0	167
1307 HERPES SIMPLEX TYPE 2	44	90	1	31	0	7	24	0	20	0	0	0	2	0	219
1399 HERPES VIRUS TYPING PENDING	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1
1401 COXIELLA BURNETII	1	0	0	4	0	4	0	0	28	0	0	3	0	0	40
1502 PICORNA VIRUS - NOT TYPED = EN	0	11	0	0	0	0	22	0	2	0	0	0	0	0	35
1514 MOLLUSCUM CONTAGIOSUM	0	0	0	0	0	0	0	0	0	0	0	2	0	0	2
1515 CONTAGIOUS PUSTULAR DERMATITIS	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
1521 MEASLES VIRUS	4	0	0	2	7	0	2	0	4	0	0	0	0	0	19
1522 RUBELLA VIRUS	0	0	0	0	0	4	1	0	2	0	0	2	0	0	9
1532 HEPATITIS B ANTIGEN	4	41	0	0	0	22	5	0	27	3	0	2	0	0	104
1535 HEPATITIS A ANTIBODY	0	9	0	0	0	9	4	0	0	0	0	0	0	0	22
1536 HEPATITIS C VIRUS	100	54	0	0	0	0	0	2	0	1	0	0	6	0	163
1537 HEPATITIS, DELTA	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
1541 CHLAMYDIA TRACHOMATIS - UNSPEC	0	42	0	27	1	11	3	0	22	6	0	0	3	5	120
1542 CHLAMYDIA TRACHOMATIS - A-K	0	0	0	0	0	0	0	0	0	0	0	10	0	0	10
1556 CMV - CYTOMEGALOVIRUS	17	4	17	2	9	6	9	4	32	1	0	3	0	0	104
1564 ROTAVIRUS	2	2	7	18	60	24	34	15	0	0	6	38	3	18	227
1565 CALICI VIRUS	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
1599 ENTEROVIRUS TYPING PENDING	0	0	0	0	12	0	2	2	0	0	0	0	0	0	16
1700 PARVOVIRUS	0	0	0	0	0	0	0	4	0	0	0	0	0	0	4
9903 NON-A, NON-B HEPATITIS (OTHER)	0	0	0	0	0	0	0	0	0	0	0	0	2	0	2
9906 BARMAH FOREST VIRUS	0	0	0	0	0	0	2	0	10	0	0	0	0	0	12
9981 DENGUE TYPE 1	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1
9982 DENGUE TYPE 2	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1
9984 DENGUE TYPE 4	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
9990 AUSTRALIAN ENCEPHALITIS	0	0	0	0	0	0	0	0	3	0	0	0	0	0	3
9992 ROSS RIVER VIRUS	0	3	0	0	0	2	0	0	75	0	0	4	0	0	84
9993 ASTROVIRUS	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
9994 SMALL VIRUS (LIKE) PARTICLE	2	0	0	0	0	0	0	1	0	0	0	0	0	0	3
9995 DENGUE NOT TYPED	0	0	0	0	0	0	0	0	2	0	0	0	0	0	2
9997 KUNJIN VIRUS	0	0	0	0	0	0	0	0	5	0	0	0	0	0	5
9998 ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	0	0	12	0	0	0	0	0	12
TOTAL	295	336	77	177	283	219	184	98	378	43	16	90	21	25	2242

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 31/07/91 TO 13/08/91

NSW: ICPMR; PHH/POW; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP; TAMWRTH LAB.
VIC: FAIRFIELD; RCH; MDU, UNI MELB.
QLD: STATE LAB, BRIS; TOOWOOMBA PATH LAB; ROYAL BRIS HOSP; DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON.
WA: STATE LAB, PERTH; PMH.
SA: IMVS.
TAS: ROYAL HOBART HOSP; DIAGNOSTIC SERVICES, LAUNCESTON; LAUNCESTON GEN HOSP; DIAGNOSTIC SERVICES, HOBART; HOBART PATH; MERSEY GEN HOSP, LATROBE.
ACT: WVH.

		NSW	VIC	QLD	WA	SA	TAS	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	8	20	11	5	10	0	0	54
0101	ADENOVIRUS TYPE 1	2	0	0	0	0	1	0	3
0102	ADENOVIRUS TYPE 2	5	4	0	0	0	0	0	9
0103	ADENOVIRUS TYPE 3	3	5	0	0	1	0	0	9
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	3	0	0	0	0	0	0	3
0106	ADENOVIRUS TYPE 6	0	0	0	0	3	0	0	3
0108	ADENOVIRUS TYPE 8	1	1	0	0	0	0	0	2
0111	ADENOVIRUS TYPE 11	1	0	0	0	0	0	0	1
0127	ADENOVIRUS TYPE 27	1	0	0	0	0	0	0	1
0129	ADENOVIRUS TYPE 29	0	1	0	0	0	0	0	1
0131	ADENOVIRUS TYPE 31	0	0	0	0	1	0	0	1
0199	ADENOVIRUS TYPING PENDING	5	8	0	0	1	0	0	14
0201	INFLUENZA A VIRUS	1	0	2	1	0	0	0	4
0203	INFLUENZA B VIRUS	3	10	1	1	13	0	1	29
0302	PARAINFLUENZA VIRUS TYPE 2	0	1	0	1	2	0	0	4
0303	PARAINFLUENZA VIRUS TYPE 3	5	8	7	1	5	0	0	26
0399	PARAINFLUENZA VIRUS TYPING PEN	0	5	0	0	0	0	0	5
0400	RESPIRATORY SYNCYTIAL VIRUS (R	115	157	30	44	24	0	20	390
0500	RHINOVIRUS (ALL TYPES)	14	24	9	2	0	2	0	51
0600	MYCOPLASMA PNEUMONIAE	9	8	3	2	0	0	0	22
0700	ORNITHOSIS-PSITTACOSIS	1	3	0	0	0	0	0	4
0809	COXSACKIEVIRUS A9	4	1	0	0	0	0	0	5
0902	COXSACKIEVIRUS B2	0	0	0	0	0	0	1	1
0904	COXSACKIEVIRUS B4	3	1	0	0	0	1	0	5
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	0	1
1017	ECHOVIRUS TYPE 17	12	0	0	0	0	0	2	14
1022	ECHOVIRUS TYPE 22	1	0	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	8	0	0	0	0	0	0	8
1101	POLIOVIRUS TYPE 1	2	0	0	0	0	0	0	2
1102	POLIOVIRUS TYPE 2	4	3	0	0	0	0	0	7
1103	POLIOVIRUS TYPE 3	3	0	0	0	2	0	0	5
1300	HERPES VIRUS GROUP - NOT TYPED	0	3	1	0	0	0	0	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	29	1	1	4	0	0	7	42
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	9	10	50	15	9	1	0	94
1303	VARICELLA-ZOSTER VIRUS	8	3	4	7	1	0	1	24
1306	HERPES SIMPLEX TYPE 1	22	36	41	47	21	0	0	167
1307	HERPES SIMPLEX TYPE 2	31	44	20	91	31	2	0	219
1399	HERPES VIRUS TYPING PENDING	0	1	0	0	0	0	0	1
1401	COXIELLA BURNETII	4	1	31	0	4	0	0	40
1502	PICORNA VIRUS - NOT TYPED = EN	22	0	2	11	0	0	0	35
1514	MOLLUSCUM CONTAGIOSUM	0	0	2	0	0	0	0	2
1515	CONTAGIOUS PUSTULAR DERMATITIS	1	0	0	0	0	0	0	1
1521	MEASLES VIRUS	2	11	4	0	2	0	0	19
1522	RUBELLA VIRUS	5	0	4	0	0	0	0	9
1532	HEPATITIS B ANTIGEN	27	4	29	41	0	0	3	104
1535	HEPATITIS A ANTIBODY	13	0	0	9	0	0	0	22
1536	HEPATITIS C VIRUS	2	100	0	54	0	6	1	163
1537	HEPATITIS, DELTA	0	0	0	1	0	0	0	1
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	14	1	27	42	27	3	6	120
1542	CHLAMYDIA TRACHOMATIS - A-K	0	0	10	0	0	0	0	10
1556	CMV - CYTOMEGALOVIRUS	19	26	35	21	2	0	1	104
1564	ROTAVIRUS	79	62	56	9	18	3	0	227
1565	CALICI VIRUS	1	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	4	12	0	0	0	0	0	16
1700	PARVOVIRUS	4	0	0	0	0	0	0	4
9903	NON-A, NON-B HEPATITIS (OTHER)	0	0	0	0	0	2	0	2
9906	BARMAH FOREST VIRUS	2	0	10	0	0	0	0	12
9981	DENGUE TYPE 1	0	0	1	0	0	0	0	1
9982	DENGUE TYPE 2	0	0	1	0	0	0	0	1
9984	DENGUE TYPE 4	0	0	0	1	0	0	0	1
9990	AUSTRALIAN ENCEPHALITIS	0	0	3	0	0	0	0	3
9992	ROSS RIVER VIRUS	2	0	79	3	0	0	0	84
9993	ASTROVIRUS	1	0	0	0	0	0	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	1	2	0	0	0	0	0	3
9995	DENGUE NOT TYPED	0	0	2	0	0	0	0	2
9997	KUNJIN VIRUS	0	0	5	0	0	0	0	5
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	12	0	0	0	0	12
TOTAL		517	578	493	413	177	21	43	2242

NOTE: DIRECT COMPARISION BETWEEN STATES IS NOT POSSIBLE SINCE:
- SOME STATES HAVE MORE THAN ONE CONTRIBUTING LABORATORY; AND
- INTERSTATE REFERRRALS OCCUR REGULARLY.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 31/07/91 TO 13/08/91

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	27	0	0	0	1	23	0	0	0	1	53
0101	ADENOVIRUS TYPE 1	0	2	0	0	0	0	0	0	0	0	0	2
0102	ADENOVIRUS TYPE 2	1	4	0	0	0	0	2	0	0	0	0	7
0103	ADENOVIRUS TYPE 3	0	0	0	0	0	0	2	0	0	0	0	2
0105	ADENOVIRUS TYPE 5	0	2	0	0	0	0	1	0	0	0	0	3
0106	ADENOVIRUS TYPE 6	0	2	0	0	0	0	0	0	0	0	0	2
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	1	0	0	0	0	1
0131	ADENOVIRUS TYPE 31	0	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	8	0	0	0	0	3	0	0	0	1	13
0201	INFLUENZA A VIRUS	0	2	0	0	0	0	0	0	0	0	0	2
0203	INFLUENZA B VIRUS	1	19	1	0	0	0	0	0	0	0	0	21
0302	PARAINFLUENZA VIRUS TYPE 2	0	4	0	0	0	0	0	0	0	0	0	4
0303	PARAINFLUENZA VIRUS TYPE 3	2	20	0	0	0	0	1	0	0	0	0	23
0399	PARAINFLUENZA VIRUS TYPING PEN	0	5	0	0	0	0	0	0	0	0	0	5
0400	RESPIRATORY SYNCYTIAL VIRUS (R	10	366	0	0	0	1	3	0	0	0	0	380
0500	RHINOVIRUS (ALL TYPES)	3	44	0	0	0	0	0	0	0	0	0	47
0600	MYCOPLASMA PNEUMONIAE	3	15	0	0	0	0	0	0	0	0	1	19
0700	ORNITHOSIS-PSITTACOSIS	1	2	0	0	0	0	0	0	0	0	0	3
0809	COXSACKIEVIRUS A9	0	2	0	2	0	0	0	0	0	0	0	4
0902	COXSACKIEVIRUS B2	0	0	0	1	0	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	2	0	1	0	0	0	0	0	0	0	0	3
0905	COXSACKIEVIRUS B5	0	0	0	1	0	0	0	0	0	0	0	1
1017	ECHOVIRUS TYPE 17	1	0	0	8	0	0	1	0	0	0	0	10
1100	POLIOVIRUS NOT TYPED	0	4	0	0	0	0	4	0	0	0	0	8
1101	POLIOVIRUS TYPE 1	1	0	0	0	0	0	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	3	4	0	0	0	0	0	0	0	0	0	7
1103	POLIOVIRUS TYPE 3	0	2	0	0	0	0	2	0	0	0	0	4
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	0	3	3
1301	HERPES SIMPLEX VIRUS - NOT TYP	7	2	1	0	0	0	0	0	0	0	15	25
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	20	21	0	0	0	0	2	6	1	0	0	50
1303	VARICELLA-ZOSTER VIRUS	2	1	1	0	0	0	0	0	0	0	19	23
1306	HERPES SIMPLEX TYPE 1	3	12	0	0	0	0	0	1	0	1	113	130
1307	HERPES SIMPLEX TYPE 2	0	0	0	0	0	0	0	0	0	0	123	123
1399	HERPES VIRUS TYPING PENDING	0	0	1	0	0	0	0	0	0	0	0	1
1401	COXIELLA BURNETII	9	3	0	0	0	0	0	2	2	0	0	16
1502	PICORNA VIRUS - NOT TYPED = EN	2	2	0	3	0	0	22	0	0	0	1	30
1514	MOLLUSCUM CONTAGIOSUM	0	0	0	0	0	0	0	0	0	0	1	1
1515	CONTAGIOUS PUSTULAR DERMATITIS	0	0	0	0	0	0	0	0	0	0	1	1
1521	MEASLES VIRUS	4	0	0	0	1	0	0	0	0	0	10	15
1522	RUBELLA VIRUS	3	0	0	0	0	0	0	0	0	0	3	6
1532	HEPATITIS B ANTIGEN	53	0	0	0	0	0	0	46	0	1	1	101
1535	HEPATITIS A ANTIBODY	4	0	0	0	0	0	1	17	0	0	0	22
1536	HEPATITIS C VIRUS	143	0	0	0	0	0	0	13	0	0	0	156
1537	HEPATITIS, DELTA	1	0	0	0	0	0	0	0	0	0	0	1
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	4	1	0	0	0	0	0	0	0	0	0	5
1542	CHLAMYDIA TRACHOMATIS - A-K	4	0	0	0	0	0	0	0	0	0	0	4
1556	CMV - CYTOMEGALOVIRUS	12	39	1	1	1	1	2	3	1	2	1	64
1564	ROTAVIRUS	11	6	0	0	0	0	208	0	0	0	1	226
1565	CALICI VIRUS	0	0	0	0	0	0	1	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	8	0	1	0	0	1	0	0	0	2	12
1700	PARVOVIRUS	0	0	0	0	0	0	0	0	1	0	1	2
9903	NON-A, NON-B HEPATITIS (OTHER)	1	0	0	0	0	0	0	0	0	0	0	1
9906	BARMAH FOREST VIRUS	6	0	0	0	0	0	0	0	0	0	0	6
9982	DENGUE TYPE 2	0	0	0	0	0	0	0	1	0	0	0	1
9984	DENGUE TYPE 4	1	0	0	0	0	0	0	0	0	0	0	1
9990	AUSTRALIAN ENCEPHALITIS	1	0	1	0	0	0	0	0	0	0	0	2
9992	ROSS RIVER VIRUS	30	0	0	0	0	0	0	0	0	0	5	35
9993	ASTROVIRUS	0	0	0	0	0	0	1	0	0	0	0	1
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	3	0	0	0	0	3
9995	DENGUE NOT TYPED	0	0	0	0	0	0	1	0	0	0	0	1
9997	KUNJIN VIRUS	3	0	0	0	0	0	0	0	0	0	0	3
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	7	0	0	0	0	0	0	0	0	0	1	8
TOTAL		361	629	7	17	2	3	286	89	5	4	304	1707

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 31/07/91 TO 13/08/91

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	0	0	0	0	1	0	0	0	1
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	0	1	1	0	2
0103	ADENOVIRUS TYPE 3	7	0	0	0	0	0	0	0	0	0	7
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	0	0	0	0	0	0	0	0	1	1
0108	ADENOVIRUS TYPE 8	2	0	0	0	0	0	0	0	0	0	2
0127	ADENOVIRUS TYPE 27	0	0	0	0	0	0	0	0	1	0	1
0129	ADENOVIRUS TYPE 29	0	0	0	0	0	0	0	0	1	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	1	0	0	0	1
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	0	2	0	0	2
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	0	2	6	0	8
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	2	0	1	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	5	1	3	1	10
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	2	1	1	0	4
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	1	0	0	1	1	0	3
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	0	1	0	0	1
0904	COXSACKIEVIRUS B4	0	0	0	0	0	0	0	0	2	0	2
1017	ECHOVIRUS TYPE 17	0	0	0	0	0	0	1	3	0	0	4
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	0	0	0	0	1	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	0	0	1	1
1103	POLIOVIRUS TYPE 3	0	0	0	0	0	0	0	0	0	1	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	1	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	13	0	0	0	0	0	1	3	0	17
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	16	9	0	0	0	9	9	0	43
1306	HERPES SIMPLEX TYPE 1	2	33	0	0	0	0	0	1	1	0	37
1307	HERPES SIMPLEX TYPE 2	0	95	0	0	0	0	0	0	1	0	96
1401	COXIELLA BURNETII	0	0	0	0	3	0	0	9	12	0	24
1502	PICORNA VIRUS - NOT TYPED = EN	0	0	1	0	0	0	0	2	1	1	5
1521	MEASLES VIRUS	0	0	0	0	1	0	0	0	3	0	4
1522	RUBELLA VIRUS	0	0	0	1	0	0	0	1	1	0	3
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	1	1	0	2
1536	HEPATITIS C VIRUS	0	0	0	0	0	0	1	2	4	0	7
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	2	113	0	0	0	0	0	0	0	0	115
1542	CHLAMYDIA TRACHOMATIS - A-K	0	5	0	0	0	0	0	0	0	0	5
1556	CMV - CYTOMEGALOVIRUS	1	4	0	3	0	4	1	6	21	0	40
1564	ROTAVIRUS	0	0	0	0	0	0	1	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	1	0	3	0	4
1700	PARVOVIRUS	0	0	0	0	0	0	0	0	2	0	2
9903	NON-A, NON-B HEPATITIS (OTHER)	0	0	0	0	0	0	0	0	1	0	1
9906	BARMAH FOREST VIRUS	0	0	0	0	3	0	0	2	1	0	6
9981	DENGUE TYPE 1	0	0	0	1	0	0	0	0	0	0	1
9990	AUSTRALIAN ENCEPHALITIS	0	0	0	0	0	0	0	0	1	0	1
9992	ROSS RIVER VIRUS	0	0	0	0	28	0	0	10	11	0	49
9995	DENGUE NOT TYPED	0	0	0	0	0	0	0	1	0	0	1
9997	KUNJIN VIRUS	0	0	0	0	2	0	0	0	0	0	2
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	1	0	0	3	0	0	4
TOTAL		15	263	17	14	39	4	16	60	94	7	529