ISSN 0725 - 3141

Communicable Diseases Intelligence

Bulletin number 90/22
Issue date: 5 November 1990

Contents:

Editor
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VIRUSES, CHLAMYDIAS, COXIELLAS, RICKETTSIAS AND MYCOPLASMAS REPORTING SCHEME:

In this period (11 October to 24 October 1990) there were 1710 reports processed.

Q fever was reported on 8 occasions, all cases being male and in the age range of 22 to 53 years. Occupational exposure details were supplied for one case; the patient being a veterinarian.

There were two reports of echovirus type 2 isolated from the cerebrospinal fluid of 13-year-old and 35-year-old males with meningitis.

Coxsackie B4 was isolated from the cerebrospinal fluid of a 30-month-old girl with meningitis.

Herpes simplex type 2 was isolated from the cerebrospinal fluid of a fatal case of meningoencephalitis.

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One case of congenital rubella was reported in a 2 month old male. The patient was small for gestational age at birth, and presented with microphthalmia, glaucoma, and peripheral pulmonary stenosis.

Respiratory syncytial virus was isolated from the nasopharyngeal secretions of 3 neonates (sex unknown) with nosocomially acquired lower respiratory tract infection. All were pre-term infants being managed in a neonatal intensive care unit at the time of infection.

There has been a marked increase in measles reporting in recent months; the total for the year now having reached 126 cases.
At 1 November 1990 available details of notified measles cases were as follows:

Victoria
Sporadic, isolated cases with a significant outbreak in the NE Shire of Benalla (39 cases notified from late August to the end of October). Measles has been a notifiable disease in Victoria since May 1990.

South Australia
13 cases reported for the first half of 1990. The following 18 weeks have seen 11 cases, of which 5 were from the same semi-rural area. Full laboratory confirmation is not yet available for the latter reports.

Queensland
The number of cases reported so far this year is unremarkable. Fewer cases have been reported to the end of September 1990 (24) than for similar periods in the previous two years (1988:55, 1989:53). However, in the last 2 weeks there have been 5 reports received, the significance of which is not yet known.

Australian Capital Territory
To date there have been 23 cases reported for 1990, the majority (17) being reported since July. Measles is not notifiable in the ACT.

Western Australia
Only sporadic cases have been reported so far this year. However, there have been 2 recent outbreaks of measles-like illness, the aetiology of which awaits laboratory confirmation.

Tasmania
Sporadic cases only, with 7 reports being received for 1990 up until 4 October. Measles became notifiable in September 1989.

Northern Territory
No cases have been reported in the last 6 months. Measles has been a notifiable disease since March 1990.

NON-VIRAL PATHOGEN REPORTS

9 positive blood cultures have been received so far for October from Toowoomba Base Hospital. The following organisms were isolated:-

Escherichia coli from 2 females aged 64 and 71 years with pneumonia;

Escherichia coli and Candida albicans from a 58-year-old female with multiorgan failure.

Methicillin resistant Straphyloccocus aureus (MRSA) from a 53-year-old female.

Neisseria meningitidis from a 1-year-old male infant who also had Salmonella Group C1 isolated from faeces.

Straphylococcus aureus from 2 male patients aged 36 and 58 years.

Streptococcus pneumoniae from a male infant aged 2 months.

Streptococcus sanguis from a 24-year-old female with endocarditis.

A report of Toxoplasma gondii identified in the CSF of a 22 week old fetus with hydrocephalus was received from the Virology Department at Fairfield Hospital. The mother tested positive for Igm antibodies to Toxoplasma gondii

For the period 7 - 13 October, 10 reports of malaria (origin unspecified) were received from the Queensland Department of Health.

OVERSEAS BRIEFS

1. CHOLERA IN GUAM

A single case of cholera was reported for the week beginning 19 August 1990.

2. DENGUE IN THE MALDIVES

CDI has recently received a report of a case of dengue 2 in a 30-year-old female who had recently spent 2 weeks in the Maldives. As far as can be determined from the report the patient had not visited any other dengue endemic or epidemic areas.

REPORTING OF NOTIFIABLE DISEASES SURVEILLANCE DATA.

There was a very good response to the article on the "Reporting of Notifiable Diseases Surveillance Data" **(CDI 90/20)**. This paper proposed a graphical format for the presentation of notification data, which will appear as a regular feature in future editions of CDI. In addition, many constructive criticisms were received.

Some readers considered that more specific information should be presented than that shown in the sample graphs. It was suggested that data on individual arboviruses could be shown in the figure. Considering the arboviruses, there are two main reasons for presenting these as a single group. Firstly, the graph is only intended to display the overall trends in notification incidence and to supplement the data presented notifiable diseases tables. It is meant to be a quick and easy to read reference.

Secondly, some States and Territories collect notification data on specific arboviral diseases, such as Epidemic Polyarthritis, whereas others assemble arboviral notifications as a single group. The graph aims to present national notifiable diseases data and this is only possible if arboviral data are presented as a single group.

Another reader suggested that the graph would be more meaningful if the horizontal axis (report number) was a log scale. If such a scale were used, it was argued that small but important increases in uncommon conditions, such as meningococcal disease, would not be overpowered by large increases in diseases such as hepatitis B. In response to this argument, the aim of the graph is not so much to compare the notification incidences of different diseases as to compare the current notification incidence, of a particular disease, with its historical average incidence. It was decided that a log scale would make the graph unnecessarily complicated.

It was also considered that the notifiable diseases reports should be expressed as "identification of pathogens" rather than "identification of diseases". Certainly, the diagnostic basis for most communicable diseases is the identification of a specific pathogen. However, notifiable diseases reports represent numbers of patients, diagnosed on the basis of both clinical and microbiological findings. As such, they complement data received from laboratories which identify and report specific pathogens (the CDI pathogen report).

A notifiable diseases surveillance system is characterised by the potential for double reporting of patients and for reporting of chronic cases of notifiable diseases (such as hepatitis B). It was suggested that hepatitis B notifications should clearly indicate those cases which represent acute infections. However, if notifiable diseases are reported in a consistent manner, then trends of notification incidence will reflect trends in the incidence of acute cases.

Finally, to improve the overall presentation of the graph, it is planned to increase the contrast in the shading of the "past" and "present" bars in the figure.

EPIDEMIC POLYARTHRITIS, EASTERN AUSTRALIA, JAN - SEPT 1990.

During the period January-September 1990, data on 163 serologically confirmed cases of epidemic polyarthritis (EPA) were provided by the Virology Unit, Department of Infectious Diseases, Institute of Clinical Pathology and Medical Research (ICPMR), Westmead Hospital, Westmead, NSW. The Virology Unit acts as a diagnostic and collating centre for EPA data which is provided by 5 laboratories throughout New South Wales and Queensland. The data are summarised in table 1 below. The sex and age distribution of cases is shown in table 2. Data for 20 cases are not included as the age was not provided for these patients.

Table 1. Serological diagnosis of epidemic polyarthritis, Jan-Sept 1990.

Month of Specimen Collection	No of cases			Probable State of acquisition				
	M	F	Total	NSW	QLD	VIC	TAS	ACT
January	4	3	7	6			1	
February	9	10	19	15	2		2	
March	9	8	17	11	1	1	3	1
April	26	27	53	50	2	1		
May	31	19	50	47	3			
June	3	3	6	4	1	1		
July	1	2	3	3				
August	3	4	7	7				
September		1	1	1				
TOTAL	86	77	163	144	9	3	6	1

Table 2. Sex and age distribution of epidemic polyarthritis cases.

Age range	Male	Female	Total
0-9		1	1
10-19	2	1	3
20-29	13	8	21
30-39	22	23	45
40-49	15	20	35
50-59	9	9	18
60-69	10	3	13
70-79	3	1	4
80-89	2	1	3
TOTAL	76	67	143

As expected from the geographical location of the reporting laboratory, the majority of cases were reported from New South Wales. The peak months were April and May, with a total of 97 cases (67% of the total reported for that State).

Overall, the male:female case ratio was almost exactly 1:1. This is in agreement with the findings of Fraser (1986) in a study of clinical cases and also corresponds with the findings of a serological survey reported by Fraser et al (1986). In other studies the disease has been reported to have a higher incidence in women. A male/female ratio of 1:1.23 has been reported from northern Queensland, 1:1.79 from central Queensland (Aaskov et al, 1981a), 1:1.7 from Fiji (Aaskov et al, 1981b), 1:1.2 from south-eastern Australia (Hawkes et al 1985) and 1:1.6 in Australia overall (Mudge and Aaskov 1983).

Of particular note is that six cases in the present series were reported from Tasmania. There is only one other published report of EPA cases from this state (Mudge et al 1981). These authors reported two serologically confirmed cases in males (27 year old, 54 year old) who had not left Tasmania in recent months. The 6 cases confirmed in the present report indicate that EPA may be more prevalent in Tasmania than is generally recognised.

McManus and Russell (1990) in a brief report have also provided evidence of EPA activity in Tasmania. These authors note serological evidence of Ross River virus activity in horses and native fauna, and isolation of the virus from *Aedes flavifrons* McManus and Russell (1990, personal communication) also report that 21 confirmed cases of EPA were recorded in 1982, with a further 66 cases in 1989. In both instances, the areas of Tasmania affected were mainly the northern and eastern coastal plains.

The age of patients ranged from 8 to 84 with a peak in the 30-49 year age group. Although symptomatic disease is thought to be uncommon in children, one case was an 8 year old year girl. The only clinical symptoms in this patient were lassitude and joint aches. As Fraser (1986) has noted, the disease is milder and briefer in children, and this author has reported that two cases came to his notice primarily through changes in temperament, clumsiness and self-restricted physical activity. Fraser (1989) also notes that although the disease appears less frequent in children, underdiagnosis cannot be excluded.

Details of clinical symptoms were provided for 132 of the 163 patients in the present series. The data are summarised in Table 3. Specific symptoms provided by medical practitioners have been consolidated under general headings. For example, 'lethargy' and 'lassitude' have been grouped under the heading 'lethargy' and 'polyarthritis' and 'arthritis' have been grouped together.

Table 3. Frequency of symptoms reported by patients with epidemic polyarthritis.

Symptom	No of reports			Percent of total cases
	M	F	Total	
Arthralgia	36	31	67	50.8
Rash	18	20	38	28.8
Arthritis	16	19	35	26.5
Fever	19	7	26	19.7
Lethargy	13	7	20	15.2
Myalgia	10	5	15	11.4
Headache	5	3	8	6.1
Malaise	6	0	6	4.5
Lymphadenopathy	5	1	6	4.5
Stiff joints	4	2	6	4.5
Aches and pains	1	5	6	4.5

Other symptoms were reported with lesser frequencies, and included sore throat and anorexia (two patients each), and night sweats and giddiness, (one patient each).

The most common symptom by far was arthralgia (51% of patients). This, together with the arthritic manifestations seen in about 26% of patients, gave rise to the name of the disease, although, as pointed out by Fraser (1986), the term epidemic polyarthritis ignores cases with other symptoms alone. In the present series, there was no evidence of joint involvement in 24 of the 132 patients for whom symptoms were given.

Rash was seen in about 29% of patients, and fever in about 20%. In light of the observation that fever is often absent Fraser (1986) has observed that the term 'Ross River virus fever' is misleading on clinical grounds.

Mudge and Aaskov (1983), and Hawkes et al (1985) have also examined the frequency of various symptoms in patients with EPA. The former study was based on data from EPA cases which occurred in 1980/81 and the latter was a report of an EPA epidemic in New South Wales during the summer of 1983/84. Although joint involvement was the most frequently reported symptom in both studies, as in the present series, findings in other respects differ. For example, myalgia and headache were reported frequently in the earlier studies, in contrast to the present series. On the other hand no patient reported lethargy in the Mudge and Aaskov study, while 15% of patients in the present study and about 63% in the Hawkes et al study presented with this symptom.

The differences noted in the frequencies of these and other symptoms may be due to the methods of data collection. For example the data presented by Mudge and Aaskov (1983) were obtained largely through a patient administered questionnaire, while the Hawkes et al (1985) and the present data were provided by the treating medical practitioner. Self reporting by patients is more likely to be inaccurate or incomplete than reporting by medical practitioners. This is particularly so in view of the multiple symptoms such as arthralgia, arthritis and myalgia which occur in some patients which may be misinterpreted by lay persons.

In addition the patient may report only the most troublesome symptoms and neglect the less severe.

A second, less likely possibility is that the frequency of various symptoms between groups of patients is inherently variable. Fraser (1989) has stated that 'the literature and personal experience also suggest regional and local temporal variations in clinical manifestations; for example frequency and features of rash, lymphadenopathy, severity, duration'. The present study lends some support to this belief;- in particular, temporal variations would appear to be evident by comparing Hawkes et al (1985) with the Westmead data, as both are predominantly from New South Wales.

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Acknowledgements

CDI is indebted to the following for provision of the clinical and laboratory data used in this report:-

1. Ms L Hueston and Associate Professor AL Cunningham, Virology Unit, Department of Infectious Diseases and Microbiology, ICPMR, Westmead Hospital, Westmead, NSW.

2. Dr M Cloonan and Mr I Carter, Virology Department, Prince Henry Hospital, Little Bay, NSW.

3. Mr P Handsaker, Serology Department, Hendry and Hampson Pathology, Newcastle, NSW.

4. Serology Department, Sullivan and Nicholaides Pathology, Queensland.

5. Dr CH Mansfield, Mansfield Pathology, Paddington, NSW.

6. Dr RC Russell, Medical Entomology Unit, Department of Infectious Diseases and Microbiology, ICPMR, Westmead Hospital, Westmead, NSW.

7. Mr TJ McManus, Department of Primary Industry, Tasmania.

Editorial Comment

Copies of the 'Clinical Handbook on Epidemic Polyarthritis' and the associated patient information sheet, published in late 1989 (see **CDI 90/1**) are available from state health authorities.
Overseas readers may obtain a copy by writing directly to:

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FLAVIVIRUS SURVEILLANCE IN VICTORIA ("Sentinel Chicken Program")

(Contributed by J Aldred, Department of Agriculture and Rural Affairs, Veterinary Research Institute, Victoria)

The Murray and Goulburn River Valleys in northern Victoria are the areas of the state considered most vulnerable to an outbreak of Australian arbo-encephalitis (AAE) and sites throughout these areas are monitored each year as part of the Victorian Arbovirus Disease Control Program (VADCP).

The "Sentinel Chicken Program" is conducted by officers of the Department of Agricultural and Rural Affairs" (DARA), based at the Veterinary Research Institute, Attwood, and in the country regions where the chicken flocks are located. The program is fully funded by the Health Department, Victoria (HDV).

The sentinel system is designed to provide the HDV with an early warning of the presence of arboviruses known to cause severe clinical disease. Chickens are the logical choice of species for use as sentinels for the presence of the two flaviviruses which can cause AAE (Murray Valley Encephalitis virus and Kunjin virus). The most important factors in favour of chickens are the early development of a strong antibody response following infection with modest doses of these these viruses, and their attractiveness to vector mosquitoes as a blood source. They are also inexpensive to purchase, easy to handle and bleed on a regular basis, and it is readily possible to maintain the required number of birds in locations close to vector mosquito breeding habitats.

They do not, however, act as sentinels for Ross River virus as this virus does not elicit a reliably detected seroconversion in chickens.

Chickens, in flocks of 20 birds each, are housed in pens close to habitat which is known to support the breeding of large numbers of vector mosquitoes during flood periods. The 10 sites chosen for location of the flocks (Figure 1.) are spaced throughout northern Victoria near the areas where clinical cases occurred during the last epidemic of AAE in 1974.

Figure 1. Location of sentinel chicken flocks in Victoria, 1990-91

Chickens are bled weekly through November to April, the period of highest mosquito activity in northern Victoria. Blood samples are taken by DARA regional staff, or by private land owners in some instances, and forwarded to the laboratory at VRI Attwood where each sample is tested by ELISA for antibody to MVE and Kunjin (KUN) viruses.

Each week, the results are forwarded to the Health Department, Victoria, and from here they are faxed to the Commonwealth Department of Community Services and Health (Canberra), the state Health Departments in NSW and SA, members of the Victorian Arbovirus Task Force (the multi-departmental group responsible for managing the VADCP) and to the collaborating laboratories at Westmead Hospital (NSW) and the University of NSW.

MVE virus has not been detected in the Murray-Goulburn Valleys since 1974. However, KUN virus has been detected on several occasions since this date, with evidence of seroconversion in sentinel chickens, detection of antibody in a domestic pig (6 months old), isolation of the virus from certain mosquitoes and isolation of the virus from the spinal cord of a horse suffering encephalitis (Table 1). There have also been two human cases of clinical encephalitis attributed to infection with KUN virus since 1974. Both occurred in the autumn of 1984, one case was at Merbein (near Mildura), the other at Toolamba .

TABLE 1 Evidence of the presence of Kunjin virus in the Murray Goulburn Valley

SENTINEL CHICKENS

Location	No. Positive	Date
Swan Hill	4	22/2/82
Mildura	5	1/3/82
Robinvale	2	5/3/82
Rutherglen	1	1/3/82
Barmah	1	15/3/82
Swan Hill	1	28/2/84
Kerang	2	28/2/84
Mildura	1	28/2/84
Tooleybuc	1	5/2/90

DOMESTIC PIG

Location	No. Positive	Date
Tallygaroopna (near Shepparton)	1	7/2/89

ISOLATION OF KUNJIN VIRUS

Location	Source	Date
Swan Hill	Sentinel Chicken Blood	2/2/82
Swan Hill	Sentinel Chicken Blood	26/2/82
Swan Hill	Culex annulirostris	22/2/82
Kerang	Culex annulirostris	28/12/83
Swan Hill	Horse spinal cord	5/4/84

HUMAN INFECTION

Location	Date
Merbein	6/4/84
Toolamba	22/3/84

A RECENT OUTBREAK OF DENGUE FEVER IN NORTH QUEENSLAND

(Contributed by D Phillips, State Health Laboratories Queensland: J Aaskov, Department of Medical Laboratory Science, Queensland University of Technology)

A small outbreak of dengue fever began on the 8 May this year in Townsville (North Eastern Queensland). On the 22 May the first patient from Cairns was diagnosed and the first cases from Thursday Island on 2 July.

Twenty-six patients have been diagnosed as having infections with dengue one on the basis of virus isolation or detection of homotypic IgM, or a combination of these procedures. Dengue one was isolated from the acute phase sera of ten of these patients.

Serum from three patients contained IgM which reacted with a range of flaviviruses (ie more than one dengue serotype). Serum from a further two patients contained IgM antibody which reacted only with dengue two. Both patients had not travelled outside Australia for at least six months prior to onset of symptoms.

Fourteen patients were male and seventeen female (median age 36 years). All but one (a male of 8 years) were old enough to have been exposed to at least one of the previous dengue outbreaks in North Queensland (1980-1981, dengue one; 1953-1955, dengue one; 1942-1944 mainly dengue two).

Cairns is the centre of the region where this dengue outbreak occurred. It has an international airport which is regularly serviced by flights from South-East Asia and the Pacific where there has been an extensive outbreak of dengue one and dengue three infection.

These are probably the first clinical dengue infections due to indigenous dengue transmission since the outbreak in the same region in 1980-1981.

CDI Editorial Comment

Total dengue fever reports to the end of the current reporting period (both indigenous and exotic cases) are considerably greater than all the annual totals for the period since the 1981-82 outbreak in Queensland (see table next page).

Year	Dengue cases reported
1980	13
81	98
82	276
83	14
84	21
85	21
86	10
87	10
88	11
89	39
90 (part)	60

Disregarding the recent indigenous reports (26) from Queensland, there appears to have been a significant increase in the number of cases of imported dengue, into Australia, in the last two years.

ARBOVIRAL SURVEILLANCE - UNITED STATES 1990

(Based on MMWR 1990;39[35]:593-598)

Through to 27 August 1990, surveillance of mosquito vectors of St. Louis encephalitis (SLE) and eastern equine encephalitis (EEE) has detected unusually early and high levels of viral transmission in several states, indicating a potential risk for epidemic transmission. This report summarises arboviral surveillance activities in Texas, Florida, Massachusetts, New Jersey, and New York. In addition, the report summarises cases of confirmed or possible arboviral infections in persons in Texas, Florida, North Carolina, and equine cases in Georgia and Maryland.

St. Louis Encephalitis

Texas In the city of Houston and in Harris County, the number and distribution of and SLE viral infection rates for *Culex quinquefasciatus* mosquitoes are monitored throughout the year. During the summer transmission season, >300 mosquito pools, from various sampling points in the county, are tested for SLE virus. This mosquito surveillance is coupled with programs of routine mosquito control and emergency measures directed at areas where viral transmission is detected.

On 19 June (approximately 1 month earlier than in previous epidemic years), SLE virus was recovered from collections in a north-eastern Houston neighbourhood. In succeeding weeks, >239 suspected SLE viral isolates were recovered from widely separated areas of the county and city, particularly in the Denver Harbour, Houston Heights, and Fifth Ward sections of central Houston. Six isolates were recovered from Baytown, the site of an SLE outbreak in 1986 (1). In central areas of Houston, minimum infection rates (MIRs) in *Cx. quinquefasciatus* have averaged 5 per 1000 mosquitoes (2). however, in an intensively studied square-mile area in the north-eastern quadrant of the city, MIRs in this species were as high as 25 per 1000 mosquitoes captured in gravid traps between 3 August and 10 August. Surveillance of wild birds in this site indicated a point seroprevalence of 60%.

After >40 SLE viral isolates had been identified in mosquitoes, the potential for an outbreak was publicised in a series of news conferences and announcements in early July. Active surveillance by telephone and mail was instituted to identify patients with central nervous system (CNS) infection in all county hospitals. Two cases were serologically diagnosed by the Houston City Health Department and confirmed by the Centers for Disease Control, Atlanta (CDC). The cases were in a young woman from north-eastern Houston and an elderly woman from Baytown; dates of onset of illness were 20 July and 21 July, respectively. Both patients died; however, the causes of death have not been fully determined.

Mosquito-control measures have been intensified at sites where viral isolates were recovered. In areas of the city where *Cx. quinquefasciatus* use storm sewers as resting sites, pyrethrins applied as thermal fogs into the sewers are the principal means of control.

Florida Florida maintains a program of SLE and EEE surveillance by monitoring seroconversions in sentinel chickens in 14 counties. In early June, seroconversions to SLE virus were noted in flocks in several central and eastern Florida counties. In July and August, increasing seroconversion rates were noted, including 100% of chickens in Indian River County and 22%-33% of chickens in Lee, Manatee, and Orange county flocks. Surveillance was then intensified, and weekly blood samples of flocks in these counties detected rising seroconversion rates: for example, in Lee and Orange counties, 50% and 80% of chickens, respectively, seroconverted during the week of 13 August. Hospital-based surveillance for encephalitis cases was initiated in the affected counties. After seroconversions to SLE virus were noted in early July, ground-based larviciding and ultra low volume (ULV) adulticiding were intensified. Five confirmed cases and one presumptive case subsequently were identified in encephalitis patients from Indian River, Lake, and Highlands counties. The dates of onset of illness in these cases ranged from 28 July to 17 August.

Eastern Equine Encephalitis

Massachusetts Since 1957, adult mosquitoes in freshwater swamps of central south-eastern Massachusetts (excluding Cape Cod, Martha's Vineyard, and Nantucket) have been monitored in a standardised surveillance program for EEE. In 1990, EEE virus was recovered earlier and in greater numbers than at any time previously in these areas.

In early June, mosquito surveillance that used unbaited miniature light traps was initiated in Bristol, Plymouth, and other counties. The first EEE viral isolate was from a known enzootic site in south-eastern Massachusetts and was obtained from a collection on June 20, nearly 1 month earlier than in previous years. The virus was recovered from a pool of *Culiseta melanura* mosquitoes, the species selectively favoured by trap design and placement, and the species believed to be the primary enzootic vector of viral

transmission and amplification in Massachusetts. Isolations of EEE have increased progressively during the summer and, in collections through to 8 August, 597 pools of *Cs. melanura* (24,836 mosquitoes tested in pools of <50) have yielded 49 EEE isolates, representing a crude MIR of approximately 2 per 1000. One site has yielded 27 isolates from 5080 mosquitoes, an MIR of more than 5 per 1000. In addition, one EEE isolate was recovered from 176 pools (9484 mosquitoes) of *Coquillitidea perturbans*, an epizootic vector that transmits the virus from the enzootic cycle to horses and humans. No isolates have been recovered from *Aedes vexans* or *Ae. canadensis*, probably the most important epizootic vectors in Massachusetts; however, the populations of these species do not usually peak until later in the season, and the risk of epizootic transmission may rise as these vector species increase in number.

The risk of EEE viral transmission in south-eastern Massachusetts in 1990 was anticipated from observations of rainfall patterns and the relative scarcity of EEE virus during the 5 preceding years. Historically, EEE activity has occurred in the second of two consecutive seasons of excessive rainfall as happened in 1989 and 1990. Preseason warnings in April 1990 to local mosquito-control districts and health departments were followed by public warnings in July, when EEE viral isolations in *Cs. melanura* exceeded the historical warning threshold of 1 per 1000 mosquitoes. During August 18 equine deaths, clinically compatible with EEE, were reported; EEE virus was recovered from the only well-preserved brain submitted, and two of five horses tested serologically were positive. In early August, a contingency plan was initiated for wide-scale aerial ULV application of maldison ('malathion') over Bristol and Plymouth counties. Shortly after the decision to schedule the ULV application for 27-29 August, serological tests of a comatose 7-year-old Plymouth County resident indicated that he had EEE. His onset of fever was 16 August and EEE antibody titres on specimens from 23 and 27 August were <10 and >40, respectively.

New York Surveillance of mosquito vectors and avian hosts of EEE virus is conducted in four counties near Syracuse (Madison, Oneida, Onondaga, and Oswego). Since 1971, outbreaks in these counties have resulted in two human deaths and dozens of equine fatalities. As of August 24, arboviral isolation attempts were completed on 1477 pools (110,900 adult female mosquitoes) collected from 23 May to 16 August. EEE virus was detected in 17 pools of *Cs. melanura*, two pools of *Cs. morsitans*, and two pools of *Ae. canadensis* mosquitoes captured in Oswego County from 23 July to 16 August. In addition, EEE virus was recovered from two pools of *Cs. melanura* collected in Madison County and Onondaga County from 30 July to 16 August; this species is the primary enzootic mosquito vector of EEE among wild avians in this region.

A total of 627 samples (brain, cerebrospinal fluid, and blood clots) from vertebrates in five upstate counties were also tested for virus. None of five equine samples from Cayuga, Otsego, and Monroe counties or 79 avian specimens from Onondaga County yielded an isolate. However, 15 of 343 wild avians (36 species) captured in Oswego County were viraemic.

EEE virus was recovered from blood clots in 15 of 384 passerine birds sampled from 16 July to 7 August. EEE was confirmed in two unvaccinated horses from Oswego County by isolation of the virus from brain tissue. Onset of clinical signs of CNS infection were noted on 15 August and 19 August, respectively.

Other than the enzootic focus near Syracuse, serological surveillance has not detected evidence of EEE transmission in Cayuga or other counties. In June, 17 (18%) of 96 wild avians in Oswego County had low-titred HI antibody (1:20-1:80) to EEE virus, indicating previous infection. In contrast, 2 weeks after the first viraemic birds were detected, 28 (39%) of 72 avians blood sampled between 29 July and 1 August were seropositive for EEE virus, and 18 (64%) of these exhibited HI titres >1:160, suggesting a recent infection.

Because this high level of viral activity indicated a potential for epidemic/epizootic transmission, on 2-3 August and 3 August respectively, the Cicero Swamp in northern Onondaga County and Toad Harbour Swamp in southern Oswego County were treated with aerial applications of an insecticide to reduce vector populations. Insecticide applications were repeated in late August in response to viral isolations from mosquitoes captured inside and near previously treated areas.

New Jersey. At coastal and inland locations, EEE viral transmission is surveyed through collections of *Cs. melanura* and epizootic vectors, including *Cq. perturbans* and *Ae. sollicitans*. In early August, a dramatic surge in abundance of *Cs. melanura* was observed in all sites; however, this surge is typical of the seasonal dynamics of this mosquito in New Jersey. Since July, EEE viral isolates have been made in all monitoring sites; MIRs have ranged from 3.8 to 7.8 per 1000 *Cs. melanura*. These rates are high for July, for the areas under surveillance, and indicate a risk for epizootic transmission. However, one presumptive equine case has been the only evidence of epizootic transmission thus far.

The risk of epizootic transmission is also surveyed by monitoring the age structure of *Ae. sollicitans*, the principal epizootic vector in coastal areas. When landing collections exceed 10 parous females per minute, indicating a relative abundance of mosquitoes that have previously fed a number of times, and therefore possibly on viraemic birds, adulticiding is intensified. This ongoing program of mosquito control aims to maintain young populations of *Ae. sollicitans*, which lowers the risk of epizootic transmission from this species.

Other States. In July, a fatal case of EEE with onset of illness on June 1 was reported in a woman from South Carolina; three equine cases were also reported from this state. From July 13 to August 2, three equine cases of EEE were reported from coastal countries of North Carolina, and a presumptive human case of EEE with onset of illness on August 1 was reported from Orange Country, an inland area where EEE rarely occurs. In April and June, three equine cases were confirmed from south-eastern Georgia; in July, two equine cases were reported from Maryland's eastern shore.

MMWR Editorial Note: SLE is the leading cause of epidemic viral encephalitis in the United States (3-5). Large outbreaks have occurred periodically in areas of the Gulf Coast and the Mississippi and Ohio valleys. The last major outbreak (in 1975) resulted in nearly 3000 reported cases. In response to that outbreak, many state and local health and mosquito-control agencies established programs of avian and mosquito surveillance to monitor SLE viral transmission in its natural cycle and conditions favouring epidemic transmission.

Because of the rare occurrence of outbreaks, rigorous evaluation of the sensitivity, specificity, and cost-benefit of avian and mosquito surveillance has been difficult.

However, on a number of occasions, human cases have been preceded by a high prevalence of vector mosquitoes, rising infection rates in vectors, and increasing seroprevalence in wild or sentinel avians (6,7).

SLE viral activity in Houston-Harris Country during 1990 has been unusual because the first viral isolates were discovered remarkably early and because of the widespread distribution of viral isolates in the country. The highest infection rates, however, remain within the city's central area, consistent with a previously observed gradient of more intense viral activity at the city's centre (8). The markedly elevated MIR in north-eastern Houston suggests that a risk for epidemic transmission exists in this area. Because enzootic SLE virus transmission in Houston usually does not wane until early October, the risk for human disease will potentially continue or rise throughout this period.

In Florida, the importance of sentinel flock seroconversions as an indicator of epidemic risk has been ambiguous. From 1982 to 1986, up to 20% of sentinel chickens seroconverted each year even though no human cases occurred. However, during that period, October was the peak month of viral transmission to chickens. In 1990, serconversion rates have been remarkably higher and have occurred 2 months earlier than usual (7). These findings suggest that viral transmission in the enzootic cycle could build higher than usual levels in Florida during the fall months, with a resultant increase in risk of transmission to humans.

EEE is a rare disease: in most years, fewer than five cases are reported nation-wide. The magnitude of EEE outbreaks generally is small; however, during epidemic years, the 30% case-fatality rate associated with the illness underscores the severity of this public health problem (9-11). EEE cases are usually sporadic; viral transmission is localised to specific and relatively constant enzootic foci, related to freshwater swampy habitats that support breeding of *Cs. melanura*, the principal enzootic vector of EEE virus (12). South-eastern Massachusetts, the four-county area of New York state described in this report, and coastal locations in New Jersey and mid-Atlantic and south-eastern states have long been recognised as areas of enzootic EEE. In these locations, individual mosquito species vary in their importance as epizootic vectors for equine and human transmission.

The physical, biologic, and ecological factors associated with epizootic transmission are complex, but the abundance of EEE virus circulating in the enzootic cycle and various characteristics of the epizootic vectors are important determinants of risk. During 1990, the early appearance of EEE virus in *Cs. melanura* in Massachusetts and New York, as well as the recovery of numerous viral isolates, has indicated a potential for epizootic transmission and triggered intensified programs of mosquito control and public warnings. In New Jersey, an ongoing program of adulticiding is linked to the maturity of *Ae. sollicitans* populations, and emergency measures have not been thought necessary (13).

This report illustrates the use of surveillance data on arboviral transmission patterns in nature to guide public health interventions before human infections occur. The approach to surveillance of arboviral diseases is unique in this respect, as are the opportunities to prevent human illness by monitoring and controlling vector mosquitoes. Additional correlations of ecological and epidemiological data are needed to assess the predictive value of these indices in forecasting arboviral epidemics.

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AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 11/10/90 TO 24/10/90

CODE 018 - MICROBIOLOGICAL DIAGNOSTIC UNIT, UNIVERSITY OF MELBOURNE (VIC)
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 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)
CODE 400 - DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON (QLD)
CODE LDS - DIAGNOSTIC SERVICES LTD, LAUNCESTON (TAS)
CODE RHH - ROYAL HOBART HOSPITAL (TAS)
CODE TPL - TOOWOOMBA PATHOLOGY LABORATORY (QLD)

	018	019	065	066	110	111	112	114	115	116	400	LDS	RHH	TPL	TOTAL
0100 ADENOVIRUS NOT TYPED	0	0	5	10	0	5	1	0	12	0	0	0	0	0	33
0101 ADENOVIRUS TYPE 1	0	0	0	0	0	8	3	0	0	0	0	0	0	0	11
0102 ADENOVIRUS TYPE 2	0	0	0	0	0	12	0	0	0	0	0	0	0	0	12
0103 ADENOVIRUS TYPE 3	0	1	0	0	0	3	1	0	0	0	0	0	0	0	5
0104 ADENOVIRUS TYPE 4	0	0	0	0	0	0	1	0	0	0	0	0	1	0	2
0105 ADENOVIRUS TYPE 5	0	0	0	0	0	4	0	0	0	0	0	0	0	0	4
0106 ADENOVIRUS TYPE 6	0	1	0	0	0	1	0	0	0	0	0	0	0	0	2
0108 ADENOVIRUS TYPE 8	0	2	0	0	0	0	0	0	0	0	0	0	0	0	2
0199 ADENOVIRUS TYPING PENDING	0	0	0	0	0	8	0	0	0	0	0	0	0	0	8
0201 INFLUENZA A VIRUS	0	0	9	0	1	7	2	3	3	2	4	0	0	0	31
0202 INFLUENZA A VIRUS SUBTYPE H3N2	0	1	0	0	0	4	0	0	0	0	0	0	0	0	5
0301 PARAINFLUENZA VIRUS TYPE 1	0	0	0	0	0	7	0	0	0	0	0	0	0	0	7
0302 PARAINFLUENZA VIRUS TYPE 2	0	1	0	0	0	1	0	0	0	0	0	0	0	0	2
0303 PARAINFLUENZA VIRUS TYPE 3	0	0	1	0	0	5	3	0	0	0	0	0	0	0	9
0399 PARAINFLUENZA VIRUS TYPING PEN	0	2	1	0	0	4	0	0	0	0	0	0	0	0	7
0400 RESPIRATORY SYNCYTIAL VIRUS (R	0	7	4	15	22	27	3	0	5	1	0	0	14	1	99
0500 RHINOVIRUS (ALL TYPES)	0	7	3	0	0	16	1	1	0	0	0	0	0	0	28
0600 MYCOPLASMA PNEUMONIAE	0	0	7	0	3	19	1	0	0	0	1	0	0	0	31
0700 ORNITHOSIS-PSITTACOSIS	0	4	0	0	0	0	0	0	0	0	0	0	0	0	4
0809 COXSACKIEVIRUS A9	0	0	0	0	0	0	1	1	0	0	0	0	0	0	2
0816 COXSACKIEVIRUS A16	0	2	0	0	0	0	0	0	0	0	0	0	0	0	2
0902 COXSACKIEVIRUS B2	0	0	0	0	0	0	1	0	0	1	0	0	0	0	2
0903 COXSACKIEVIRUS B3	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
0904 COXSACKIEVIRUS B4	0	6	0	0	0	0	0	0	0	0	0	0	0	0	6
0905 COXSACKIEVIRUS B5	0	0	0	0	0	0	1	0	0	0	0	0	0	0	1
1006 ECHOVIRUS TYPE 6	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
1011 ECHOVIRUS TYPE 11	0	4	0	0	0	3	0	0	0	0	0	0	0	0	7
1014 ECHOVIRUS TYPE 14	0	0	0	0	0	3	0	0	0	0	0	0	0	0	3
1018 ECHOVIRUS TYPE 18	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1
1033 ECHOVIRUS TYPE 33	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1
1100 POLIOVIRUS NOT TYPED	0	0	0	0	0	14	0	0	0	0	0	0	0	0	14
1101 POLIOVIRUS TYPE 1	0	3	0	0	0	0	0	0	0	0	0	0	0	0	3
1102 POLIOVIRUS TYPE 2	0	0	0	0	0	0	1	1	0	0	0	0	0	0	2
1103 POLIOVIRUS TYPE 3	0	1	0	0	0	0	1	0	0	0	0	0	0	0	2
1200 MUMPS VIRUS	0	0	1	0	0	0	1	0	0	0	0	0	0	0	2
1300 HERPES VIRUS GROUP - NOT TYPED	0	1	2	0	0	0	0	0	0	10	0	0	0	0	13
1301 HERPES SIMPLEX VIRUS - NOT TYP	0	2	1	3	1	0	28	2	1	0	0	1	0	0	39
1302 EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	14	0	13	0	0	5	1	0	27	0	0	0	60
1303 VARICELLA-ZOSTER VIRUS	0	8	10	0	2	0	1	2	2	0	0	0	0	0	25
1306 HERPES SIMPLEX TYPE 1	0	43	70	0	21	0	2	1	18	0	0	0	0	1	156
1307 HERPES SIMPLEX TYPE 2	0	35	147	1	29	0	12	0	24	0	0	3	0	0	251
1399 HERPES VIRUS TYPING PENDING	0	0	0	0	0	3	0	0	0	0	0	0	0	0	3
1401 COXIELLA BURNETII	0	1	2	0	0	0	1	0	3	0	1	0	0	0	8
1502 PICORNIA VIRUS - NOT TYPED = E	0	0	4	0	0	0	0	1	4	0	0	0	0	0	9
1514 MOLLUSCUM CONTAGIOSUM	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1
1521 MEASLES VIRUS	0	11	0	0	0	4	3	0	1	4	0	0	0	0	23
1522 RUBELLA VIRUS	0	3	0	0	1	0	0	1	0	0	3	0	0	0	8
1532 HEPATITIS B ANTIGEN	0	8	43	0	7	1	29	0	48	9	1	0	0	0	146
1535 HEPATITIS A ANTIBODY	0	0	5	0	2	0	0	0	0	0	0	0	0	0	7
1536 HEPATITIS C VIRUS	0	0	13	0	0	0	0	0	0	0	0	0	0	0	13
1537 HEPATITIS, DELTA	0	0	1	0	0	0	0	0	0	0	0	0	0	0	1
1541 CHLAMYDIA A - C. TRACHOMATIS	20	0	75	0	26	0	16	0	13	7	0	2	5	25	189
1543 CHLAMYDIA A - LGV TYPE	0	0	1	0	0	0	0	0	0	0	2	0	0	0	3
1556 CMV - CYTOMEGALOVIRUS	0	26	7	3	2	7	0	0	23	0	9	2	1	0	80
1564 ROTAVIRUS	0	4	1	2	77	71	36	5	0	0	48	4	13	31	292
1566 NORWALK AGENT	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1
1599 ENTEROVIRUS TYPING PENDING	0	0	0	0	0	10	0	0	0	0	0	0	0	0	10
9903 NON-A, NON-B HEPATITIS	0	0	0	0	0	0	0	0	0	0	3	0	0	0	3
9992 ROSS RIVER VIRUS	0	0	0	0	0	0	0	0	6	0	4	0	0	0	10
9995 DENGUE	0	0	1	0	0	0	0	0	2	0	0	0	0	0	3
9998 ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1
TOTAL	20	185	431	34	208	250	150	24	166	34	104	12	34	59	1710

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 11/10/90 TO 24/10/90

NSW: ICPMR; PHH/POW; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP.
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	1	5	12	15	0	0	0	33
0101	ADENOVIRUS TYPE 1	3	8	0	0	0	0	0	11
0102	ADENOVIRUS TYPE 2	0	12	0	0	0	0	0	12
0103	ADENOVIRUS TYPE 3	1	4	0	0	0	0	0	5
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	1	0	2
0105	ADENOVIRUS TYPE 5	0	4	0	0	0	0	0	4
0106	ADENOVIRUS TYPE 6	0	2	0	0	0	0	0	2
0108	ADENOVIRUS TYPE 8	0	2	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	8	0	0	0	0	0	8
0201	INFLUENZA A VIRUS	5	7	7	9	1	0	2	31
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	5	0	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	7	0	0	0	0	0	7
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	3	5	0	1	0	0	0	9
0399	PARAINFLUENZA VIRUS TYPING PEN	0	6	0	1	0	0	0	7
0400	RESPIRATORY SYNCYTIAL VIRUS (R	3	34	6	19	22	14	1	99
0500	RHINOVIRUS (ALL TYPES)	2	23	0	3	0	0	0	28
0600	MYCOPLASMA PNEUMONIAE	1	19	1	7	3	0	0	31
0700	ORNITHOSIS-PSITTACOSIS	0	4	0	0	0	0	0	4
0809	COXSACKIEVIRUS A9	2	0	0	0	0	0	0	2
0816	COXSACKIEVIRUS A16	0	2	0	0	0	0	0	2
0902	COXSACKIEVIRUS B2	1	0	0	0	0	0	1	2
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	6	0	0	0	0	0	6
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	1	0	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	0	7	0	0	0	0	0	7
1014	ECHOVIRUS TYPE 14	0	3	0	0	0	0	0	3
1018	ECHOVIRUS TYPE 18	1	0	0	0	0	0	0	1
1033	ECHOVIRUS TYPE 33	0	1	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	14	0	0	0	0	0	14
1101	POLIOVIRUS TYPE 1	0	3	0	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	2	0	0	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	1	1	0	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	1	0	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	2	0	0	10	13
1301	HERPES SIMPLEX VIRUS - NOT TYP	30	2	1	4	1	1	0	39
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	5	0	28	14	13	0	0	60
1303	VARICELLA-ZOSTER VIRUS	3	8	2	10	2	0	0	25
1306	HERPES SIMPLEX TYPE 1	3	43	19	70	21	0	0	156
1307	HERPES SIMPLEX TYPE 2	12	35	24	148	29	3	0	251
1399	HERPES VIRUS TYPING PENDING	0	3	0	0	0	0	0	3
1401	COXIELLA BURNETII	1	1	4	2	0	0	0	8
1502	PICORNIA VIRUS - NOT TYPED = E	1	0	4	4	0	0	0	9
1514	MOLLUSCUM CONTAGIOSUM	0	0	1	0	0	0	0	1
1521	MEASLES VIRUS	3	15	1	0	0	0	4	23
1522	RUBELLA VIRUS	1	3	3	0	1	0	0	8
1532	HEPATITIS B ANTIGEN	29	9	49	43	7	0	9	146
1535	HEPATITIS A ANTIBODY	0	0	0	5	2	0	0	7
1536	HEPATITIS C VIRUS	0	0	0	13	0	0	0	13
1537	HEPATITIS, DELTA	0	0	0	1	0	0	0	1
1541	CHLAMYDIA A - C. TRACHOMATIS	16	20	38	75	26	7	7	189
1543	CHLAMYDIA A - LGV TYPE	0	0	2	1	0	0	0	3
1556	CMV - CYTOMEGALOVIRUS	0	33	32	10	2	3	0	80
1564	ROTAVIRUS	41	75	79	3	77	17	0	292
1566	NORWALK AGENT	0	1	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	10	0	0	0	0	0	10
9903	NON-A, NON-B HEPATITIS	0	0	3	0	0	0	0	3
9992	ROSS RIVER VIRUS	0	0	10	0	0	0	0	10
9995	DENGUE	0	0	2	1	0	0	0	3
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	1	0	0	1
TOTAL		174	455	328	465	208	46	34	1710

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 11/10/90 TO 24/10/90

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	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	16	0	0	0	10	0	0	0	0	27
0101	ADENOVIRUS TYPE 1	2	5	0	1	0	0	0	0	0	0	8
0102	ADENOVIRUS TYPE 2	0	10	0	1	0	1	0	0	0	0	12
0103	ADENOVIRUS TYPE 3	2	2	0	0	0	0	0	0	0	0	4
0105	ADENOVIRUS TYPE 5	0	3	0	0	0	1	0	0	0	0	4
0106	ADENOVIRUS TYPE 6	0	2	0	0	0	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	0	4	0	0	0	0	0	0	0	0	4
0201	INFLUENZA A VIRUS	3	23	0	0	0	0	0	0	0	0	26
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	5	0	0	0	0	0	0	0	0	5
0301	PARAINFLUENZA VIRUS TYPE 1	0	7	0	0	0	0	0	0	0	0	7
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	1	7	0	0	0	0	0	0	0	0	8
0399	PARAINFLUENZA VIRUS TYPING PEN	1	5	0	0	0	0	0	0	0	0	6
0400	RESPIRATORY SYNCYTIAL VIRUS (R	3	94	0	0	0	0	0	0	0	0	97
0500	RHINOVIRUS (ALL TYPES)	1	20	0	0	0	0	0	0	0	0	21
0600	MYCOPLASMA PNEUMONIAE	1	21	1	1	0	0	0	0	0	2	26
0700	ORNITHOSIS-PSITTACOSIS	0	4	0	0	0	0	0	0	0	0	4
0809	COXSACKIEVIRUS A9	0	0	0	0	1	0	0	0	0	0	1
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	0	0	0	2	2
0902	COXSACKIEVIRUS B2	0	0	0	0	0	1	0	0	0	0	1
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	0	0	0	0	1
0904	COXSACKIEVIRUS B4	0	1	0	1	0	1	0	0	0	1	4
0905	COXSACKIEVIRUS B5	0	1	0	0	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	1	0	0	0	0	0	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	0	3	0	1	0	0	0	0	0	1	5
1014	ECHOVIRUS TYPE 14	0	1	0	1	0	0	0	0	0	0	2
1018	ECHOVIRUS TYPE 18	0	1	0	0	0	0	0	0	0	0	1
1033	ECHOVIRUS TYPE 33	0	0	0	1	0	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	3	0	1	4	0	0	0	0	0	8
1101	POLIOVIRUS TYPE 1	0	2	0	0	0	1	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	1	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	1	0	0	0	0	1	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	0	0	0	0	0	0	0	6	7
1301	HERPES SIMPLEX VIRUS - NOT TYP	10	3	0	0	0	1	0	0	0	14	28
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	16	5	0	0	0	0	2	0	0	0	23
1303	VARICELLA-ZOSTER VIRUS	0	1	0	0	0	0	0	0	0	21	22
1306	HERPES SIMPLEX TYPE 1	2	8	1	0	0	0	0	1	2	109	123
1307	HERPES SIMPLEX TYPE 2	1	0	0	0	2	0	0	0	0	147	150
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	3	3
1401	COXIELLA BURNETII	2	2	0	0	0	0	0	0	0	0	4
1502	PICORNIA VIRUS - NOT TYPED = E	1	3	0	1	0	3	0	0	0	0	8
1514	MOLLUSCUM CONTAGIOSUM	0	0	0	0	0	0	0	0	0	1	1
1521	MEASLES VIRUS	4	0	1	0	0	0	0	0	0	11	16
1522	RUBELLA VIRUS	0	1	0	0	0	0	0	0	0	2	3
1532	HEPATITIS B ANTIGEN	73	0	0	0	0	0	65	0	0	1	139
1535	HEPATITIS A ANTIBODY	1	0	0	0	0	0	6	0	0	0	7
1536	HEPATITIS C VIRUS	5	0	0	0	0	0	7	0	0	0	12
1537	HEPATITIS, DELTA	0	0	0	0	0	0	1	0	0	0	1
1541	CHLAMYDIA A - C. TRACHOMATIS	8	0	0	0	0	0	0	0	0	0	8
1543	CHLAMYDIA A - LGV TYPE	1	0	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	10	20	0	0	2	1	1	2	5	0	41
1564	ROTAVIRUS	17	1	0	0	0	271	0	0	0	0	289
1599	ENTEROVIRUS TYPING PENDING	0	5	0	0	0	0	0	0	0	2	7
9992	ROSS RIVER VIRUS	4	0	0	0	0	0	0	0	0	0	4
9995	DENGUE	1	0	0	0	0	0	0	0	0	1	2
TOTAL		177	294	3	9	9	293	82	3	7	324	1201

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 11/10/90 TO 24/10/90

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	4	0	0	0	0	0	1	1	0	0	6
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	0	3	0	0	3
0103	ADENOVIRUS TYPE 3	0	0	0	0	0	0	0	1	0	0	1
0104	ADENOVIRUS TYPE 4	2	0	0	0	0	0	0	0	0	0	2
0108	ADENOVIRUS TYPE 8	2	0	0	0	0	0	0	0	0	0	2
0199	ADENOVIRUS TYPING PENDING	1	0	0	0	0	0	2	1	0	0	4
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	1	1	3	0	5
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	0	1	0	1
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	0	0	1	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	1	1	0	2
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	5	0	2	7
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	1	0	1	0	3	0	5
0809	COXSACKIEVIRUS A9	0	0	0	0	0	0	1	0	0	0	1
0902	COXSACKIEVIRUS B2	0	0	0	0	0	0	0	0	1	0	1
0904	COXSACKIEVIRUS B4	0	0	0	0	0	0	0	2	0	0	2
1011	ECHOVIRUS TYPE 11	0	0	0	0	0	0	0	2	0	0	2
1014	ECHOVIRUS TYPE 14	0	0	0	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	1	2	2	1	0	6
1200	MUMPS VIRUS	0	1	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	6	0	0	0	0	0	0	0	0	6
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	9	0	0	0	0	0	0	2	0	11
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	14	6	1	0	1	7	8	0	37
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	0	3	0	3
1306	HERPES SIMPLEX TYPE 1	3	25	0	0	0	0	0	1	4	0	33
1307	HERPES SIMPLEX TYPE 2	0	100	0	0	0	0	0	0	1	0	101
1401	COXIELLA BURNETII	0	0	0	0	0	0	1	3	0	0	4
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	0	0	0	1	1
1521	MEASLES VIRUS	0	0	0	0	0	0	1	4	2	0	7
1522	RUBELLA VIRUS	0	0	0	1	0	2	0	0	2	0	5
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	7	0	7
1536	HEPATITIS C VIRUS	0	0	0	1	0	0	0	0	0	0	1
1541	CHLAMYDIA A - C. TRACHOMATIS	0	181	0	0	0	0	0	0	0	0	181
1543	CHLAMYDIA A - LGV TYPE	0	1	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	1	1	0	2	0	3	3	5	24	0	39
1564	ROTAVIRUS	0	0	0	0	0	0	0	0	3	0	3
1566	NORWALK AGENT	0	0	0	0	0	0	0	1	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	1	2	0	0	3
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	0	0	0	3	0	3
9992	ROSS RIVER VIRUS	0	0	0	0	5	0	0	1	0	0	6
9995	DENGUE	0	0	0	0	0	0	0	0	1	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	1	0	0	0	1
TOTAL		13	324	14	10	7	6	16	45	70	3	508