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Communicable Diseases Intelligence

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Contents:

Editor *Dr Robert Hall*

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

Reports were processed in two separate lots this period. The first was for the 1289 reports received as usual this reporting period (15-28 March 1990). The second was for the 581 reports received for the State Health Laboratory, Brisbane (SHLB), which cover the period May 1989 to January 1990. The tabulated data have been modified to present these data separately. An additional table has been included which shows pathogen identification by date of sample collection for the SHLB, and the pathogen identifications by clinical information have been tabulated with and without those reports. Updated tables detailing pathogen identifications by month for 1989 and incorporating the SHLB data will be published in the near future.

Six cases of psittacosis were reported. One case was in a 69-year-old man who keeps pigeons. He suffered a chest infection with fever, aches and dark urine.

There were 91 reports of rubella, 82 from the SHLB. Cases from the SHLB included a 19-year-old pregnant woman and a 23-year-old pregnant woman. The receipt of these reports has served to highlight again the high number of rubella cases reported since August 1989.

Other Editorial Staff: Ms Jenny Hargreaves, Ms Evon Bowler, Dr Lance Sanders.

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

- Figures given may be subject to revision.

There were 24 reports of Q fever, 22 from the SHLB. Occupational details indicating exposure to animal carcasses were provided for 11 of the patients; 7 were employed as meat workers, 2 as graziers/stationhands, one as a farmer and one as an abattoir worker.

A report was received of a case of coxsackie B3 virus infection in a one-month-old boy who developed meningitis. The virus was isolated from a cerebrospinal sample.

Enterovirus type 71 was isolated from a faeces sample of a four-month-old girl who was admitted to hospital with overwhelming sepsis and later died. No other organism was isolated. At autopsy, there was evidence of myocarditis, and unusual manifestation of this infection.

There were 143 reports of Ross River Virus; 120 of these were from the SHLB (55 in January and 22 in February). Locations were provided for 126 cases: Townsville (QLD) - 55, Rockhampton (QLD) - 18, Toowoomba (QLD) - 12, Cairns (QLD) - 10, Darwin (NT) - 4, Brisbane (QLD) - 3, Mackay (QLD) - 2, Northern NSW - 2, Sydney (NSW) - 2, Kununurra (WA) - 2, and one each from Lismore (NSW), Western Queensland, Nathalia (VIC), Shepparton (VIC), Swan Hill (VIC), Moulamein (NSW), Wyndham (WA), Mt Isa (QLD), Newcastle (NSW), Maryborough (QLD), Gympie (QLD), Western NSW, Pinjarra (WA), Halls Creek (WA), Binningup (WA?), and an unspecified place in NSW.

There were 22 reports of dengue fever, 21 from the SHLB. The identifications included six dengue-3 and two dengue-1 viruses. The cases included Australians who had been travelling in Fiji, Papua New Guinea, India and unspecified areas in the Pacific Islands and Southeast Asia.

The SHLB reported 10 cases of Barmah Forest disease. The Barmah Forest virus, an arthropod-borne flavivirus, has only recently been recognised as a human pathogen, and a specific diagnostic test has only recently been developed for it. The cases were reported from Bundaberg, Northern NSW, Cairns, Brisbane (3), Townsville, Rockhampton, Mackay and an unknown location in NSW. The patients were males aged 41(2), 39, 38 and 32, and females aged 51(2), 47, and 21 (one not stated). No clinical information was available for 5 of the patients. The other 5 experienced general malaise and/or mild fever (1), skin and/or mucous membrane disease (1) and muscle and/or joint disease (4).

A diagnosis of Kunjin virus disease was made by the SHLB. The patient was a 43-year-old Cairns woman who had experienced muscle and/or joint disease. (This virus is also a flavivirus, closely related to the Murray Valley Encephalitis virus. It is probably transmitted from bird reservoirs by mosquito vectors, probably *Culex annulirostris*. A small number of cases is reported in most years.)

A case of Sindbis virus has been reported to the Viruses Reporting Scheme for the first time since 1982. The patient was a 55-year-old woman from Toowoomba. She had experienced general malaise and/or mild fever and muscle and/or joint disease. (The Sindbis virus is an alphavirus, presumably transmitted from birds or other reservoir animals, by mosquitos, possibly *Culex annulirostris*.)

OVERSEAS BRIEFS:

1. CHOLERA IN MALAWI

A report has been received of an outbreak of cholera which began in Malawi in November. To 30 March 1990, 10,350 cases had been reported and there had been 489 deaths. No details on the affected areas are presently available. Control measures are being undertaken.

2. CHOLERA IN ZAMBIA

Recent reports indicate that the large numbers of cases of cholera are still occurring in Zambia. During the week 6 to 12 March, there were 151 cases and 10 deaths.

AUSTRALIAN ENCEPHALITIS FATALITY

(Based on information received from Dr R. Lugg, Principal Medical Officer for Public Health, and Tony Wright, Medical Entomologist, Health Department of Western Australia, and Dr David Smith, Princess Margaret Hospital, Perth.)

Western Australia has reported a fatal case of Australian Encephalitis, a rare, mosquito-borne viral disease. It occurred in a 17-month-old Aboriginal boy who was most probably infected while living in a community 15km Southeast of Halls Creek in the Kimberley region in the North of the State.

The boy's illness was complicated by a preceeding *Haemophilus influenzae* type B meningitis. This responded to antibiotic treatment, but the boy died in Princess Margaret Hospital in Perth in mid-March.

The diagnosis was made serologically. IgM to Murray Valley encephalitis virus was found, and the IgG titre was 1:160. Brain tissue was not made available for attempting virus isolation.

The Health Department of Western Australia issued a press release to announce the death of the boy and to warn Kimberley residents to take precautions against mosquitos. A team was also sent to the Halls Creek area to investigate the transmission of the disease in this case.

The boy had been infected in late February, following a period of heavy rains and subsequent high mosquito activity. It has not been possible to determine the mosquito vector responsible, because there may have been changes in the mosquito populations during the weeks between the time of infection and the time of investigation. The vector may have been either *Aedes normanensis,* which was not found during the investigation, but has been detected in the Halls Creek area in previous years, or *Culex annulirostris*, which was found during the investigation, and which is thought to be the usual vector of this disease. Ultra-low volume fogging was used to apply insecticide around Halls Creek and the community to reduce the adult mosquito population and the chance that further cases would occur.

A few cases of Australian Encephalitis occur in the North of Australia each year. This was the first case in Western Australia this year, and only the second death known to have been caused by this disease in the State. Further details of this case will be published as available.

AUSTRALIAN ENCEPHALITIS - PREDICTION OF EPIDEMICS

Australian encephalitis (AE) is a mosquito-borne viral disease which occurs as infrequent but severe epidemics in Australia. Prodromal symptoms are generally non-specific, and include fever, headache, nausea and vomiting (1). Less frequently observed symptoms include dizziness, myalgia and photophobia Later, evidence of brain dysfunction may present, including drowsiness, irrational behaviour, disorientation, inability to concentrate, ataxia, and speech disturbances.

Mortality rates can be high. In the 1917 and 1918 epidemics, a case fatality rate of 94 out of 134 was reported (70%) (2). In the 1974 epidemic, there were 58 cases and 13 deaths, a mortality rate of 22%. Of those who survived, about 50% suffered permanent neurological sequelae, with physical and/or intellectual handicap. Treatment is largely supportive, although antibiotics are sometimes used prophylactically or to treat secondary bacterial infections.

Most epidemics occur in the south eastern parts of Australia but have also been reported from Queensland. In addition, cases of AE occur sporadically in the northern parts of Australia in interepidemic years. Most cases present in the period January to April. The aetiological agents of AE are Murray Valley encephalitis virus (MVEv) and rarely, kunjin virus. Both are flaviviruses related to the Japanese encephalitis, dengue and yellow fever viruses.

Australian encephalitis is a zoonotic disease, with aquatic birds, particularly some species of heron, as the primary vertebrate hosts. *Culex annulirostris*, a mosquito which breeds in fresh water is the major vector, and although MVEv has also been isolated from *Aedes normanensis* (3), the role of this species in disease transmission is uncertain.

The first comprehensive report of an AE outbreak was published in 1919 (2). This report published details of the 1917 and 1918 epidemics of the disease, then known as X-disease. Since 1917 there have been 7 major epidemics (more than 10 cases) of AE (Table 1). These occurred in 1917, 1918, 1922, 1925, 1951, 1974 and 1981. In 5 of these, a large proportion of cases was centred on the Murray-Darling basin.

Almost 40 years ago it was postulated that epidemics of AE might follow excess rainfall in the Darling River catchment area (4) or in the Gulf of Carpentaria region (5) in the previous spring. The implication was that high rainfall was a prerequisite to favourable waterfowl and mosquito breeding patterns, and hence virus transmsission. However, although all epidemics in the Murray-Darling basin have been preceded by high rainfall in the Darling basin, the converse does not apply. Less than one third of years of high spring rainfall were followed by epidemics of AE in south eastern Australia.

Table 1: Numbers of cases of Australian Encephalitis since 1917 by State*

YEAR	NSW	VIC	SA	QLD	NT	WA	TOTALS
1917	70			44			114
1918	49	13		5			67
1922				75			75
1925	10			11			21
1951	10	34	4				48
1956		3					3
1969						1	1
1971	1			1			2
1974	5	27	10	10	5	1	58
1978						8	8
1979						2	2
1981				2	1	8	11
1984						2	2
1986				1		1	2
1987				1	1	1	3
1988				1	3		4
1989						1	1
1990**						1	1
TOTALS	145	77	14	151	10	26	423

* There were no cases in the years not included in the table.

** As at 30/3/90

Forbes (6) later postulated that 2 consecutive summers with above average rainfall might be a more accurate predictor of AE activity. The Forbes hypothesis relies on rainfall patterns in the 4 major water catchment areas of central - eastern Australia. In brief, he postulates that rainfall in all 4 watersheds will exceed the 7th decile in one or both quarters (Oct-Dec, Jan-March) of the previous summer and the final quarter of the year preceding an outbreak. A shortcoming of the Forbes hypothesis is the time taken to analyse rainfall data for the Oct- Dec Quarter. This limits its usefulness as a predictor of AE activity, as most epidemics begin in early January.

More recently attempts have been made to correlate a phenomenon known as the Southern Oscillation (SO) with AE epidemics (7). The SO is a relationship between deviations in barometric pressure measurements made in Tahiti and Darwin from long term means (measured since 1882). The Southern Oscillation Index (SOI), a mathematical derivation of these data, is calculated on a monthly basis and has been used to develop methods for prediction of rainfall in northern and eastern Australia in spring and early summer. In the simplest terms a positive SOI is correlated with above average rainfall subsequently. Conversely, a negative SOI is associated with drier than normal conditions. Nicholls (7) has used the mean spring Darwin barometric pressure as an indicator of the SOI, and to estimate the likelihood of an AE epidemic. In years where the mean

spring barometric pressure in Darwin was ≥1010 hPa, essentially no cases of AE have been recorded in south-eastern Australia in the following summer. As a positive predictor, however, the correlation between Darwin barometric pressure and AE cases is not as good. In only 7 of 22 years where the Darwin spring barometric pressure was less than 1010 hPa have there been cases of AE reported. Nevertheless, the Nicholls theory does give some indication of those years in which greater emphasis should be placed on vector control and public education.

The SOI itself may also be used as a predictor of the likelihood of AE activity in south-east Australia (Table 2). This table shows all those years since 1917 in which AE has been reported from NSW, Victoria or SA, and those years in which the mean SOI for the previous spring has been >6.0. The table shows that in 6 of the 8 years since 1917 in which the mean SOI for the previous spring has exceeded 9.0, AE has been reported from at least one of these states. The anomalous years were 1965 and 1989. Conversely, in only one of 66 years (1917) in which the mean SOI for the previous spring has been ≤9.0 has cases of AE been reported from those states (assuming that no cases occur for the remainder of 1990).

Table 2: Southern oscillation indices and epidemics of Australian Encephalitis

YEAR	NO OF CASES OF AE *	MEAN SOI FOR SEPT-NOV OF PREVIOUS YEAR
1917	70	6.83
1918	62	21.8
1922	0	7.86
1923	0	6.6
1925	10	9.27
1929	0	6.73
1930	0	6.33
1939	0	7.63
1944	0	6.4
1948	0	6.26
1951	48	12.27
1956	3	15.43
1963	0	6.6
1965	0	10.17
1969	0	-7.4
1971	1	14.2
1974	42	18.43
1975	0	6.63
1978	0	-11.97
1979	0	-2.1
1981	0	-3.0
1984	0	4.6
1986	0	-2.1
1987	0	-3.87
1988	0	-5.8
1989	0	18.7
1990**	0	3.9

* AE cases tabulated for NSW, VIC and SA only

** To 30/3/90

Thus, in agreement with the Nicholls hypothesis, the spring SOI has good negative predictive value. However, the spring SOI value does appear to have a higher positive predictive value.

On the basis of this model, it is unlikely that cases of AE will occur in the Murray-Darling basin during the remainder of 1990 (Table 3). From January to June 1989, 3 month moving averages for SOIs were high. From about that time they gradually decreased, with spring mean SOI being 3.9.

Table 3: Southern oscillation indices January 1989 to date

	MONTH	SOI
1989	Jan	12.7
	Feb	8.5
	Mar	5.5
	Apr	18.1
	May	15.1
	Jun	6.1
	Jul	8.9
	Aug	-5.6
	Sep	5.8
	Oct	7.8
	Nov	-1.8
	Dec	-6.3
1990	Jan	-1.9
	Feb	-18.4

Currently, SOI values are negative, and although future values cannot be predicted, it would appear that if present trends continue, the risk of an AE epidemic during the 1990/91 summer is minimal.

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HEPATITIS C ANTIBODY TESTING

(Based on Infectious Disease Alert Vol.9/No.7, 1 January 1990)

Non-A, non-B hepatitis remains the most common serious consequence of blood transfusion.

Approximately 50% of patients infected in this way have evidence of chronic hepatitis and 20% of these patients have evidence of cirrhosis. In addition, non-A, non-B hepatitis may be causally related to hepatocellular carcinoma.

Until recently, it was difficult to define the agent of non-A non-B hepatitis. However, with the development of an assay based on a cloned portion of the hepatitis C virus, the ability to diagnose this disease either by radio- or enzyme-linked immune assays has been accomplished.

Alter and colleagues recently evaluated radioimmune assays for anti-HCV by analysing stored blood samples from prospective studies of transfusion associated hepatitis (1). A total of 15 patients with chronic transfusion associated non-A, non-B hepatitis were examined before and after their transfusions. The mean age of the patients was 51.8 years; 27% were younger than 50 and 67% were male.

Elevations in liver enzymes of the patients persisted for eight or more years. The presence of liver disease was confirmed by biopsy in 14 patients: 3 had chronic persistent hepatitis, 7 had chronic active hepatitis, and 4 had cirrhosis.

Each of the 15 patients with chronic hepatitis was negative for anti-HCV before transfusion; all became positive at varying times after transfusion.

Similar data were demonstrated in five patients with non-A, non-B hepatitis that resolved spontaneously within the first year after transfusion. Seroconversion to anti-HCV occurred in only 3 of the 5 patients with resolving disease compared to all those in the chronic disease group.

The mean interval to anti-HCV seroconversion was 21.9 weeks and the mean interval between onset of hepatitis and anti-HCV seroconversion was 15 weeks.

There was no consistent clinical or biochemical distinction between the two resolving cases in which anti-HCV failed to develop, and the three cases where it did.

As a group, patients with transient cases had more severe acute episodes, as manifested by higher mean alanine aminotransferase levels and more frequent jaundice. The relationship between the donor and recipient markers that might distinguish the presence of this infection was more difficult to determine. Overall, an anti-HCV positive donor was detected in 88% of the fully evaluable cases of non-A, non-B hepatitis. Of anti-HCV positive donors, 53% would have also been excluded (as donors) by the surrogate assays of either alanine aminotransferase elevations or the presence of anti-Hepatitis B.

CDI Editorial Comment

The test for anti-HCV can detect most cases of non-A non-B transfusion-associated hepatitis. The screening test, which is beginning to be routinely used to screen blood donations in Australia, quantitates antibody against what is probably a structural protein of the virus. The test has been shown to have high reliability, but until a confirmatory test is availble, it is expected that there will be some difficulties in interpreting the results of the tests.

Some cases of transfusion-associated non-A non-B hepatitis are still negative in all the hepatitis serological tests. This may be because the length of time taken for seroconversion can be so long that the diagnosis can be missed, suggesting that multiple tests may be required to establish the presence of the agent. Serology may also be negative if there is a further, as yet undiscovered agent involved in this form of hepatitis.

The problem of missed diagnoses due to slow seroconversion may be solved in the future by the development of hepatitis C antigen detection systems or by the use of techniques such as the polymerase chain reaction and *in situ* hybridisation to detect viral nucleic acids (2). The continued use of surrogate markers in screening for this or similar agents in blood products may also still be warranted in some countries.

Hepatitis C cases were reported to the CDI for the first time recently (Issue 90/5). Such reports are encouraged from clinicians and diagnostic laboratories. They can be made either through the Viruses Reporting Scheme or by direct correspondence. Clinicans and diagnostic laboratories may also notify hepatitis C to State and Territory health authorities as non-A, non-B hepatitis.

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LASSA FEVER: THE FIRST CONFIRMED CASE IMPORTED INTO CANADA

(Based on Canada Diseases Weekly Report 1989 15:193-198)

Introduction

Lassa fever (LF) is an acute zoonotic arenavirus disease of West Africa. Infection is mainly transmitted to humans from the multimammate rodent reservoir, *Mastomys natalensis*, in or around homes in rural areas (1-4). Person-to-person transmission does not readily occur as initially thought, and secondary infections among contacts of patients have not been reported in cases exported outside West Africa (3-5). The disease varies in severity and has a wide range of clinical manifestations including fever, headache, malaise, sore throat,

joint and low back pain, and weakness (5-7). Up to one third of patients develop bleeding, which, together with diarrhoea and fever of >39°C is highly associated with a fatal outcome in severe cases (6,7). An estimate based on the epidemiology and ecology of this virus suggests that as many as 100,000 to 300,000 Lassa infections with 5,000 deaths may occur annually in West Africa. Few cases are imported into countries outside West Africa. This is an account of the first confirmed case imported into Canada.

Case Report

The case was a 38-year-old Canadian male who had been living for 3 years with his wife and 3 children, ages 5, 7 and 10 years in Banyan, Nigeria. He was immunised against typhoid and yellow fever in 1985 and was receiving standard human immune globulin every 6 months.

On 17 December 1988 he felt ill while at a party, complaining of severe headache, malaise, nausea, high fever, and chills. The patient was given chloroquine, but did not respond for 2 days. His fever continued and he developed a sore throat and cough. He was treated with chloroquine, sulfadoxine and pyrimethamine (Fansidar) and trimethoprim and sulphamethoxazole (Septrin), with no improvement.

The patient was examined on 23 December 1988 at a hospital and found to be weak with inflamed conjunctiva and pharynx, but without exudate. There were no overt signs of haemorrhage. The lymph nodes were not enlarged and his chest and abdomen were clear. He had a leucocyte count of 3.9 X 10^9 /L with 48% neutrophils, 51% lymphocytes and 1% monocytes. His transaminases were: SGOT, 11 U/L and SGPT, 40 U/L; he also had albuminuria. Blood smears for malaria were negative and the patient was suspected of having LF and placed under observation.

He was seen 3 days later with pleuritic chest pains, dyspneoa, cough, rigors and papular eruptions on his back, and was admitted to hospital. The albuminuria persisted and the SGOT rose to 74. Titres to O and H antigens in the Widal test (for Typhoid fever) were 160 and 80, respectively, on 27 December and became 80 and 40, 2 days later. He was given a course of chloramphenicol, although the diagnosis of typhoid fever was unlikely. The patient started to improve on 28 December, with decreased fever and improved appetite; the rash, however, had spread to the trunk and extremities and became maculopapular. Improvement continued and he was discharged on 31 December.

Two days later he was re-admitted to hospital because he was feverish again and had developed bilateral orchitis. Serological tests excluded brucellosis but antibiotic therapy was changed to tetracycline. His temperature dropped within a few days and he was discharged again on 9 January 1989. For 2 weeks he felt quite weak but at follow-up he was well and the whole family returned to Toronto, Ontario on 31 January 1989.

Throughout this episode both his wife and 10-year-old daughter felt well and were asymptomatic. His 5-year-old son became ill on 27 December 1988 and developed jaundice; his 7-year-old daughter felt ill with fatigue and weakness 2 weeks later. Subsequent tests showed that the children had contracted hepatitis A (see below).

The entire family was examined following their return to Canada and have been well since.

Serological Findings

The earliest available patient's serum, collected on 10 January 1989, was tested by immunofluorescence technique (IFT) for antibodies to 6 viral haemorrhagic fevers using pooled spot-antigens prepared from suspensions of Vero cells. The pooled preparation included antigens for Crimean-Congo, Rift Valley, Ebola-Zaire, Ebola-Sudan, Lassa, and Marburg haemorrhagic fevers. The serum was found positive for specific IgG with a titre of 512, and the IF staining was compatible with LF antigen. These results were confirmed using monovalent LF antigen; no antibodies were detected with Ebola-Zaire and Rift Valley fever antigens used as controls. Specific LF IgM was positive, furthermore, at a dilution of 64.

A second serum obtained from this same patient on 8 February had an IgG titre of 256 to LF and an IgM titre of 16, a 4 fold decrease. LF specific IgM could not be detected 2 weeks later, or 67 days after onset of the disease, but the patient's IgG antibody titre remained at 256.

Sera also collected from the rest of the family on this same date had no detectable antibodies to either LF or any of the other 5 haemorrhagic fever antigens. The patient's wife and children were tested again on 21 April, more than 4 months after onset of his disease, and they remained seronegative for LF. Meanwhile, both the asymptomatic daughter, her brother and sister who fell ill in late December, 1988, had specific IgM for hepatitis A virus (HAV) up to 22 February 1989. The father and mother were seronegative for both IgG and IgM to HAV.

Discussion

This patient had been living in an area with LF activity. His last trip prior to returning to Canada was to Ethiopia, 2 months before his illness. He had several clinical manifestations compatible with the most predictive of LF in hospitalised cases in endemic areas(7). The provisional clinical diagnosis was serologically confirmed; the IgG titre of 512, IgM of 64, with subsequent seroconversion indicated by a decline of IgM, satisfy the criteria for case definition and specific diagnosis of the disease (The patient's recovery was uneventful except for the occurrence of orchitis; infection with mumps was excluded. Orchitis seems to be rare in LF; it was recorded in 1 out of 41 confirmed cases of LF(7).)

There was no evidence that the patient's wife and children had had prior infection with LF or had contracted it through their close contact with him, before or after the family's return. These findings are consistent with other episodes of exported cases. To this date, there has been no evidence of either LF disease or infection in several hundred contacts, including health-care workers, of cases imported from West Africa to Europe, the United States and Japan (5,8,9). Furthermore, in 1985, using rigorous nursing barrier techniques and strict patient isolation, there was no transmission of infection to health-care workers who attended to the management of a severe

case of LF in Sierra Leone (10). It should not be forgotten, however, that 27 cases of LF with 11 deaths were recorded up to 1977 among health-care workers handling cases in endemic areas (11).

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Canada Diseases Weekly Report Comment

This is the first confirmed case of Lassa fever (LF) imported into Canada; as such, this report is cause for both reassurance and caution. The reassuring news is the further evidence for low transmissibility of LF by the respiratory route, as documented by the fact that neither the children nor even the wife of the patient were infected by him. The reason for caution is the documentation of this disease in a Canadian, indicating that Canadian public health officials and clinicians must be vigilant and capable of quick response to imported cases of LF or other viral haemorrhagic fevers (VHFs).

The published Canadian Contingency Plan of 1978 (1) is no longer completely applicable. All the plastic patient isolator units, which were to have been used for both the care and the transport of patients with suspected or confirmed VHF, have been removed from the National Defence Medical Centre, selected armed forces bases, and selected Canadian airports. The plastic of these infrequently used units had deteriorated badly. The units were not replaced because of the technical difficulties of caring for patients in them, the fear that patient care would thus be compromised, and the mounting evidence that this type of containment was no longer necessary to protect others. As discussed in this report, the risk of nosocomially acquired LF among health-care workers attending to patients is much lower than was first feared. This may apply to the other VHFs as well.

Infection of laboratory workers by exposure to aerosolised body fluids of VHF patients remains a valid concern. Health and Welfare Canada hopes to have available in the future a mobile Pathoflex containment laboratory unit and High Efficiency Particulate Air (HEPA) filter masks. It is intended that when a

case of strongly suspected or proven VHF is reported, the mobile lab unit, the HEPA filter masks, and the personnel trained in their use will be sent to the institution where the patient is hospitalised. In the meantime, Canada is following the VHF guidelines of the U.S. Centers for Disease Control (CDC) (2) which have offered to make their mobile lab unit available to Canada when so requested, if the unit is not in use.

Severely ill patients with LF should be administered parenteral ribavirin, and oral ribavirin should be administered to high-risk contacts of LF cases. Although of unproven efficacy in the treatment of other VHFs, the use of ribavirin may be considered (2) for these diseases as well.

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CDI Editorial Comment

Only one case of Lassa Fever has ever been imported into Australia. This occurred when a woman arrived from Nigeria in 1985 in the convalescent stage of the disease.

Australia's policy for the management of patients with a Viral Haemorrhagic Fever (VHF) such as Lassa Fever is under review. Currently, Australia's National High Security Quarantine Unit (NHSQU) at Fairfield Hospital, Melbourne, is available for the safe management of patients with VHFs. Special transit isolators are kept in readiness around the country and suitable aircraft and trained medical/nursing teams can be quickly made available for aeromedical evacuation of a patient to Melbourne, if necessary.

Patients who have been in Africa in the previous three weeks could present with symptoms of Lassa Fever or other VHFs: pyrexia of unknown origin, shock and haemorrhage. Initially, the suspect case should be isolated and strict full barrier-nursing procedures instituted while consideration is given to transferring the patient to the NHSQU. Special precautions should be taken when handling specimens from such patients, as this has been the cause of most cases of secondary infections of VHFs.

The VHFs are quarantinable under the Quarantine Act 1908, and any suspected or confirmed case should be notified to the Director of the Commonwealth Department of Community Services and Health in your State or Territory, or to the Communicable Diseases Section in Canberra (phone 06 2898345). More details are available in the 'Handbook on measures to control quarantinable viral haemorrhagic fevers in Australia' (1986) published by the Department of Community Services and Health. Copies are available by phoning 06 2898221.

NOTICE - STD HANDBOOK

The National Health and Medical Research Council has released a revised edition of 'Handbook on sexually transmitted diseases'. This handbook updates previous information on the diagnosis and treatment of STDs and includes new chapters on the human immunodeficiency virus and the human papilloma virus. The NHMRC aims, in the publication of this handbook, to standardise the diagnosis and treatment of STDs and to provide an authoritative textbook for medical students.

The handbook is being distributed free of charge to subscribers of 'Australian Family Physician'. Other practioners who would like a copy should write to the NHMRC, PO Box 9848, Canberra Act 2601.

CDI INDEX 1990

Entries indicate issue/page number.
VRS = Viruses, Chlamydias, Coxiellas, Rickettsias and Mycoplasmas Reporting Scheme

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE
VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 15/3/90 TO 28/3/90

1. CODE 018 - MDU, UNI MELB(VIC)
2. CODE 019 - FAIRFIELD (VIC)
3. CODE 065 - STATE LAB(WA)
4. CODE 066 - PMH(WA)
5. CODE 110 - IMVS(SA)
6. CODE 111 - RCH(VIC)
7. CODE 112 - ICPMR(NSW)
8. CODE 113 - PHH POW(NSW)
9. CODE 114 - RACH(NSW)
10. CODE 115 - STATE LAB(QLD)
11. CODE 116 - WVH(ACT)

		019	065	066	110	111	113	114	115	116	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	18	0	0	4	0	14	0	36
0101	ADENOVIRUS TYPE 1	0	0	0	1	0	0	0	0	0	1
0103	ADENOVIRUS TYPE 3	1	0	0	3	0	0	0	0	0	4
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	1	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	0	0	0	9	0	1	0	0	11
0201	INFLUENZA A VIRUS	0	0	0	1	0	1	0	15	0	17
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	0	26	0	26
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	10	4	0	0	1	0	15
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	0	0	4	0	0	1	0	6
0303	PARAINFLUENZA VIRUS TYPE 3	1	1	0	0	13	0	0	17	0	32
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	0	2	3	0	5
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	1	2	2	3	8	0	16
0500	RHINOVIRUS (ALL TYPES)	4	1	0	8	15	0	2	0	2	32
0600	MYCOPLASMA PNEUMONIAE	1	2	0	7	2	2	0	6	1	21
0700	ORNITHOSIS-PSITTACOSIS	2	2	0	0	0	0	0	2	0	6
0903	COXSACKIEVIRUS B3	1	0	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	1	0	0	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	1	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	1	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	1	0	0	0	0	0	0	0	0	1
1200	MUMPS VIRUS	0	1	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	0	0	0	1	0	0	6	8
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	1	2	0	0	0	1	39	0	43
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	1	15	0	8	3	0	3	53	0	83
1303	VARICELLA-ZOSTER VIRUS	7	0	0	0	1	4	0	7	0	19
1306	HERPES SIMPLEX TYPE 1	31	19	0	19	2	0	0	0	1	72
1307	HERPES SIMPLEX TYPE 2	44	43	0	24	0	0	0	2	0	113
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	2	0	0	0	0	2
1401	COXIELLA BURNETII	0	1	0	1	0	0	0	22	0	24
1502	PICORNIA VIRUS - NOT TYPED = E	0	1	0	0	0	4	0	24	0	29
1521	MEASLES VIRUS	1	0	0	0	0	0	0	1	0	2
1522	RUBELLA VIRUS	1	1	0	6	1	0	0	82	0	91
1532	HEPATITIS B ANTIGEN	18	18	0	18	1	6	3	41	8	113
1535	HEPATITIS A ANTIBODY	2	1	0	4	0	1	1	0	0	9
1541	CHLAMYDIA A -*C. TRACHOMATIS	0	104	1	12	1	0	1	21	4	144
1553	LCM - LYMPHOCYTIC CHORIOMENING	1	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	25	0	2	7	6	3	2	39	0	84
1564	ROTAVIRUS	0	0	4	0	0	1	0	0	0	5
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	0	0	0	1	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	4	17	3	0	0	24
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	0	0	0	1	1
9906	BARMAH FOREST VIRUS	0	0	0	0	0	0	0	10	0	10
9991	SINDBIS VIRUS	0	0	0	0	0	0	0	1	0	1
9992	ROSS RIVER VIRUS	5	12	0	6	0	0	0	120	0	143
9995	DENGUE	0	1	0	0	0	0	0	21	0	22
9997	KUNJIN VIRUS	0	0	0	0	0	0	0	1	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	2	0	0	0	4	0	6
TOTAL		154	224	27	139	70	47	24	581	23	1289

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM STATE LAB, QLD (CODE 115)
BASED ON DATE OF SAMPLE COLLECTION

		89								90			
		3	5	7	8	9	10	11	12	1	2	3	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	0	0	1	0	0	0	0	9	4	14
0201	INFLUENZA A VIRUS	0	0	0	2	7	4	2	0	0	0	0	15
0203	INFLUENZA B VIRUS	0	0	0	0	10	13	3	0	0	0	0	26
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	0	0	0	1	0	0	0	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	1	0	0	0	0	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	4	4	6	1	0	2	0	17
0399	PARAINFLUENZA VIRUS TYPING PEN	0	0	0	0	0	0	0	0	1	2	0	3
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	1	0	0	1	0	3	3	8
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	3	0	2	0	1	0	0	6
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	2	0	0	0	0	2
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	0	0	0	0	0	0	0	22	17	39
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	0	1	13	11	6	10	11	1	0	53
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	1	1	3	0	1	0	1	7
1307	HERPES SIMPLEX TYPE 2	0	0	0	0	0	0	0	0	2	0	0	2
1401	COXIELLA BURNETII	0	1	0	1	7	4	4	1	2	2	0	22
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	0	0	0	0	0	0	13	11	24
1521	MEASLES VIRUS	0	0	0	0	1	0	0	0	0	0	0	1
1522	RUBELLA VIRUS	0	0	0	5	18	19	14	12	11	3	0	82
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	1	27	13	41
1541	CHLAMYDIA A - C. TRACHOMATIS	2	0	0	0	0	0	0	0	0	0	19	21
1556	CMV - CYTOMEGALOVIRUS	0	0	0	1	6	2	5	5	4	14	2	39
9906	BARMAH FOREST VIRUS	0	0	0	0	2	2	1	0	3	2	0	10
9991	SINDBIS VIRUS	0	0	0	0	0	1	0	0	0	0	0	1
9992	ROSS RIVER VIRUS	0	0	0	0	8	10	9	16	55	22	0	120
9995	DENGUE	0	0	1	0	5	3	5	3	4	0	0	21
9997	KUNJIN VIRUS	0	0	0	0	0	0	1	0	0	0	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	1	3	0	0	0	4
TOTAL		2	1	1	10	88	74	65	52	96	122	70	581

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1B - EXCLUDING REPORTS
FROM STATE LAB, QLD (CDOE 115)

PERIOD 15/3/90 TO 28/3/90

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	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	11	0	0	0	0	6	0	0	0	0	17
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
0103	ADENOVIRUS TYPE 3	0	3	0	0	0	0	0	0	0	0	0	3
0199	ADENOVIRUS TYPING PENDING	0	8	0	1	0	0	0	0	0	0	0	9
0201	INFLUENZA A VIRUS	0	2	0	0	0	0	0	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	0	14	0	0	0	0	0	0	0	0	0	14
0302	PARAINFLUENZA VIRUS TYPE 2	0	5	0	0	0	0	0	0	0	0	0	5
0303	PARAINFLUENZA VIRUS TYPE 3	0	11	0	0	0	0	0	0	0	0	0	11
0399	PARAINFLUENZA VIRUS TYPING PEN	0	2	0	0	0	0	0	0	0	0	0	2
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	8	0	0	0	0	0	0	0	0	0	8
0500	RHINOVIRUS (ALL TYPES)	0	28	0	1	0	1	0	0	0	0	0	30
0600	MYCOPLASMA PNEUMONIAE	2	11	0	0	0	0	0	0	0	0	0	13
0700	ORNITHOSIS-PSITTACOSIS	0	3	0	0	0	0	0	0	0	0	0	3
0903	COXSACKIEVIRUS B3	0	0	0	1	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	0	0	1	0	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	0	1	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	1	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	0	0	0	0	0	3	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	0	0	0	0	0	0	0	0	0	4	4
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	7	1	0	0	0	0	0	2	0	0	0	10
1303	VARICELLA-ZOSTER VIRUS	3	0	0	0	0	0	0	0	0	0	7	10
1306	HERPES SIMPLEX TYPE 1	0	2	0	0	0	0	0	0	1	0	46	49
1307	HERPES SIMPLEX TYPE 2	2	0	0	0	0	0	0	0	0	0	53	55
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	0	2	2
1401	COXIELLA BURNETII	1	0	0	0	0	0	0	0	0	0	0	1
1502	PICORNIA VIRUS - NOT TYPED = E	0	0	0	1	0	0	4	0	0	0	0	5
1521	MEASLES VIRUS	0	0	0	0	0	0	0	0	0	0	1	1
1522	RUBELLA VIRUS	3	0	0	0	0	0	0	0	0	0	3	6
1532	HEPATITIS B ANTIGEN	35	0	0	0	1	0	0	22	0	0	0	58
1535	HEPATITIS A ANTIBODY	3	0	0	0	0	0	0	6	0	0	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	0	2	0	0	0	0	0	0	0	0	0	2
1556	CMV - CYTOMEGALOVIRUS	4	5	1	2	1	0	1	4	0	1	0	19
1564	ROTAVIRUS	0	0	0	0	0	0	5	0	0	0	0	5
1599	ENTEROVIRUS TYPING PENDING	0	3	1	1	0	1	17	0	0	0	0	23
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	0	0	1	0	0	0	1
9992	ROSS RIVER VIRUS	6	0	0	0	0	0	0	0	0	0	4	10
9995	DENGUE	1	0	0	0	0	0	0	0	0	0	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	1	0	0	0	0	0	0	0	0	0	0	1
TOTAL		68	123	2	8	2	2	35	35	1	1	123	400

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2B - EXCLUDING REPROTS
FROM STATE LAB, QLD

PERIOD 15/3/90 TO 28/3/90

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	16	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	2	0	0	0	0	0	3	0	0	5
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	0	0	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	0	1	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	1	0	0	1	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	4	0	0	4
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	1	0	1	2
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	0	2	0	2
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	1	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	0	0	1	0	0	1
1200	MUMPS VIRUS	0	0	0	0	0	0	0	1	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	2	0	0	0	0	0	2	0	4
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	11	1	0	2	0	6	0	20
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	2	0	0	2
1306	HERPES SIMPLEX TYPE 1	2	13	0	0	0	1	1	6	0	23
1307	HERPES SIMPLEX TYPE 2	0	55	0	0	0	0	1	0	0	56
1401	COXIELLA BURNETII	0	0	0	0	0	0	1	0	0	1
1522	RUBELLA VIRUS	0	0	2	0	0	0	0	1	0	3
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	1	13	0	14
1541	CHLAMYDIA A - C. TRACHOMATIS	5	116	0	0	0	0	0	0	0	121
1553	LCM - LYMPHOCYTIC CHORIOMENING	0	0	0	0	0	0	0	1	0	1
1556	CMV - CYTOMEGALOVIRUS	2	0	1	0	0	0	6	17	0	26
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	0	0	0	0	1	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	0	1
9992	ROSS RIVER VIRUS	0	0	0	13	0	0	0	0	0	13
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	1	0	0	0	1
TOTAL		13	186	14	14	1	4	22	53	1	308

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1A - ALL LABORATORIES

PERIOD 15/3/90 TO 28/3/90

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	5	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	1	18	1	0	0	0	10	0	0	0	0	30
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
0103	ADENOVIRUS TYPE 3	0	3	0	0	0	0	0	0	0	0	0	3
0199	ADENOVIRUS TYPING PENDING	0	8	0	1	0	0	0	0	0	0	0	9
0201	INFLUENZA A VIRUS	2	8	0	0	0	0	1	0	0	0	1	12
0203	INFLUENZA B VIRUS	5	5	0	0	0	0	0	0	0	0	1	11
0301	PARAINFLUENZA VIRUS TYPE 1	0	14	0	0	0	0	0	0	0	0	0	14
0302	PARAINFLUENZA VIRUS TYPE 2	0	5	0	0	0	0	0	0	0	0	0	5
0303	PARAINFLUENZA VIRUS TYPE 3	4	21	0	0	0	0	0	0	0	0	0	25
0399	PARAINFLUENZA VIRUS TYPING PEN	0	5	0	0	0	0	0	0	0	0	0	5
0400	RESPIRATORY SYNCYTIAL VIRUS (R	1	14	0	0	0	0	0	0	0	0	0	15
0500	RHINOVIRUS (ALL TYPES)	0	28	0	1	0	1	0	0	0	0	0	30
0600	MYCOPLASMA PNEUMONIAE	2	17	0	0	0	0	0	0	0	0	0	19
0700	ORNITHOSIS-PSITTACOSIS	1	4	0	0	0	0	0	0	0	0	0	5
0903	COXSACKIEVIRUS B3	0	0	0	1	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	0	0	1	0	0	0	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	0	1	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	1	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	0	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	0	0	0	0	0	3	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	4	0	0	0	0	0	0	0	0	26	30
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	20	11	0	1	0	1	0	3	1	0	1	38
1303	VARICELLA-ZOSTER VIRUS	6	0	0	0	1	0	0	0	0	0	10	17
1306	HERPES SIMPLEX TYPE 1	0	2	0	0	0	0	0	0	1	0	46	49
1307	HERPES SIMPLEX TYPE 2	2	0	0	0	0	0	0	0	0	0	53	55
1399	HERPES VIRUS TYPING PENDING	0	0	0	0	0	0	0	0	0	0	2	2
1401	COXIELLA BURNETII	7	0	0	0	0	0	0	2	1	0	0	10
1502	PICORNIA VIRUS - NOT TYPED = E	0	12	0	1	0	2	14	0	0	0	0	29
1521	MEASLES VIRUS	0	0	1	0	0	0	0	0	0	0	1	2
1522	RUBELLA VIRUS	23	2	0	0	0	0	1	1	0	0	29	56
1532	HEPATITIS B ANTIGEN	35	0	0	0	1	0	0	63	0	0	0	99
1535	HEPATITIS A ANTIBODY	3	0	0	0	0	0	0	6	0	0	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	9	2	0	0	0	0	0	0	0	0	0	11
1556	CMV - CYTOMEGALOVIRUS	16	13	1	2	1	0	1	9	1	4	1	49
1564	ROTAVIRUS	0	0	0	0	0	0	5	0	0	0	0	5
1599	ENTEROVIRUS TYPING PENDING	0	3	1	1	0	1	17	0	0	0	0	23
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	0	0	1	0	0	0	1
9906	BARMAH FOREST VIRUS	5	0	0	0	0	0	0	0	0	0	1	6
9992	ROSS RIVER VIRUS	71	3	0	0	0	0	0	0	0	0	11	85
9995	DENGUE	15	1	0	0	0	0	1	0	0	0	1	18
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	5	0	0	0	0	0	0	0	0	0	0	5
TOTAL		233	207	4	9	3	5	52	85	4	4	187	793

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2A - ALL LABORATORIES

PERIOD 15/3/90 TO 28/3/90

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	3	0	0	0	0	0	0	3	0	0	6
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	0	0	0	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	0	0	1	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	1	0	0	1	0	2
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	0	4	1	0	5
0203	INFLUENZA B VIRUS	0	0	0	2	1	0	0	10	2	0	15
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	0	0	0	0	1	0	0	1
0303	PARAINFLUENZA VIRUS TYPE 3	0	0	0	0	0	0	0	6	1	0	7
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	1	0	0	1
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	1	0	1	2
0600	MYCOPLASMA PNEUMONIAE	0	0	0	0	0	0	0	0	2	0	2
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	0	1	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	0	0	0	1	0	0	1
1200	MUMPS VIRUS	0	0	0	0	0	0	0	0	1	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	2	0	0	0	0	0	0	2	0	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	12	0	0	0	0	0	0	0	0	13
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	11	11	2	0	3	8	10	0	45
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	2	0	0	2
1306	HERPES SIMPLEX TYPE 1	2	13	0	0	0	0	1	1	6	0	23
1307	HERPES SIMPLEX TYPE 2	0	57	0	0	0	0	0	1	0	0	58
1401	COXIELLA BURNETII	0	0	0	0	1	0	0	11	2	0	14
1522	RUBELLA VIRUS	0	0	2	5	5	0	1	16	6	0	35
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	1	13	0	14
1541	CHLAMYDIA A - C. TRACHOMATIS	5	128	0	0	0	0	0	0	0	0	133
1553	LCM - LYMPHOCYTIC CHORIOMENING	0	0	0	0	0	0	0	0	1	0	1
1556	CMV - CYTOMEGALOVIRUS	2	1	1	0	1	1	0	8	21	0	35
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	0	0	0	0	0	1	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	0	1	0	1
9906	BARMAH FOREST VIRUS	0	0	0	0	4	0	0	0	0	0	4
9991	SINDBIS VIRUS	0	0	0	0	0	0	0	1	0	0	1
9992	ROSS RIVER VIRUS	0	0	0	1	40	0	1	7	9	0	58
9995	DENGUE	0	0	0	0	0	0	0	4	0	0	4
9997	KUNJIN VIRUS	0	0	0	0	1	0	0	0	0	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	0	0	1	0	0	0	1
TOTAL		15	213	14	19	55	2	7	88	82	1	496

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 15/3/90 TO 28/3/90

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.
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	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	4	0	14	18	0	0	36
0101	ADENOVIRUS TYPE 1	0	0	0	0	1	0	1
0103	ADENOVIRUS TYPE 3	0	1	0	0	3	0	4
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	10	0	0	0	0	11
0201	INFLUENZA A VIRUS	1	0	15	0	1	0	17
0203	INFLUENZA B VIRUS	0	0	26	0	0	0	26
0301	PARAINFLUENZA VIRUS TYPE 1	0	4	1	0	10	0	15
0302	PARAINFLUENZA VIRUS TYPE 2	0	5	1	0	0	0	6
0303	PARAINFLUENZA VIRUS TYPE 3	0	14	17	1	0	0	32
0399	PARAINFLUENZA VIRUS TYPING PEN	2	0	3	0	0	0	5
0400	RESPIRATORY SYNCYTIAL VIRUS (R	5	2	8	0	1	0	16
0500	RHINOVIRUS (ALL TYPES)	2	19	0	1	8	2	32
0600	MYCOPLASMA PNEUMONIAE	2	3	6	2	7	1	21
0700	ORNITHOSIS-PSITTACOSIS	0	2	2	2	0	0	6
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	1	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	1	0	0	0	0	1
1018	ECHOVIRUS TYPE 18	1	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	1	0	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	1	0	1
1102	POLIOVIRUS TYPE 2	0	1	0	0	0	0	1
1200	MUMPS VIRUS	0	0	0	1	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	1	1	0	0	0	6	8
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	0	39	3	0	0	43
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	3	4	53	15	8	0	83
1303	VARICELLA-ZOSTER VIRUS	4	8	7	0	0	0	19
1306	HERPES SIMPLEX TYPE 1	0	33	0	19	19	1	72
1307	HERPES SIMPLEX TYPE 2	0	44	2	43	24	0	113
1399	HERPES VIRUS TYPING PENDING	0	2	0	0	0	0	2
1401	COXIELLA BURNETII	0	0	22	1	1	0	24
1502	PICORNIA VIRUS - NOT TYPED = E	4	0	24	1	0	0	29
1521	MEASLES VIRUS	0	1	1	0	0	0	2
1522	RUBELLA VIRUS	0	2	82	1	6	0	91
1532	HEPATITIS B ANTIGEN	9	19	41	18	18	8	113
1535	HEPATITIS A ANTIBODY	2	2	0	1	4	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	1	1	21	105	12	4	144
1553	LCM - LYMPHOCYTIC CHORIOMENING	0	1	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	5	31	39	2	7	0	84
1564	ROTAVIRUS	1	0	0	4	0	0	5
1571	ENTEROVIRUS TYPE 71 (BCR)	1	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	20	4	0	0	0	0	24
9903	NON-A, NON-B HEPATITIS	0	0	0	0	0	1	1
9906	BARMAH FOREST VIRUS	0	0	10	0	0	0	10
9991	SINDBIS VIRUS	0	0	1	0	0	0	1
9992	ROSS RIVER VIRUS	0	5	120	12	6	0	143
9995	DENGUE	0	0	21	1	0	0	22
9997	KUNJIN VIRUS	0	0	1	0	0	0	1
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	4	0	2	0	6
TOTAL		71	224	581	251	139	23	1289

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