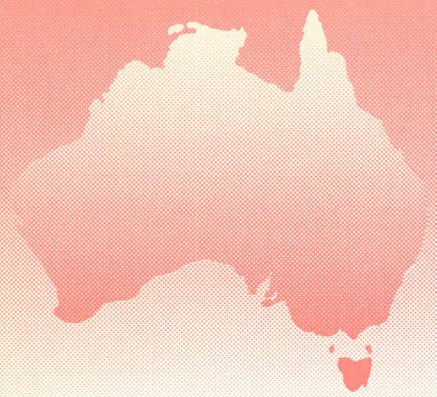

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COMMUNICABLE DISEASES NETWORK-AUSTRALIA
A National Network for Communicable Diseases Surveillance

DIAGNOSIS AND REPORTING OF ARBOVIRUS INFECTIONS IN AUSTRALIA

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At least five arboviruses are known to cause human disease in Australia; the flaviviruses, Murray Valley encephalitis[1,2], Kunjin[2,3], and serotypes of dengue viruses[4,5], and the alphaviruses, Ross River[6] and Barmah Forest viruses[7,8]. Occasional human disease has also been described following infections with Gan Gan (a bunyavirus)[9], Sindbis (an alphavirus)[10], and Kokobera (a flavivirus)[11] viruses. Associations of other arboviruses with human disease in Australia have not been confirmed.

The diagnosis of arboviral disease depends on recognition of an appropriate clinical pattern and clinical pathology as well as laboratory diagnosis of arboviral infection. Various laboratories throughout Australia undertake arboviral diagnosis and subsequent reporting of human arboviral infections, but there has been little or no uniformity between laboratories.

As a consequence of this lack of uniformity in interpreting and reporting results, serious concerns have arisen with respect to the validity of interpretations and subsequent reports to *Communicable Diseases Intelligence*. For example, it has been reported that unusual manifestations may be caused by human pathogens (for example, polyarthritis associated with Murray Valley encephalitis or Kunjin virus, both of which are more usually associated with fever, headache and encephalitis syndromes) and disease syndromes may be attributed to viruses previously unrecognised as human pathogens (for example, associated with Kokobera, Stratford, Alfuy, or other flaviviruses). Therefore we propose that reports of infections with such unusual aetiologies should not be issued unless they meet certain criteria. Indeed, reports of preliminary and other clinical unconfirmed diagnoses provided in the absence of the laboratory findings are a disservice to the patient, to the medical and scientific communities, to our funding agencies, and to ourselves. We have therefore devised the following series of definitions for consideration by laboratories concerned with such testing and reporting. They are a synthesis of a number of discussions initiated during the Sixth Symposium on Arbovirus Research in Australia held in Brisbane in December 1992.

Diagnosis of infection

A number of detailed descriptions of diagnostic tests for arboviruses have been published (for example, Calisher and Monath, 1988[12]; Beaty, Calisher and Shope, 1989[13]). It is generally recognised that the classical techniques for serological diagnosis include the haemagglutination inhibition (HI)[14], complement fixation (CF)[15], neutralisation[16] and indirect immunofluorescent antibody (IFA)[17] tests. The acceptable standard has been a fourfold increase or decrease in antibody titres determined by these methods. More recently, particularly with the availability of monoclonal antibodies, the enzyme-linked immunosorbent assay (ELISA) has been frequently employed, both for IgM and IgG assays. The following definitions relate to diagnosis by these methods.

(a) Paired sera = two serum samples, one of which is an acute-phase sample and the other of which is a convalescent-phase sample. Paired serum samples must be tested together (in the same test run) to obtain comparable results.

(b) Acute-phase = within 7 days of onset of symptoms or signs of illness

(Early) convalescent-phase = within 8 to 14 days of onset of symptoms or signs of illness

(Late) convalescent-phase = within 15 to 28 days of onset of symptoms or signs of illness.

(c) Low antibody titres = haemagglutination inhibition, 80; neutralisation, 80; complement fixation, 32; fluorescent antibody, 32; equivalent IgG ELISA values or titres, depending on individual methodology.

Moderate to high antibody titres = haemagglutination inhibition, 80; neutralisation, 80, complement fixation, 32; fluorescent antibody, 32; equivalent IgG ELISA values or titres, depending on individual methodology.

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It should be noted that the specificity of these tests varies and that neutralisation is the most specific, whereas haemagglutination inhibition is the most broadly reactive, particularly with respect to flaviviruses[12,18]. Indeed in areas of hyperendemicity for flaviviruses (such as northern Australia), broad cross-reactions may occur in HI, CF and even neutralisation tests, limiting their usefulness in defining the infecting agent[19]. Moreover, prior infection may result in a specific rise in antibodies to the heterologous (related) virus responsible for the earlier infection, rather than in antibodies to the current infecting virus[20]. In the case of primary infection, however, any one of these serological methods applied to paired acute- and early convalescent-phase serum samples can provide a specific diagnosis[12,13] providing an appropriate panel of viruses are used as antigens.

The development of IgM antibody assays has provided tools for more rapid serodiagnostic testing because, unlike IgG antibody which is detected later in infection and persists for years after a primary infection, IgM antibody is detected in serum soon after infection with arboviruses and, in many cases, does not persist in high titre. Presence of IgM antibodies, therefore, indicates recent infection in most cases (see below). IgM antibodies are also more specific than IgG antibodies, although cross-reactions remain an interpretive problem in some instances[12]. The most commonly employed IgM assays include IgM antibody capture-ELISA (MAC-ELISA) and indirect immunofluorescence tests, of which the former is the most widely employed method. It should be noted that measurement of IgM antibody in cerebrospinal fluid is an extremely useful method of serodiagnosis of flavivirus encephalitis[12].

Because infections are dynamic processes, serodiagnostic interpretations cannot be made with confidence without having results from tests of paired acute-phase and convalescent-phase serum samples. Furthermore, routine laboratory interpretative comments should not be dependent on the patient's clinical condition. Assigning 'relevance' to results should be left to those with knowledge of the clinical and/or epidemiological circumstances. For example, it is recognised that IgM antibody can sometimes persist for months (flaviviruses; for example, Monath, 1971[21]) or even years (Ross River virus; for example, Carter *et al.*, 1985[22]) after infection, and hence the presence of IgM antibody by itself in a single serum sample or in appropriately paired serum samples has little significance unless IgM antibody titres are determined. In addition to IgM antibody persistence, IgM antibody is recognised to be cross-reactive between closely related viruses. Therefore, the presence of IgM antibody at the screening dilution of acute- or convalescent-phase samples should not be taken as definitive evidence of recent infection with the virus towards which that IgM antibody is reactive. The likelihood of cross-reaction will depend on the virus and type of test, but must be taken into consideration when interpreting results. Within this context, the most specific test is the neutralisation test and the most broadly reactive is the haemagglutination inhibition test. In addition, it is apparent that these standards cannot be applied uniformly and inflexibly to all arbovirus infections.

In Australia, a wide variety of serological tests for arbovirus infections is employed by different laboratories, with both in-house and commercial kits. It is not the purpose of this report to compare or promote individual diagnostic techniques. However, it has been suggested[12] that MAC-ELISA be used for rapidly detecting antibody in situations of immediate importance, and neutralisation, HI, IFA or IgG assays for determining antibody in paired serum samples and for serosurveys. In individual instances in which IgM antibody has been shown to be present, HI and CF can be used as accessory tests, and neutralisation tests can be used for definitive and confirmatory determinations of the infecting agent.

Finally, virus isolation from the patient is the most specific laboratory diagnosis in arboviral infections, but as this is rarely achieved, serological tests will have to suffice. The interpretation of such tests however, should be as uniformly and as clearly reported as possible. We suggest the following categorisations be used for serological diagnosis.

Categories

1. Confirmed infection

(a) At least a fourfold rise or fall in antibody titre between paired sera. The initial specimen should be collected as early as possible in the illness, but is acceptable up to early convalescence (that is, for as long as two weeks after onset). A rise in CF antibody titre may occur later than that detected by HI, ELISA or neutralisation, and may fall earlier than the titre detected by these other tests.

(b) As in 1(a) but with a significant four-fold or greater rise or fall in antibody titre to two or more related arboviruses, with at least a four-fold higher titre to one virus than to the other(s).

Tests for IgM antibody to a particular virus are not necessary, but may be done to confirm acute infection or to provide additional information that may be useful in identifying the causative virus.

2. Presumptive infection

(a) Only an acute phase serum available with IgM antibody and with a negative or low titre by other tests (for example, IgG). (A second specimen collected at 10 to 14 days after onset may show a diagnostic rise in titre and the diagnosis can be changed to 'confirmed'.)

(b) A moderate or high antibody titre in a single specimen with IgM antibody. (If this is an acute or early-convalescent phase serum, then a second in 10 to 14 days may show a diagnostic rise or fall in titre and the diagnosis can be changed to 'confirmed'.)

3. Inconclusive infection

(a) Only an acute phase serum available; IgM antibody negative and/or with negative or low antibody titre

by other tests (a second specimen collected at 10 to 14 days after onset may show a diagnostic rise in titre and the diagnosis can then be changed to 'confirmed'.

(b) Stable, moderate to high titre in two convalescent serum specimens or high titre in a single late convalescent specimen, but no IgM antibody. (Possible infection by a related virus without cross-reacting IgM, or evidence of a past infection with a member of the same antigenic group to which the tested virus(es) belong.)

4. Negative infection:

No IgM antibody and no other specific antibody, or only low titre in a convalescent serum. (The latter could indicate a past infection by that or a related virus.)

Table. Interpretive comments for initial testing

Category	Flavivirus	Alphavirus
Confirmed		
A fourfold increase or decrease in antibody titre. If IgM antibody tests are done, IgM antibody present to the only virus tested.	Indicates recent flavivirus infection.	Indicates recent infection with ---------. Infection with a closely related virus cannot be excluded, particularly if infection occurred overseas.
A fourfold increase or decrease in antibody titre. If IgM antibody tests are done, IgM antibody present to one of several viruses tested.	Indicates recent flavivirus infection due most likely to ----------. Infection with a closely related virus cannot be excluded.	Indicates recent infection with ----------. Infection with a closely related virus cannot be excluded, particularly if infection occurred overseas.
A fourfold increase or decrease in antibody titre, with IgM antibody to more than one virus.	Indicates recent flavivirus infection, but the infecting virus cannot be determined.	Indicates recent alphavirus infection, but the infecting virus cannot be determined.
Presumptive		
IgM antibody and no or low antibody titre by other test(s).	Probable recent infection with a flavivirus. If onset was less than 2 weeks before sampling, a repeat sample after 10-14 days will help confirm recent infection.	Probable recent infection with ----------. If onset was less than 2 weeks before sampling, a repeat sample after 10-14 days will help confirm recent infection.
IgM antibody plus high antibody titre as determined by other test(s).	Recent or past infection or vaccination with a flavivirus. If onset was less than 2 weeks before sampling, a repeat sample after 10-14 days will help separate recent from past infection.	Recent or past infection with ----------. If onset was less than 2 weeks before sampling a repeat sample after 10-14 days will help separate recent from past infection.
Inconclusive		
No IgM antibody, low antibody titre in other test(s).	No significant antibody response to flaviviruses. A second specimen 10-14 days after the first is required to detect or exclude acute infection.	No significant antibody response to----------. A second specimen 10-14 days after the first is required to detect or exclude acute infection.
No IgM antibody, high antibody titre in other test(s).	Probable past infection with a flavivirus. We cannot exclude infection with a closely-related virus for which we have not performed an IgM test.	Past infection with----------.
Negative		
	No evidence of recent infection with a flavivirus.	No evidence of recent infection with ----------.

We emphasise that serodiagnostic confirmation can be obtained only by determining fourfold or greater antibody changes. It is, therefore, essential that every effort is made to obtain a confirmed diagnosis for indicator cases, although a presumptive diagnosis may be sufficient in epidemic situations. It is recognised that some laboratories only undertake IgM testing and in those instances it is essential that reports are issued as presumptive diagnoses. It is also recognised that a second serum sample may not always be available due to various factors including patient or family choice, remote medical assistance, patient's death or other condition, or problems of medical over-servicing.

Serological diagnosis of encephalitis due to Murray Valley encephalitis or Kunjin viruses, or serodiagnosis of a disease syndrome attributed to a virus unrecognised as a human pathogen, should be subjected to further confirmatory diagnosis in a second reference laboratory.

Reporting

1. To *Communicable Diseases Intelligence*

We suggest that reporting the result of serodiagnostic tests through the Laboratory Reporting Schemes to *Communicable Diseases Intelligence* should be as follows.

Flavivirus results should be restricted to confirmed, clinically-recognised cases; presumptive diagnoses and subclinical cases should **not** be included unless they are:

(i) of epidemiological importance (for example, during specific epidemics), or

(ii) cases of clinically-confirmed dengue infection with a single positive serum containing IgM and/or a high IgG antibody titre; or

(iii) international travellers exposed to infection overseas and for whom only a single IgM specimen was available.

In submitting summary or brief reports to *Communicable Diseases Intelligence*, cases involving presumptive diagnoses should be clearly denoted with full clinical history. Subclinical cases of flavivirus diseases are of considerable epidemiological interest, and confirmed subclinical infections should be submitted as brief reports to *Communicable Diseases Intelligence*.

Alphavirus results should be reported either as confirmed, clinically-recognised cases or, when only single serum specimens were submitted for diagnosis, as presumptive (IgM positive) clinically-recognised cases. Indeed the Laboratory Reporting Schemes have the capacity to compile data on the method of diagnosis, and thus 'confirmed' cases can be reported as fourfold changes, and 'presumptive' cases as IgM positive. Subclinical cases of alphavirus diseases are also of epidemiological interest, and confirmed sub-clinical infections should be submitted as brief reports.

If the identity of the infecting virus is uncertain, it would be appropriate to use the virus codes for unspecified flavivirus (9998) or unspecified alphavirus (9901).

For epidemiological purposes, it is invaluable to record the location of acquisition of infection or, if not available or known, the location of the patient, and all arbovirus reports should provide this information to *Communicable Diseases Intelligence* through the Laboratory Reporting Schemes.

2. To physicians requesting laboratory diagnoses

We suggest the interpretive comments detailed in the Table be used for reporting the results of **initial** laboratory tests to requesting physicians.

In unusual or atypical infections, or when the diagnosis is of public health significance, further tests should be carried out to confirm or clarify the results. This should be done by a reference laboratory in each of two different States. The major reference laboratories are State Health Laboratory Services, Perth; Institute of Medical and Veterinary Science, Adelaide; Fairfield Hospital, Melbourne; State Health Laboratories, Brisbane; and Westmead Hospital, Sydney.

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CDI Editorial Comment

CDI has been aware of the lack of uniformity in interpreting and reporting results of laboratory diagnosis of arbovirus infection. In particular, we have been aware of concern that some workers have had about diagnoses based on the demonstration of IgM in a single specimen. These concerns have increased when there have been consequent reports of unusual clinical syndromes (for example polyarthritis being attributed to Kunjin virus), and of clinical disease associated with arboviruses, such as Sindbis, not usually recognised as human pathogens. These problems have accompanied other difficulties in the interpretation of the data which have arisen as some arbovirus diagnosis has moved from contributing laboratories to laboratories not involved in the *CDI* Laboratory Reporting Schemes.

The definitions and reporting guidelines proposed in this article are welcomed, as they will provide a consistent base for the *CDI* Laboratory Reporting Schemes surveillance of arbovirus infection in Australia. Laboratories are strongly encouraged to use the case definitions and reporting guidelines when reporting arbovirus infections to the *CDI* Schemes.

Surveillance case definitions for the *CDI* Laboratory Reporting Schemes

In summary, reports of the following will be accepted:

- flaviviruses
  - confirmed cases, reported as fourfold changes, and
  - presumptive cases, reported as IgM, only if
    - of epidemiological importance, or
    - of clinically confirmed dengue, or
    - of international travellers exposed overseas
- alphaviruses
  - confirmed cases, reported as fourfold changes
  - presumptive cases, reported as IgM.

Confirmed and presumptive cases are as defined in the preceding article.

Information on the attributed clinical syndrome is important for interpretation of the data, and should be included in the DIAGNOSIS (LabVISE and new blue forms) or SYNDROME (old green or white forms) fields. Confirmed cases of subclinical infection should be submitted as brief reports for inclusion in the Communicable Diseases Surveillance section of *CDI*.

If a second sample becomes available for either a flavivirus or an alphavirus infection, and a diagnosis is changed from presumptive to confirmed, laboratories are encouraged to submit a second report, as a 'fourfold change'. If the patient identifying information is reported in the same way each time, the first report can

Table. *CDI* Laboratory Reporting Schemes arbovirus reports 1982 to 21 April 1993, by reported diagnosis method

Arbovirus	Total reports	Reported diagnosis method (per cent[1])			
		Fourfold change 'confirmed'	IgM 'presumptive'	Single high titre or total antibody	Other or unknown
Ross River virus	11,138	3.2	95.9	0.6	0.2
Barmah Forest virus	367	6.3	90.7	3.0	0
Unspecified alphavirus	287	47.4	34.1	18.5	0
Dengue type 1	24	0	100.0	0	0
Dengue type 2	313	0.3	88.2	11.5	0
Dengue not typed	562	6.6	92.7	0.4	0.4
Kunjin virus	67	1.5	95.5	3.0	0
Murray Valley encephalitis virus	22	0	100.0	0	0
Unspecified flavivirus	242	14.5	81.0	4.5	0
All arboviruses[2]	13,036	4.5	93.8	1.4	0.2

1. Percentages do not all total 100, due to rounding.
2. The total includes the 14 reports of other arboviruses (6 dengue type 3, 1 dengue type 4, 2 Kokobera, 3 Stratford and 2 Sindbis).

be detected as a duplicate and deleted, so that the case is not counted twice.

Laboratories are also encouraged to report any information which they have on the location of acquisition of infection, or location of the patient. For laboratories reporting using the computerised LabVISE system or the new blue forms, location can be recorded either in the POSTCODE field, or the name of the town or area can be added in the COMMENTS field. For laboratories reporting on the old green or white forms, the postcode or the name of the town or area can be written in the area for COMMENTS. A history of overseas travel can either be recorded as a risk factor (code 64) in the RISK field (LabVISE and blue forms) and/or reported in the COMMENTS.

We recognise that in many instances, a single serum specimen is all that is available to laboratories, so presumptive cases will be reported more commonly than confirmed cases. Indeed, for most of the reports of arbovirus infection made to *CDI* Laboratory Reporting Schemes since 1982, the reported method of diagnosis was a demonstration of IgM (Table). Only 4.5% were of fourfold changes in antibody titre. There were small numbers reported for single high titre or total antibody (1.4%) and other or unknown (0.2%). The proportion of reports based on IgM was high for all arboviruses, except for 'unspecified alphavirus', a category reported mainly prior to 1985, and not at all since 1989. No reports of isolation of arboviruses or of IgM in cerebrospinal fluid have been received by the *CDI* Schemes, but they would be welcomed.

The *CDI* Schemes are the only form of national laboratory arbovirus surveillance in Australia. The surveillance case definitions used have, necessarily, been a compromise between sensitivity, specificity, simplicity and lack of ambiguity. The inclusion in the past of cases which would now be categorised as presumptive has enabled the Schemes to record the generally accepted pattern of arbovirus activity in Australia. If we were to have only accepted the small number of 'confirmed' cases in the past, the Schemes' record of Australian arbovirus activity would be very different. Seasonal peaks of Ross River virus activity would not be clear, and the recent dengue 2 activity in Queensland would not have been recorded at all. Of course, there would also have been no problems with the reporting of unusual 'cases'!

The guidelines for reporting proposed here will ensure that the *CDI* Schemes will be able to continue to accept most of the reports of arbovirus infection which we currently receive, and so continue to provide a picture of arbovirus activity which is generally accepted. However, they will also ensure that potentially misleading information is not included. Presumptive cases will be able to be readily identified in all reports in *CDI* and elsewhere, and their relative importance will be able to be assessed, as appropriate to the different uses made of the data.

It should be noted that all the above comments refer only to the *CDI* Laboratory Reporting Schemes. Arbovirus infections are also notifiable diseases in all the States and Territories, and the National Notifiable Diseases Surveillance System includes notifications of infection with Ross River virus, dengue (all types) and unspecified arboviruses. Currently there are no case definitions for this System, although a set has been proposed.

LABORATORY DIAGNOSIS OF ROSS RIVER VIRUS INFECTION

(Gordon Rich, John McKechnie, Ian McPhan and Brett Richards, Western Diagnostic Pathology, Myaree, Western Australia)

In issues of *CDI* since November 2 1992, there have been reports of Ross River virus (RRV) infection apparently diagnosed by detection of IgM. In *CDI* of March 22 1993 (Vol. 17 No. 2), for example, it states 'one report from Victoria was a four-fold change in antibody titre; the remainder of reports were of IgM detection (presumptive cases)'. The four-fold rise in titre is an accepted rule for serological diagnosis. A presumptive diagnosis by detection of IgM alone may be acceptable provided that the IgM is detected by a method of proven specificity.

Western Diagnostic Pathology uses the only commercial antibody detection available and it is likely that it is used by many reporting laboratories. In our hands we are aware that we cannot assume the IgM detected is RRV specific and therefore believe that there may have been a significant over-reporting of cases. We cannot comment on the specificity of IgM in those laboratories that use 'in-house' methods of detection.

This laboratory service operates 15 laboratories which obtain specimens from all five health districts of the Northern Territory and from all health regions of Western Australia except the Kimberley. RRV infection is endemic in all these regions/districts and some, such as the Darwin and East Arnhem districts in the Northern Territory, and the South West region of Western Australia have had significant epidemics in recent years, including 1992. All the RRV serology testing for our service is carried out in our central laboratory in Myaree (Perth).

On the basis of our assumption, our results are given with the comments as follows:

1. IgG detected, IgM detected. 'Consistent with recent infection with Ross River virus.'
2. IgG detected, IgM not detected. 'Note that IgM may disappear after 3-4 months. If the patient's illness is longer than this RRV may be the pathogenic agent.'
3. IgG not detected, IgM detected. 'Results are suggestive of recent RRV infection, however, cross reactions with other viral antibodies do occur. Please repeat serology in 2 to 4 weeks to confirm.'

The use of the first comment automatically generates the further comment 'your patient is suffering from a notifiable infection'. This does not occur when either of the other comments is used.

In order to substantiate our stated assumption that IgM detection by the commercial kit lacks diagnostic specificity we have analysed our results for all assays done in 1992. In 1992, we tested 6,413 patients for RRV antibody; 1,009 (16%) had antibody detected (Table 1). In 171 patients (2.7%), IgM was detected but not IgG. In 112 of these patients further specimens were sent for testing but six were delayed for 13 weeks or longer and have been excluded from analysis. Of the 106 patients tested within 12 weeks (average interval was 4.5 weeks), 58 patients (55%) had developed IgG by the time of the second test, leaving 48 (45%) who we assumed were false positives. If this ratio were the same for those 59 patients who were not rechecked then a

Table 1. Detection of antibody to Ross River virus in 6,413 patients

	Number of patients	Per cent	Comment Used
IgG positive only	464	7.0	2
IgM positive only	171	2.7	3
IgG and IgM positive	374	6.0	1
Negative	5,404	84.3	-

Table 2. Results of repeat samples for antibodies to Ross River virus and Barmah Forest virus[1]

	1st Specimen	2nd Specimen (2-4 weeks)	3rd Specimen (7 weeks)
RRV IgG	N	N	N
RRV	9	7	1
RRV IgM	P	P	N
		P	
		2	
		P	
BFV IgG	N	N	P
BFV	8	6	1
BFV IgM	P	P	P
	P	P	
	1	3	
	P	P	

1. N negative, P positive

total of 75 (27 + 48) patients could be construed as having had a false positive diagnosis.

Using the accepted diagnostic criterion 'IgM detection' indicating a positive case, we had 545 patients (8.5%) as confirmed. It would appear that probably 75 were false positives, reducing the number confirmed to 470 patients (7.3%). This may not be of great epidemiological importance but it is a considerable inaccuracy. It is important to be aware that 14% or patients are given the wrong diagnosis.

It is possible, or course, that some of those patients who had both IgG and IgM detected could have had a false positive result because of the same poor specificity. This would happen in an individual who had past infection and was IgG positive and had some other infection which provoked a non-specific IgM response. For inapparent reasons 70 patients in this category were retested, although unfortunately, 22 of these were 13 or more weeks after the first specimen was submitted. Of the 48 who were retested within eight weeks, four patients (8%) did not have IgM detected in their second specimen. On the bases of rapid disappearance of IgM indicating a cross reaction, these four may have been false positive reactions.

Causes of false positive RRV IgM

In those patients whose second serum sample failed to either confirm or exclude RRV infection, further testing was undertaken to attempt to identify a cross reacting virus infection. We selected three agents which we believe produce clinical syndromes similar to RRV and which, in our experience, could cause cross reactions with RRV IgM. These were Barmah Forest virus (BFV), rubella virus and Epstein Barr virus (EBV).

We found no cross reactions due to EBV infection.

The situation with rubella was exacerbated by the rubella epidemic in the last four months of 1992 (a total of 158 adult cases were serologically confirmed in our laboratory). In six patients, concurrent testing showed acute rubella but no evidence of recent RRV infection. In seven patients there were IgM cross reactions and in three repeat samples at 2 to 4 weeks the RRV IgM had disappeared but rubella seroconversion had occurred. In the seventh patient, the pattern was RRV IgG positive and IgM positive in both sera with rubella IgM positive in both and IgG only becoming detectable in the second specimen.

Taking into account that there was no epidemic of BFV infection to potentiate confusion, it seems that cross reactions between RRV and BFV antibodies are frequent. In nine patients IgM to both RRV and BFV was initially demonstrated (one patient also with IgG to BFV, Table 2). Repeat sera were obtained from all at between 2 and 4 weeks. In one patient seroconversion to BFV was apparent in the second specimen. In two patients seroconversion to both agents was present in the second specimen raising the possibility of dual infection. In one of these, a third specimen was fortunately submitted in which neither RRV IgG or IgM antibody could be detected. As both IgG and IgM to BFV were still present this strongly suggests that BFV was the sole infecting agent. This is a salutory lesson that nothing is certain about serological diagnosis!

PRELIMINARY REPORT OF AUSTRALIAN ENCEPHALITIS IN WESTERN AUSTRALIA AND THE NORTHERN TERRITORY, 1993

David Smith, State Health Laboratory Services, Perth, Western Australia; John Mackenzie, Annette Broom, Department of Microbiology, The University of Western Australia; Dale Fisher, Michael Williams, Jim Burrow, Bart Currie, Royal Darwin Hospital, Northern Territory)

Australian encephalitis is caused by two mosquito-borne flaviviruses, Kunjin (KUN) and Murray Valley encephalitis (MVE) viruses. These viruses are endemic in the Kimberley region of Western Australia and the Top End of the Northern Territory and epidemic in the Pilbara and Gascoyne regions of Western Australia. The last cases in Western Australia and the Northern Territory occurred in 1991[1,2]. The following is a brief report on the first seven cases appearing this year and follows heavy rain with flooding in the Kimberley region and the Top End.

Case 1

A 12 month old Aboriginal male was seen in late March at Fitzroy Crossing in the Kimberley region with a nonspecific illness progressing rapidly to an encepholopathic state with fitting. Following hospitalisation, the child recovered to apparent normality over 10 days. He was resident near Fitzroy Crossing and had not travelled in the month preceding the onset. His flavivirus antibody by haemagglutination inhibition (HI) rose from 1:20 to 1:160 and MVE specific IgM was detected in serum and cerebrospinal fluid (CSF) by indirect immunofluorescence (IIF). Specific MVE antibody was confirmed by a blocking enzyme immunoassay (EIA) using monoclonal antibody.

Case 2

A 12 month old Aboriginal male from Billiluna (Kimberley region) presented in late March with a three day history of malaise, cough and fever. He subsequently became obtunded, tachypnoeic and hypertonic and began having focal convulsions. He was transferred to the Royal Darwin Hospital (RDH) on day 4 where he required artificial ventilatory support. His progress was poor with signs of brain-stem involvement and development of a flaccid paralysis. Pneumonia ensued and he died on day 14 of his illness. One serum speci-

men collected three days before death had a flavivirus HI titre of 1:640 or greater with MVE IgM detected by IIF. MVE IgM was also detected in CSF collected at the same time, and both serum and CSF antibodies were confirmed as being MVE specific by monoclonal blocking EIA.

Case 3

A two year old Aboriginal female from Turkey Creek (on the Western Australia-Northern Territory border) presented in mid-April with a three day history of cough, fever and drowsiness. By day 5 her condition had deteriorated and a generalised convulsion occurred necessitating her transfer to RDH. She was hypertonic and hyperreflexic but gradually her state improved allowing discharge on day 19. She remained irritable but otherwise normal and had no fits for one week after withdrawal of anticonvulsants. A serum collected on arrival at RDH had an HI titre of 1:160. Serum and CSF examination revealed MVE IgM. MVE specific antibody was confirmed by monoclonal blocking EIA.

Case 4

A 24 year old male Caucasian station hand from near Halls Creek presented in late April with a one day history of headache, fever and increasing confusion. There was evidence of confusion for the preceding two weeks, associated with minor headache. Respiratory failure following a lumbar puncture necessitated intubation and transfer to RDH. He was extubated the following day but remained initially unconscious. By day 11 of this admission, mentation, speech and movement remained slow. Focal signs of fitting were never present. A serum specimen taken on admission had a flavivirus HI titre of 1:160 with a positive MVE IgM by IIF in the serum and CSF.

Case 5

A 38 year old Caucasian female was holidaying at Kununurra between 3 and 11 April. Her family could recall mosquito bites. In early May, she presented to RDH with a 10 day history of headache, vomiting and fever and one day of confusion. Her mental state deteriorated for a further two to three days before improvement was noted. By day 9 of her admission, she was continuing to recover although assistance with feeding and mobilising were still required and speech was relatively incoherent. Neither convulsions nor focal signs were a feature of her illness. Serum collected on admission had an HI titre of 1:20 and both serum and CSF were positive for MVE IgM by IIF.

Case 6

A six month old Aboriginal female from Beswick (80km east of Katherine) presented in late April with a three day history of illness including fever, drowsiness and convulsions. She was transferred to RDH and received treatment for pneumonia (presumed aspiration). Her encephalopathy did not progress to the point of requiring intubation. By day 16 she was continuing to improve. There have been no further fits one week after cessation of anticonvulsants. Sera collected two and four days after admission had flavivirus titres of <1:10 and 1:10 respectively. MVE IgM was detected by IIF in serum and CSF.

Case 7

A 14 month old Aboriginal female from Lajamanu (Hooker Creek) on the edge of the Tanami Desert in the Northern Territory was transferred to RDH at the end of April with a three day history of fever, tachypnoea, irritability and increasing drowsiness. Generalised fits precipitated the need for intubation and ventilation. Focal convulsions and long tract signs followed with associated brainstem abnormalities and subsequent flaccid paralysis. However improvement was observed on day 12 of her illness and has continued. On day 17 she remained intubated and ventilated with a flaccid paralysis, but improving conscious state and brainstem function. Serum collected on admission ha[redacted] a flavivirus HI titre of 1:20, while MVE IgM was detected by IIF in serum and CSF.

Most of these cases are undergoing continuing investigation and specimens will also be referred to another reference laboratory for confirmation. Follow-up specimens are being obtained to help show definite acute infections.

These cases appeared following flooding in the Kimberley region which extended into the Northern Territory beyond Katherine with a subsequent increase in potential vector mosquito numbers. Disease activity had been preceded by seroconversions to MVE in sentinel chicken flocks located at Kununurra, Wyndham, Halls Creek, Fitzroy Crossing and Broome[3]. A high level of virus activity has continued to be observed in chicken flocks during April, indicating an ongoing potential for further cases of Australian Encephalitis in the region. Field collections of mosquitoes for viral and epidemiological studies are currently being unde[redacted] taken. Local health staff have been alerted and th[redacted] public has been advised regarding personal protection against mosquito bites.

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A TIME BOMB IN NORTH QUEENSLAND: CASE REPORT OF INTRODUCED MALARIA SOUTH OF THE NINETEENTH PARALLEL

(S Murray-Smith, Bedside Manor, Charters Towers, Queensland, and P Weinstein, Department of Zoology, James Cook University, Townsville, Queensland)

Background

In 1981, the World Health Organization certified Australia as being free not from malaria, as is generally quoted, but from indigenous (endemic) malaria[1]. This certification meant only that 'malaria acquired by mosquito transmission in an area where malaria occurs regularly'[2] could no longer be found in Australia. Imported malaria clearly still occurs, with over 700 cases annually in recent years[3,4]. It is inevitable that a few of these cases will be gametocytaemic, and therefore capable of infecting local mosquitoes. It comes as no surprise then that introduced malaria, or 'malaria acquired by mosquito transmission from an imported case in an area where malaria does not occur regularly'[2] still occurs in Australia. An outbreak involving six cases occurred in north Queensland as recently as 1987[5], and cases from the Torres Strait are reported annually[3,4].

Given the worsening malaria situation worldwide and particularly in South-East Asia[6], we feel that it is only a matter of time before imported gametocytaemic cases and introduced cases again 'occur regularly' enough for malaria to be considered indigenous in Australia. This is obviously most likely to happen in the so called 'malaria receptive zone' north of the nineteenth southern parallel. In the case report which follows, we discuss yet another instance of introduced malaria, and emphasise the urgent need to redefine this traditionally accepted 'receptive zone'.

Case report from Charters Towers

Charters Towers is a rural city in north Queensland situated just south of the twentieth parallel, 132 kilometres south-west of Townsville. The city has four private schools which cater for students from Papua New Guinea (PNG), sponsored both privately and by the Australian International Development Assistance Bureau (AIDAB).

Routine malaria screening is carried out for all students both upon their initial arrival and upon returning from PNG after each holiday. The rapid QBC test[7,8] is used as the initial screening test and any positives are rescreened using traditional Giemsa staining of thick and thin smears.

Of 147 students screened in February 1993, 10 were found to be asymptomatically parasitaemic and three students presented with florid malaria. Two of these florid cases were noteworthy.

Case 1

Case 1 was a female student (date of birth 1 December 1975) who was a native Papuan, resident of Alotau, PNG. She arrived in Australia on 2 February 1993. A screen taken on 4 February 1993 was negative. She presented on 16 February 1993 with high temperature and rigors. Her QBC test was positive and thick and thin smears demonstrated falciparum malaria. The initial blood sample was rechecked and again proved negative.

The timespan of 14 days between arrival and development of florid malaria, although not conclusive, tends to point to acquisition of infection within Australia (*Plasmodium falciparum* incubation 12 days (range 9-14)[9]).

Case 2

Case 2 was a caucasian male student (date of birth 1 March 1976) who was an Australian citizen, residing in Rabaul, PNG. He developed fever at home in Rabaul, and a smear taken by a local health worker demonstrated malaria. He was given a three day course of chloroquine with a treatment course of primaquine. The course of primaquine was not completed.

He arrived in Australia on 30 January 1993, and developed rigors on 31 January. His QBC test was positive and smears demonstrated falciparum malaria. Treatment commenced using quinine 600mg three times per day and 'Fansidar' three tablets at once on day three. Follow-up was by weekly bloods for four weeks. All smears were thought to contain degenerate trophozoites. A sample of the patient's blood was sent to the Army Malaria Research Unit at Ingleburn where these anomalies were demonstrated to be gametocytes. Formal gametocidal treatment with 30mg of primaquine was instituted with subsequent resolution of symptoms.

The timespan of 18 days between the arrival of case 2 and the presentation of case 1 could allow for replication in a vector and subsequent incubation in case 1 (*P. falciparum* sporogony 9-22 days[9]).

Discussion

In the cases we have outlined, the evidence, although circumstantial, points to local transmission of falciparum malaria from case 2 to case 1, and case 1 therefore represents an instance of introduced malaria. To the proponent of the nineteenth parallel theory, an introduced case south of the twentieth parallel may come as a surprise. However, historical records indicate that during the Second World War, malaria was

indigenous as far south as Sellheim, some 175 km south of Townsville near the twenty-first parallel[10]. Infected troops in these indigenous areas were carefully kept from the vector population by screens and nets during acute phases, then removed to vector free areas either in the Atherton tablelands or southwards.

The Australian anophelines capable of malaria transmission also range further than is generally recognised. *Anopheles farauti* s.l. and *An. punctulatus* are well documented vectors of malaria in northern Australia. They are generally regarded as being confined to areas north of the nineteenth parallel, which is part of the reason that the 'receptive zone' has been considered to lie north of this boundary. However, *An. farauti* has been collected as far south as Mackay, south of the twenty -first parallel (D. Sinclair, personal communication). The range of another endemic anopheline, *An. annulipes*, is known to extend into Tasmania[11], but the species tends not to be taken seriously as a potential vector[12]. This is despite a comprehensive list of introduced cases from New South Wales and Victoria, in which *An. annulipes* was the presumed vector[13]. Studies by Mackerras and Roberts[14] demonstrated that this species does not differ from *An. farauti* and *An. punctulatus* in its susceptibility to *P. vivax* and *P. falciparum*, and it does feed on humans. In tandem with demographic or climatic changes, *An. annulipes* should therefore be considered capable not only of transmitting indigenous malaria south of the nineteenth parallel, but also of transmitting introduced malaria as far south as Tasmania. The species features in qualitative trappings from Charters Towers, and quantitative sampling (conducted by the Departments of Zoology and Public Health and Tropical Medicine at James Cook University) is imminent.

Some 150 students arrive annually in Charters Towers from areas indigenous for malaria, chiefly PNG. The screening we have reported demonstrates that 6% of students are parasitaemic upon arrival; add to this students returning from school holidays later in the year, and parasitaemic students from a number of similar rural cities, and it can be concluded that a number of gametocytaemic cases must be introduced into Australia on a regular basis. With this ill defined but undoubtedly significant risk of infecting local mosquitoes and humans, it is anomalous that responsibility for the organisation and funding of local screening programs lies largely with local practitioners. Screening can therefore be inconsistent, and is very dependent on individual enthusiasm.

Conclusion

In the above case report we have outlined the circumstances under which malaria is believed to have been transmitted locally at Charters Towers, south of the twentieth parallel. Historical case reports and present vector distributions indicate that local transmission outside the traditionally accepted 'receptive zone' is not exceptional nor surprising. We feel therefore that there is good reason to redefine the 'malaria receptive zone' in northern Australia in terms of demography and mosquito vector ecology. It would be timely to do so before the ecology of our local vectors is potentially altered by the advent of global warming and increased rainfall, or by the introduction of new anopheline vectors from Melanesia. Only with such studies will we be able to define a scientific baseline from which to monitor and manage the changes in Australian malaria epidemiology which may confront us.

Acknowledgments

We thank Geoff Davis, AIDS/Communicable Diseases Branch, Department of Health, Housing, Local Government and Community Services; David Sinclair, Tropical Public Health Unit, Peninsula and Torres Strait Regional Health Authority; and Rick Speare, Department of Public Health and Tropical Medicine, James Cook University, for historical references and for reviewing the manuscript.

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OVERSEAS BRIEFS

In the last two weeks, the following information has been supplied by the World Health Organization and the Institut Pasteur, Paris.

Cholera Update

Cases have been reported for February, March and April from Argentina, Belize, Bolivia, Brazil, Cameroon, Chile, Ecuador, El Salvador, Guatemala, Honduras, Malawi, Mexico, Mozambique, Nicaragua, Panama, Peru, Rwanda, Zambia and Zimbabwe.

Sussundenga District in the Manica Province of Mozambique has recently been declared infected.

Influenza Update

Influenza B has been detected by immunofluorescence in 4 infants in Papua New Guinea, and in a patient in South Africa who had arrived from the United States and the United Kingdom.

In the Northern Hemisphere, the incidence of influenza is declining in most areas. In the United States, influenza was reported on 24 April as sporadic in 17 States and regional in 8, decreasing from previous weeks. In Europe, the epidemic is over or declining in most countries, although sporadic cases are being reported from Austria, Denmark, Finland, France (influenza A H_3N_2), Norway, Switzerland and the United Kingdom. The proportions of influenza A and influenza B have differed from country to country.

COMMUNICABLE DISEASES SURVEILLANCE

Laboratory Reporting Schemes

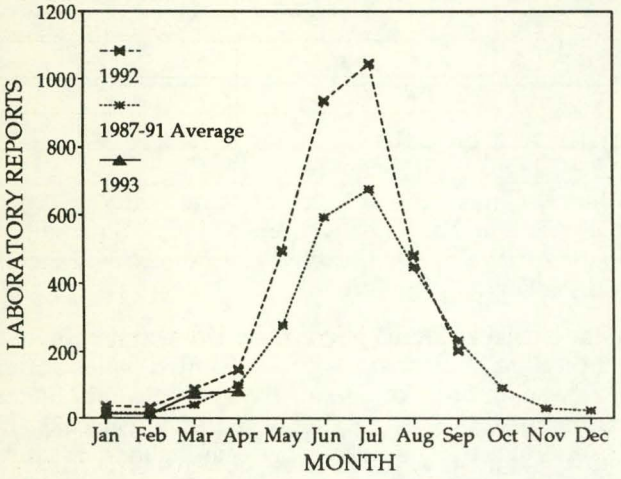
There were 1,523 reports received in the *CDI* Virology and Serology Reporting Scheme this fortnight (Tables 7,8 and 9), and 43 reports of isolates from normally sterile sites (LabDOSS, Table 3).

- **Measles** was reported for 13 patients. Six of the patients were in the 20 to 45 year age group. There have been 154 cases reported so far this year.
- There have been 14 **rubella** reports this fortnight. Included were 5 reports in females in the age group 15 to 44 years, and 9 reports in males ages ranging from 17 years to 30 years. There has been a total of 486 reports so far this year,
- A total of 193 reports of **hepatitis C** were received. Forty-five of these reports were from asymptomatic persons, 7 reports were from patients with hepatitis.
- There were 78 reports of **Ross River virus** infection. Seventy-five diagnoses were presumptive (IgM positive only). Three patients showed a fourfold rise in titre indicating confirmed infection.
- Locations (or reporting laboratories) were Victoria (15 reports), New South Wales (1), Western Australia (12), Queensland (23)and South Australia (27). There have been 1,034 reports of Ross River virus infection so far this year, mainly from South Australia, Queensland and Victoria.
- **Barmah Forest virus** was reported for 10 patients, all were presumptive (IgM positive only). States for reporting laboratories were Western Australia (5), Queensland (3) and South Australia (2).

Table 1. Laboratory reports of cytomegalovirus congenital infections, by State or Territory and year of specimen collection, 1990 to 1993

	State or Territory of reporting laboratory						
YEAR	ACT	NSW	Qld	SA	Vic	WA	Total
1990	1	16	4	6	23	13	63
1991	0	22	5	4	18	9	58
1992	2	18	17	4	36	3	80
1993	0	5	2	0	5	0	12
Total	3	61	28	14	82	25	213

Figure 1. Respiratory syncytial virus laboratory reports, 1987-91 average, 1992 and 1993, by month of specimen collection



- **Murray Valley encephalitis virus** was reported from Western Australia. More details about this case are in this issue of *CDI* (case 2, page 209).
- There were 72 reports of **cytomegalovirus** infection, 46 of which were from Victoria. Included were 4 congenitally infected infants. The number of congenitally infected infants with CMV since 1990 reported to *CDI* with respect to State has been recorded in Table 1.
- **Parvovirus** infection was reported in 5 patients from Victoria; included were a 38 year old female with arthritis and a 10 year old female with anaemia(spherocytosis).
- There were 5 reports of **influenza A** (four single high titres and one unknown serological result) and 3 reports of **influenza B** (2 isolates and one single high titre) this fortnight, all from South Australia. One isolate of influenza A was from a male 81 years of age.
- There continues to be an increase in numbers of **parainfluenza virus type 3** with 22 cases reported this fortnight.
- **Respiratory syncytial virus** was reported from the 38 patients last fortnight, the trend evident in previous years with a sharp rise beginning in April-May has not yet become obvious (Figure 1).
- The 19 cases of **Q fever** reported came from laboratories in Queensland, New South Wales and Western Australia. Western Australia has already reported as many cases as reported for any of the previous years since 1990.
- **Echovirus type 7** was reported from 10 patients this fortnight. Four cases were from CSF (one month old male, 4 month old male, 4 year old male and a 15 year old male).
- *Mycoplasma pneumoniae* has been reported in 68 patients so far for May (predominantly in the eastern States) indicating that 1992-93 will show a significant level of *Mycoplasma pneumoniae* activity. This follows the trend that has emerged from data since 1978 (Figure 2).

Figure 2. *Mycoplasma pneumoniae* laboratory reports, 1978 to 1993, by month of specimen collection

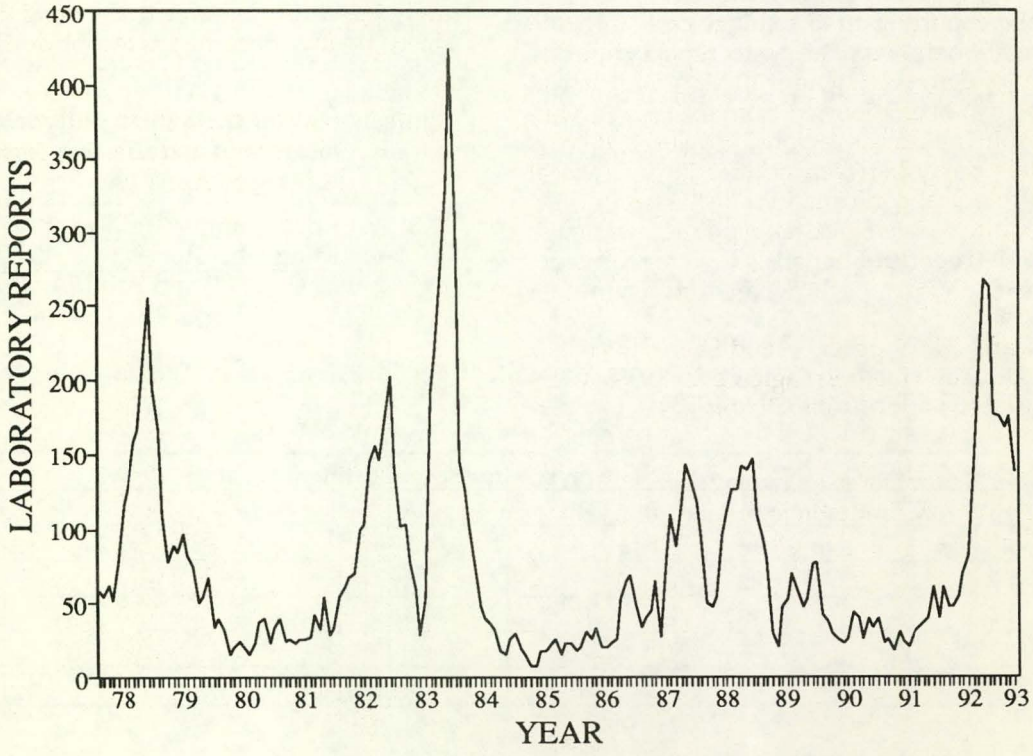


Table 2. Australian Sentinel Practice Research Network, Weeks 18 and 19, 1993

Condition	Week 18, to 2 May 1993		Week 19, to 9 May 1993	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	32	6.1	39	7.0
Measles	0	0	0	0
Rubella	1	0.2	5	0.9
Pertussis	0	0	0	0
Genital herpes	3	0.6	1	0.2
Gastroenteritis	67	12.7	67	12.0

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network collected data from 5,275 patient encounters in Week 18 and from 5,600 patient encounters in Week 19 (Table 2). Influenza was reported at a higher rate than has been usual for recent months.

Dengue in Far North Queensland

The first case of locally acquired dengue known to have occurred in Far North Queensland since the epidemic of dengue 2 began in Townsville in May 1992 was serologically confirmed at the end of April 1993. The patient's symptoms began in mid-March, but dengue was not clinically suspected until minor haemorrhagic manifestations occurred 2 weeks later.

The patient had no recent travel history either abroad or to the Townsville-Charters Towers region[1]. Mosquito surveillance revealed *Aedes aegypti* larvae in properties adjacent to the patient's residence.

Following the reporting of the initial case, all local medical practitioners were asked to report clinically suspect cases of dengue. Up until 10 May, a further 12 suspect cases had been reported from the Cairns-Mulgrave-Atherton Tablelands regions. However, there is a considerable delay between notification of clinical cases and serological confirmation. In the meantime, further mosquito surveillance and public awareness campaigns are in progress.

Reference

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(C Streeton, Centre for Disease Control, Cairns, and the National Centre for Epidemiology and Population Health, Canberra)

An outbreak of Q fever in Western Australia

An outbreak of Q fever has occurred related to one abattoir in the south-west of Western Australia. Twelve cases of Q fever have been notified to the Health Department of Western Australia so far this year compared with an average of 5 for the last 3 years. Nine of the cases notified this year worked at the same abattoir or its subsidiary.

A case was defined in the outbreak investigation as a patient who presented with symptoms such as profuse sweating, fever, headache and myalgia; worked at the particular abattoir; and showed either a fourfold rise in phase II Q fever antibody titre or a very high titre.

There were 8 males and 1 female, aged from 14 to 42 years. The dates of onset extended from 3 February to 16 April 1993 (Figure 3). As diagnosis is often delayed by the time required for the laboratory tests, data for April and May are incomplete.

Vaccination for Q fever is available but had not been offered to employees due to the rapid turnover of staff. The company concerned is reconsidering this practice.

Figure 3. Q fever cases in an outbreak in an abattoir, Western Australia, by fortnight of onset, January to April 1993

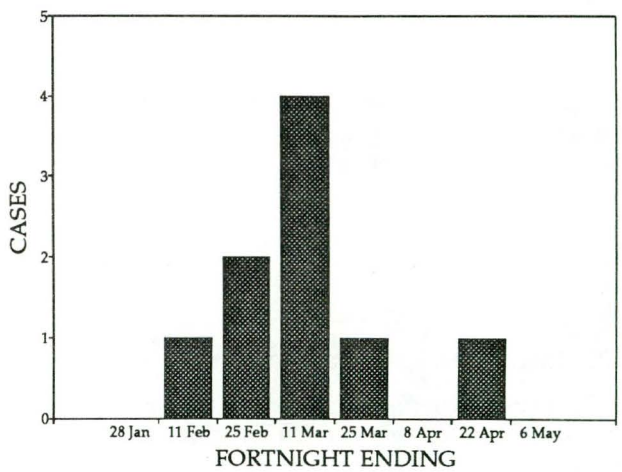


Table 4. LabDOSS reports of blood isolates

Organism	Total[1]	Clinical Information						Risk Factors				
		Bone/Joint	Lower respiratory	Endocarditis	Gastrointestinal	Urinary Tract	Skin	Surgery	Immunosuppressed	IV line	Perinatal	Neonatal
Staphylococcus aureus	8[2]	1								1		
Escherichia coli	8					2						

1. Only organisms with 5 or more reports are included in this table.
2. MRSA 1.

Very few of the cases were actually treated as they had recovered by the time the results were available. If the patients' work colleagues are clinically diagnosed with Q fever, it would be advisable to treat with tetracycline before waiting for the results of the laboratory tests.

Q fever, caused by *Coxiella burnetii*, affects mainly meat workers, dairy workers, farmers and veterinarians. Transmission is airborne or by direct contact with infected animals, placental tissue, birth fluids, excreta and other contaminated materials. Chronic infection can result in granulomatous hepatitis and endocarditis.

(Margaret Ashwell, Health Department of Western Australia and National Centre for Epidemiology and Population Health, Canberra)

Sterile Sites Surveillance (LabDOSS)

Data for this fortnight have been provided by 5 laboratories. A total of 43 reports have been included: Northern Tasmanian Pathology Service 9, Nambour General Hospital 7, TB Lynch Pathologists, Rockhampton 4, Toowoomba General Hospital 10, New England Pathology Tamworth 13.

Organisms reported 5 or more times from blood are detailed in Table 4. Other blood isolates not included in Table 4 were:

Gram positive: 1 *Streptococcus* Group A, 2 *Streptococcus* Group B (1 male neonate), 1 *Streptococcus* Group G, 1 *Streptococcus pneumoniae* (1 year old female), 2 *Staphylococcus epidermidis*, 1 *Staphylococcus hominis*.

Gram negative: 1 *Acinetobacter baumannii*, 4 *Klebsiella pneumoniae*, 1 *Serratia marcescens*, 1 *Haemophilus influenzae* type b (8 month old male), 2 *Pseudomonas aeruginosa*, 1 *Pseudomonas* species.

Anaerobes: 1 *Peptostreptococcus* species, 1 *Propionobacterium* species.

Fungi: 1 *Candida tropicalis*.

CSF Isolates and meningitis reports

1 *Haemophilus influenzae* type b (2 year old male), 1 *Neisseria meningitidis* (group pending, 6 year old female).

Isolates from sites other than blood or CSF

Joint fluid: 2 *Staphylococcus aureus*, 1 *Streptococcus* Group A.

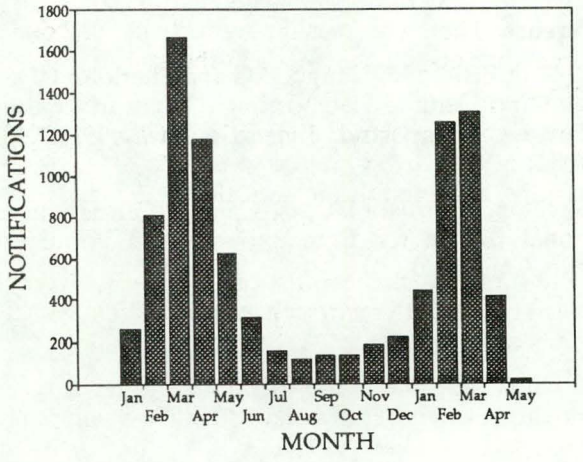
Other: 1 *Klebsiella oxytoca*.

National Notifiable Diseases Surveillance System, 18 April to 1 May 1993

This period 2,495 reports of notifiable diseases were received (Tables 4, 5 and 6 and Figure 6).

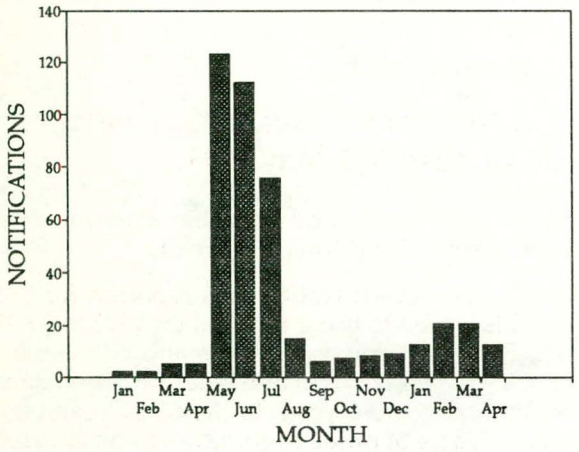
- **Ross River virus infection** was reported for 373 cases this period to bring the total for 1993 to 3,602 cases (Figure. 4). There were 198 males, 174 females and sex was unknown in one case. Reported ages ranged from the 0-4 years to the 80-84 years age groups. Dates of onset were recorded as January in 4 reports, February in 12, March in 91, April in 265 and June in one. Reports were for cases in statistical divisions in many areas of rural and urban Australia.
- **Dengue** activity has increased with 30 cases being reported this period. This brings the total for the

Figure 4. Notifications of Ross River virus infection, January 1991 to May 1993, by month of onset



year to 74 (Figure 5). One case was reported from Brisbane, 19 from Townsville, 9 from Charters Towers and one from Cairns. This represents a major extension of the epidemic of dengue over the last 2 years (see also *CDI* 1993;**17**:182-183, and this issue, page 215). The National Notifiable Diseases Surveillance System does not collect information on type. The cases comprised 19 males and 11 females. Ages ranged from the 10-14 to the 65-69 years age groups, with the modal group for sexes combined being 25-29 years with 8 cases. For cases reported this period, onset dates were recorded as January (1), February (3), March (14) and April (12).

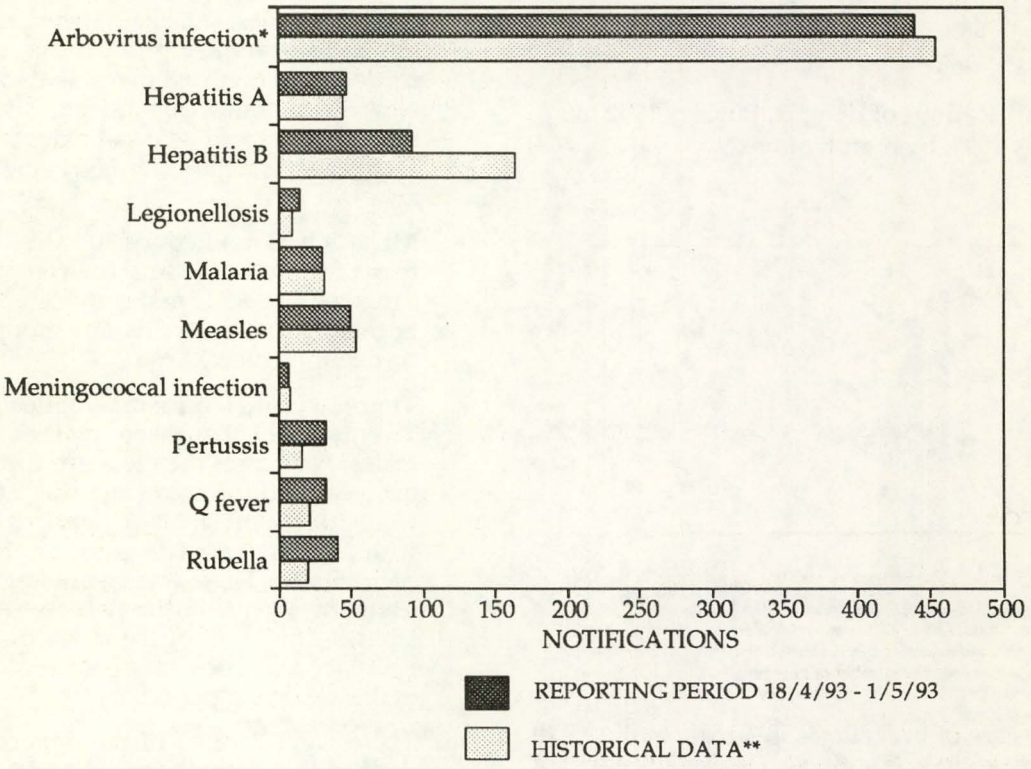
Figure 5. Notifications of dengue, January 1992 to May 1993, by month of onset



- A single case of **brucellosis** in a male in the 25-29 years age group was notified. He was a resident of suburban Melbourne.
- This period 60 notifications of **gonococcal infection** were received. They were for 46 males, 13 females and sex was not recorded in one case. There was one female in the 10-14 years age group.
- ***Haemophilus influenzae* type b infection** was notified for 18 cases. There were 11 males and 7 females, of these, 9 (6 males and 3 females) were aged less than one year and 16 were aged less than 5 years. There was one apparent cluster of 2 cases occurring on the same day in the same postcode area.
- Forty-eight reports of **hepatitis A** were received. Ages ranged from the 0-4 to the 65-69 years age groups, the modal age was the 25-29 years age group with 9 cases. The cases comprised 24 males and 24 females. Ten of the cases occurred in residents of the Brisbane statistical division, with other cases being in 15 metropolitan and rural statistical divisions.
- A single case of **hydatid infection** was reported in a male from rural Queensland in the 50-54 years age group.
- There were 15 reports of **legionellosis** received this period. Seven cases were from the Melbourne statistical division and 5 were from the Sydney statistical division. There were 11 males and 4 females. The average age of cases was 58.6 years, ranging from the 35-39 to the 80-84 years age groups. Two were from the same postcode area in Sydney with onset dates separated by an interval of 3 days and 3 were from adjacent postcode areas in Melbourne with onset dates over a 2 day period.
- Eleven cases of **leptospirosis** were notified, all bar one were males, and ages ranged from the 20-24 to the 60-64 years age groups (one with age unrecorded). All occurred in residents of rural areas of New South Wales, Queensland, Tasmania and Victoria. Four cases had onset dates within 5 days of each other (3 on the same day) in the same postcode area.
- **Malaria** was notified for 30 cases this period. Of these, 8 were in the 'malaria receptive' zone. The cases comprised 21 males and 9 females with ages in the 0-4 to 45-49 years age groups (age not recorded for 2 cases).
- There were 51 **measles** notifications received. Twenty-six of these were males and 25 were females. For 8 cases the age was recorded as less than one year and the mean age was 9.0 years. There were 2 apparent small clusters in 2 postcode areas with 2 cases each with onset on the same day in each cluster. There was an outbreak of 26 cases in southern Tasmania (in Hobart and surrounding areas). The onset of the index case was in early February and cases have been recorded to the end of this reporting period.
- There were 8 cases of **meningococcal infection** notified this period, 2 males and 6 females. Ages ranged from the 0-4 years to the 70-74 years age groups. There was no apparent clustering of cases.
- **Pertussis** was notified for 33 cases, 12 males and 21 females. One of these cases was less than one year of age and 3 were aged less than 5 years. There were 4 apparent clusters in separate postcode areas with 2-6 cases each, onset dates were on the same day or were spread over a period of 12 days.
- There were 34 cases **of Q fever** reported this period, 27 males and 14 females. Ages ranged from the 10-14 years to the 60-64 years age groups. There have been 230 notifications of Q fever so far this year, compared with 147 by this time last year.
- Forty-one reports of **rubella** were received. Sex was recorded as male for 27 cases and female for 14. Ages ranged from the 5-9 to the 60-64 years age groups and the mean age was 18.4 years. There were 9 reports for females in the 15-44 years age group. There were 3 apparent clusters in separate postcode areas with 2 cases each. Onset dates were on the same day or separated by intervals up to 7 days.

- Sixty notifications of **syphilis** were received. Thirty-four were males and 26 were females. Two cases were aged less than 10 years, one being less than one year.
- There were 24 cases of **tuberculosis** notified this period. One case was aged less than one year and the remainder were in the 35-39 to the 75-79 years age groups (5 with age unrecorded). Males accounted for 9 cases, females for 14 and sex was not recorded in one case.

Figure 6. Selected National Notifiable Diseases Surveillance System reports, and historical data **



* Includes Ross River virus and Dengue

** The historical data are the averages of the number of notifications in 6 previous 2-week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 4. Notifiable Diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation for the reporting period 18 April to 1 May 1993

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[1] This Period 1993	This Period 1992	Year to Date 1993	Year to Date 1992
Diphtheria	0	0	0	0	0	0	0	0	0	0	7	4
Measles	1	10	0	7	2	25	5	1	51	20	404	325
Mumps	0	0	NN	NN	NN	NN	0	0	0	0	0	10
Pertussis	0	12	0	6	9	1	2	3	33	15	493	167
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0
Rubella[2]	0	2	0	24	4	0	11	0	41	21	1087	164
Tetanus	0	0	0	NN	0	0	0	0	0	2	3	6

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

2. NT, Tas: CRS only; ACT, NSW, Qld: rubella only.

NN Not Notifiable.

Table 5. Other Notifiable Diseases[1], for the reporting period 18 April to 1 May 1993

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This Period 1993	This Period 1992	Year to Date 1993	Year to Date 1992
Arbovirus infection (NEC)[3]	0	8	NN	20	0	0	9	0	37	18	270	136
Ross River virus infection	0	29	23	186	33	NN	89	13	373	526	3602	3544
Dengue	0	-	0	30	-	NN	0	NN	30	4	74	11
Campylobacteriosis[4]	3	-	11	78	32	9	35	33	201	329	2695	2805
Chlamydial infection (NEC)[5]	1	NN	10	76	0	15	25	0	127	446	1862	2554
Donovanosis	0	NN	0	1	NN	NN	0	0	1	2	14	22
Gonococcal infection[6]	1	8	13	18	0	0	6	14	60	139	934	960
Haemophilus influenzae b infection[7]	0	7	NN	2	2	0	7	0	18	8	145	128
Hepatitis A	0	8	4	24	4	0	5	3	48	71	657	707
Hepatitis B	2	0	1	60	1	1	0	27	92	147	777	1558
Hepatitis C	0	0	8	151	NN	4	44	0	207	259	1683	2537
Hepatitis (NEC)	0	0	0	1	0	0	0	NN	1	4	29	13
Legionellosis	0	5	0	0	1	0	9	0	15	31	60	67
Leptospirosis	0	2	0	6	0	1	2	0	11	1	64	35
Listeriosis	0	0	NN	1	NN	0	1	0	2	3	16	14
Malaria	0	0	0	25	0	0	1	4	30	24	247	246
Meningococcal infection	0	3	0	2	1	0	1	1	8	10	74	56
Ornithosis	0	NN	0	0	0	0	5	0	5	3	34	35
Q fever	0	13	0	19	0	0	0	2	34	14	230	147
Salmonellosis (NEC)	2	31	14	28	13	4	18	40	150	219	1939	2167
Shigellosis[4]	0	-	3	5	1	0	4	9	22	30	344	212
Syphilis	0	15	17	21	0	0	2	5	60	136	704	847
Tuberculosis	1	2	0	2	3	1	12	3	24	40	263	248
Typhoid[8]	0	0	0	0	0	0	0	0	0	0	17	23
Yersiniosis (NEC)[4]	0	-	0	5	3	0	2	0	10	30	160	254

1. For HIV and AIDS, see *CDI* 1993;17:193. For rarely notified diseases, see Table 6.
2. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
3. SA, Tas: includes Ross River virus and dengue.
   WA: includes dengue.
4. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
5. WA: genital only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. SA: only as 'bacterial meningitis'; meningococcal infection is separately notified; Tas: only as 'non-meningococcal meningitis'; Vic: epiglottitis and meningitis only.
8. NSW and Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 6. Rarely Notified Diseases[1] for the reporting period 18 April to 5 May 1993

DISEASES	Total This Period	Reporting States or Territories	Year to Date 1993
Botulism	0		0
Brucellosis	1	Vic	10
Chancroid	0		1
Cholera	0		2
Hydatid infection	1	Qld	13
Leprosy	0		4
Lymphogranuloma venereum	0		0
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 50 cases of each of these diseases were notified each year during the period 1987 to 1992.

Table 7. Laboratory reports by State or Territory of reporting laboratory for the reporting period 22 April to 5 May 1993, historical data[1], and total reports for the year

	STATE OR TERRITORY OF REPORTING LABORATORY						Total this fortnight	Historical data [1]	Total reported this year
	NSW	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA									
Measles virus	5	3	3		2		13	8.2	154
Mumps virus			1		1		2	.7	22
Rubella virus	1	11	2				14	5.0	486
HEPATITIS VIRUSES									
Hepatitis A virus	2	9	4			2	17	9.8	249
Hepatitis B virus	27	18	8		20	8	81	76.2	1,032
Hepatitis C virus	11	29	107			46	193	38.7	1,396
ARBOVIRUSES									
Ross River virus	1	23	27		15	12	78	112.7	1,034
Barmah Forest virus		3	2			5	10	13.7	99
Dengue not typed						3	3	2.5	14
MVE virus						1	1	.7	2
ADENOVIRUSES									
Adenovirus type 1			1				1	3.2	34
Adenovirus type 2	2				4		6	6.0	34
Adenovirus type 3	10		5		2		17	1.8	90
Adenovirus type 4			2		1		3	.7	52
Adenovirus type 5	1				2		3	1.0	16
Adenovirus type 8					2		2	.8	9
Adenovirus type 19					1		1	.3	1
Adenovirus type 26					1		1	.7	2
Adenovirus type 40	1						1	.2	7
Adenovirus not typed/pending	15		32		12	7	66	32.0	480
HERPES VIRUSES									
Herpes simplex virus type 1	13	30	37		35	17	132	108.5	1,681
Herpes simplex virus type 2	19	54	37		35	50	195	135.0	1,942
Herpes simplex not typed/pending	17				4	1	22	25.7	249
Cytomegalovirus	6	6	1		46	13	72	57.8	637
Varicella-zoster virus	7	12	8		18	7	52	18.0	391
Epstein-Barr virus	4	9	10		11	10	44	56.8	775
Herpes virus group - not typed			1			1	2	3.0	12
OTHER DNA VIRUSES									
Contagious pustular dermatitis (Orf virus)						1	1	.2	3
Parvovirus					5		5	2.5	56
PICORNA VIRUS FAMILY									
Coxsackievirus A9	3				1		4	.3	27
Coxsackievirus B1	1				1		2	1.0	52
Coxsackievirus B3					1		1	.3	5
Echovirus type 7	5				5		10	.2	81
Echovirus type 9	1				2		3	7.0	42
Echovirus type 11	2	1			5		8	.2	20
Echovirus type 14	2						2	.5	9
Echovirus type 15	1						1	.0	1
Echovirus type 22	1			1	1		3	.0	9
Echovirus type 25					1		1	.2	14

Table 7. Laboratory reports by State or Territory of reporting laboratory for the reporting period 22 April to 5 May 1993, historical data[1], and total reports for the year, continued

	STATE OR TERRITORY OF REPORTING LABORATORY						Total this fortnight	Historical data [1]	Total reported this year
	NSW	Qld	SA	Tas	Vic	WA			
Echovirus type 30					1		1	.0	4
Poliovirus type 1 (uncharacterised)	2						2	1.7	23
Poliovirus type 2 (uncharacterised)	3						3	1.5	16
Poliovirus type 3 (uncharacterised)	1						1	1.2	12
Poliovirus not typed/pending					2		2	3.2	2
Rhinovirus (all types)	3		1		18	3	25	17.7	289
Enterovirus not typed/pending	2	2			9	6	19	31.3	269
ORTHO/PARAMYXOVIRUSES									
Influenza A virus			5				5	17.3	48
Influenza B virus			3				3	2.3	25
Parainfluenza virus type 1					1		1	17.5	11
Parainfluenza virus type 2	1				4	1	6	5.5	21
Parainfluenza virus type 3	2	1	5		14		22	17.0	204
Parainfluenza virus typing pending					5		5	5.7	10
Respiratory syncytial virus	18	3	2		13	2	38	44.5	205
OTHER RNA VIRUSES									
HIV-1						2	2	1.5	35
HTLV-1						1	1	.3	5
Rotavirus	8				11	13	32	43.8	360
Reovirus (unspecified)	1						1	.7	3
Calici virus	1						1	1.3	6
Norwalk agent	1				1		2	.2	7
Small virus (like) particle	1						1	3.5	21
OTHER									
Chlamydia trachomatis not typed	11	60	15	2	4	46	138	88.3	1,209
Chlamydia psittaci					2		2	3.0	38
Chlamydia spp typing pending			1				1	.5	4
Mycoplasma pneumoniae	14	20	3	1	28	2	68	19.8	834
Coxiella burnetii (Q fever)	7	7				5	19	10.8	173
Streptococcus group A		12					12	.0	112
Bordetella pertussis					11		11	.0	59
Bordetella species		4					4	.0	91
Leptospira hardjo				1			1	.0	3
Treponema pallidum	16	1					17	.0	264
Toxoplasma gondii		2			1		3	.0	27
TOTAL	250	320	323	5	359	266	1,523	1,072.0	15,610

1. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 8. Laboratory reports by clinical information for the reporting period 22 April to 5 May 1993

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
MEASLES, MUMPS, RUBELLA													
Measles virus					1			6				6	13
Mumps virus												2	2
Rubella virus								2		1		11	14
HEPATITIS VIRUSES													
Hepatitis A virus							6					11	17
Hepatitis B virus						1	10					70	81
Hepatitis C virus							7					186	193
ARBOVIRUSES													
Ross River virus								18		21		39	78
Barmah Forest virus								2		1		7	10
Dengue not typed												3	3
MVE virus	1												1
ADENOVIRUSES													
Adenovirus type 1					1								1
Adenovirus type 2	1				2	1		1				1	6
Adenovirus type 3					4	4		1	4			4	17
Adenovirus type 4					2				1				3
Adenovirus type 5					3								3
Adenovirus type 8									2				2
Adenovirus type 19									1				1
Adenovirus type 26												1	1
Adenovirus type 40						1							1
Adenovirus not typed/pending					18	33		1	7			7	66
HERPES VIRUSES													
Herpes simplex virus type 1					3			70	7		43	9	132
Herpes simplex virus type 2								64	1		118	12	195
Herpes simplex not typed/pending	1				1			11	3		1	5	22
Cytomegalovirus			1	4	18	1	2					46	72
Varicella-zoster virus	1							44				7	52
Epstein-Barr virus					2		2	1		4		35	44
Herpes virus group - not typed	1							1					2
OTHER DNA VIRUSES													
Contagious pustular dermatitis (Orf virus)								1					1
Parvovirus								2		1		2	5
PICORNA VIRUS FAMILY													
Coxsackievirus A9		1	1					1				1	4
Coxsackievirus B1												2	2
Coxsackievirus B3					1								1
Echovirus type 7		4	1			2						3	10
Echovirus type 9		2						1					3
Echovirus type 11		1	1		2	1			1			2	8
Echovirus type 14						1		1					2
Echovirus type 15												1	1
Echovirus type 22					2							1	3

Table 8. Laboratory reports by clinical information for the reporting period 22 April to 5 May 1993, continued

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
Echovirus type 25						1							1
Echovirus type 30		1											1
Poliovirus type 1 (uncharacterised)					1							1	2
Poliovirus type 2 (uncharacterised)												3	3
Poliovirus type 3 (uncharacterised)						1							1
Poliovirus not typed/pending					2								2
Rhinovirus (all types)					20							5	25
Enterovirus not typed/pending	1	3			7	1			1			6	19
ORTHO/PARAMYXOVIRUSES													
Influenza A virus					2							3	5
Influenza B virus					2					1			3
Parainfluenza virus type 1					1								1
Parainfluenza virus type 2					5							1	6
Parainfluenza virus type 3					19							3	22
Parainfluenza virus typing pending					5								5
Respiratory syncytial virus					37							1	38
OTHER RNA VIRUSES													
HIV-1												2	2
HTLV-1												1	1
Rotavirus						32							32
Reovirus (unspecified)												1	1
Calici virus						1							1
Norwalk agent						2							2
Small virus (like) particle						1							1
OTHER													
Chlamydia trachomatis not typed					2				1		125	10	138
Chlamydia psittaci					1							1	2
Chlamydia spp typing pending												1	1
Mycoplasma pneumoniae			1		38			1		1		27	68
Coxiella burnetii (Q fever)												19	19
Streptococcus group A					1							11	12
Bordetella pertussis					11								11
Bordetella species					4								4
Leptospira hardjo												1	1
Treponema pallidum												17	17
Toxoplasma gondii												3	3
TOTAL	6	12	5	4	218	84	27	229	30	30	287	591	1523

Table 9. Laboratory reports by contributing laboratories for the reporting period 22 April to 5 May 1993

STATE OR TERRITORY	LABORATORY	REPORTS
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	159
	Prince Henry/Prince of Wales Hospitals, Sydney	4
	Royal Alexandra Hospital for Children, Camperdown	33
	South West Area Pathology Service, Liverpool	50
	Tamworth Laboratory, New England Pathology	4
Queensland	Queensland Medical Laboratory, West End	299
	State Health Laboratory, Brisbane	21
South Australia	Institute of Medical & Veterinary Science, Adelaide	323
Tasmania	Northern Tasmanian Pathology Service, Launceston	5
Victoria	Fairfield Hospital, Melbourne	218
	Microbiological Diagnostic Unit, University of Melbourne	4
	Royal Children's Hospital, Melbourne	137
Western Australia	Princess Margaret Hospital, Perth	30
	State Health Laboratory Services, Perth	236
TOTAL		1523