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Communicable Diseases Intelligence

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

In this period (16 August to 29 August 1990) there were 1293 reports processed.

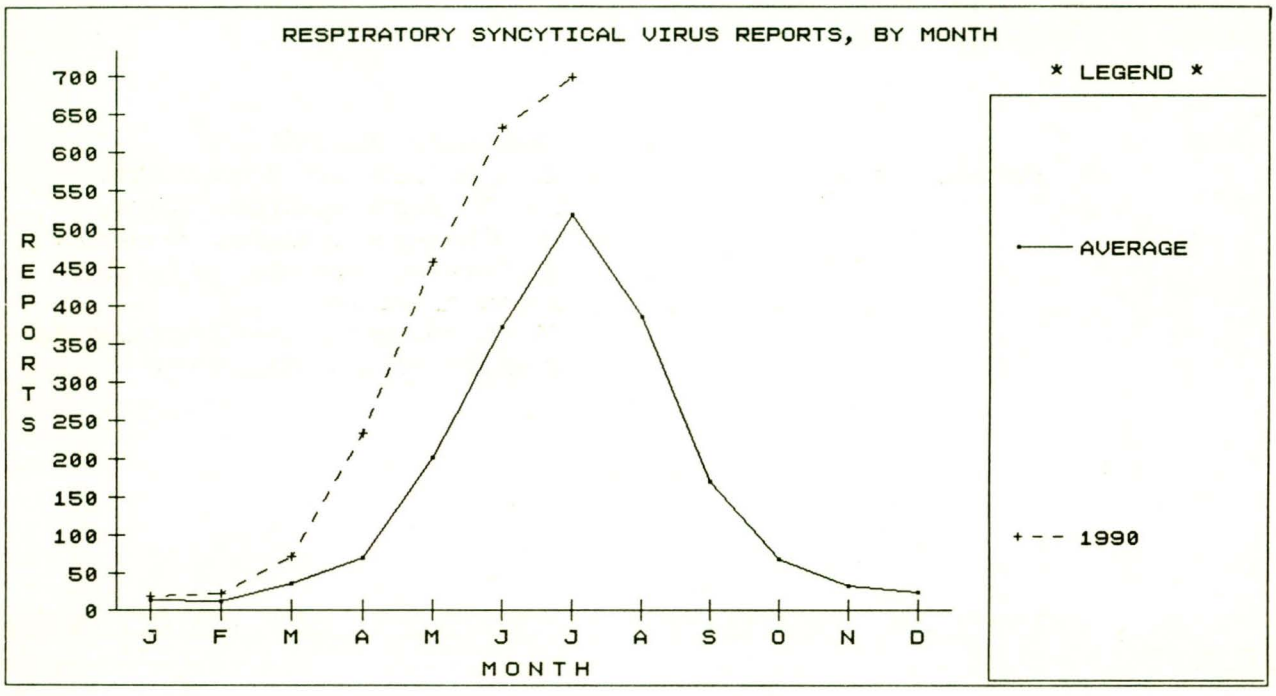
A total of 18 reports of influenza [types A, A(H3N2), B] were received for the period, 7 of which were isolates (4 from WA, 3 from QLD). All of the isolates were type A, with 3 being H3N2.

There was a single report of human herpesvirus-6, the newest of the human herpesviruses to be identified. This is only the second report of this virus to be received by CDI since it was added to the virus reporting list early this year. The patient was a 13-month-old male presenting with roseola infantum, the diagnosis was based on indirect immunofluorescence.

- The Bulletin is compiled and distributed by the Communicable Diseases Section, Communicable Diseases and Social Health Branch, Telephone: (062) 89 1555, Department of Community Services and Health.
- Contributions are solicited, and do not preclude later publication elsewhere.

- Figures given may be subject to revision.

There were a further 278 reports of respiratory syncytial virus, bringing the total for the year to date to 2350. This continues the above average reporting of RSV that was last mentioned in CDI 90/16. The updated graph of RSV cases appears below:



Two cases of Q fever (both males, aged 25 and 38 years) were reported. No exposure details were provided.

NON-VIRAL PATHOGEN REPORTS

Four positive blood culture reports were received for July from the Hobart Pathology Laboratory, all from patients with septicaemia. The following organisms were isolated:

- Staphylococcus aureus from a 57-year-old male patient
- Staphylococcus epidermidis from a 71-year-old female, and two male patients aged 33 and 63 years.

Pasteurella multocida was isolated from the pus of animal bite wounds of 4 female patients (aged 19, 60, 74 years and one age not known), two patients were bitten by dogs and the other two by cats.

OVERSEAS BRIEFS

1. CHOLERA IN MOZAMBIQUE

An outbreak of cholera has recently been reported from Mozambique. For the period 12 August to 18 August 1990 40 cases, with no fatalities, were recorded.

2. CHOLERA IN NEPAL - UPDATE

Further details of the recently reported cholera outbreak (CDI 90/16, 90/15) in Nepal have been supplied. As of 31 August 1990, cases reported for the Kathmandu valley totalled 575, with a single fatality.

3. CHOLERA IN ROMANIA- UPDATE

As of 23 August 1990, district totals were Tulcea, 63 cases; Galat, 12 cases and Braila, 10 cases. No deaths have been reported.

INFLUENZA UPDATE FROM THE NATIONAL WHO INFLUENZA REFERENCE CENTRE CSL - No 4 week commencing 3 SEPTEMBER 1990

Australia

Once again there is still no indication of significant influenza activity in Australia. The WHO Influenza Reference Centre has not received any further virus isolates, the last being a type A H3N2 from the Brisbane State Health Laboratory, isolated in late July.

New Zealand

Influenza activity in New Zealand has been on the increase during August, reaching its peak in the third week.

A total of 24 influenza isolates were received from Christchurch (20 Flu A type H3N2, 1 Flu A type H1N1, 3 Flu type B) and a total of 27 from Dunedin (17 Flu A type H3N2, 10 Flu type B).

The population group most affected were school-age children and younger adults. There were relatively few over 60's cases reported. Absenteeism rates in primary schools ranged between 30% - 50%, reaching as high as 90% in some schools.

LYME DISEASE

General

Lyme disease, currently the most prevalent arthropod-borne disease reported in the United States was described in that country for the first time in 1977 (1). It came to notice because of a clustering of children in Lyme, Connecticut who were initially thought to have juvenile rheumatoid arthritis. It is a multisystem disease with protean manifestations, and which affects the skin, nervous system, heart and joints. A characteristic skin rash seen in many of these patients was similar to that seen in a syndrome, first described in Europe in 1909, known as erythema migrans (or sometimes erythema chronicum migrans). Symptoms of other syndromes, separately described and named in Europe, bore a resemblance to symptoms seen in some US patients with Lyme disease.

In the early 1980s it was shown that all of these syndromes (Lyme disease, erythema migrans, acrodermatitis chronica atrophicans, Bannwarth's syndrome) were caused by infection with a previously unrecognised spirochaete, Borrelia burgdorferi. Lyme disease has now also been recognised in Asia. A similar syndrome has been described in Australia (see below). B. burgdorferi is transmitted by Ixodes ticks belonging to the Ixodes ricinus group. These include I. dammini in northeastern and midwestern United States, I. pacificus in western United States, I. scapularis in eastern United States, I. ricinus in Europe, and I. persulcatus in Asia. The lone star tick (Amblyomma americanum) may also be a vector in the US. After becoming infected by biting an infected host, the tick transmits the spirochaete during a subsequent blood meal from another vertebrate host. B. burgdorferi has been found in other species of tick, and in deer flies (Family: Tabanidae) and mosquitoes, but the importance of these in the transmission of Lyme disease is unknown. There is one recent report from the United States of disease transmission by a biting fly (2).

In north America, the animal hosts important in the life cycle of I. dammini are the white tailed deer and the white footed mouse, although only the mouse is crucial directly to the life cycle of B. burgdorferi. Man appears to be an accidental host for the tick. Clinical Lyme disease is known to occur in dogs, horses and cattle, although the importance of these species in transmission to man is unknown.

Clinical picture

Lyme disease generally occurs in stages, referred to as localised (stage 1), disseminated infection (stage 2) and late infection (stage 3).

Stage 1 is typified by a characteristic skin rash, erythema migrans (EM), sometimes referred to as erythema chronicum migrans. The rash begins as an erythematous macule or papule 4 - 20 days after a tick bite and gradually enlarges to form a large erythematous

annular lesion. In untreated patients, EM usually resolves within 3 - 4 weeks, but may recur. EM may be accompanied by headache, malaise, fever, chills, stiff neck, arthralgia, myalgia, conjunctivitis and elevated transaminases. Regional lymphadenopathy is sometimes seen.

Stage 2 is characterised by neurological disorders and cardiac abnormalities which present about a month after stage 1 (range 2 - 21 weeks) in 10 - 15% of patients. The most common symptom is aseptic meningitis, but other signs and symptoms include cranial nerve palsies, peripheral radiculoneuropathy, and atrioventricular block. Of the cranial nerves, the facial (VII) is the most commonly involved, with unilateral or bilateral facial palsies seen in up to 11% of patients. Less frequently, involvement of the oculomotor (III) or trochlear (IV) nerves is seen. The only facial nerves in which dysfunction has not been observed in Lyme disease are I and XI. Cardiac manifestations occur in about 8% of patients, and usually present within 21 weeks of the bite (mean 5 weeks). These include AV block, myopericarditis and left ventricular dysfunction. Cardiac signs and symptoms usually resolve within 6 weeks, although severe cases of AV block sometimes require temporary pacing.

Other systems which may be involved are the eyes, liver, respiratory, kidney and genitourinary systems. Constitutional symptoms may include severe malaise and fatigue.

Stage 3 of the disease presents months to years after Stage 1 in 50-60% of untreated patients. It is typified by intermittent episodes of oligoarthritis in the larger joints. After several years the arthritis becomes chronic in about 10% of patients, and permanent cartilage damage can occur in patients with long standing disease. Acrodermatitis chronica atrophicans and other neurological disorders may present in this stage, with persistence of some of the neurological signs and symptoms seen in stage 2.
There is some clinical variation reported in the pattern of Lyme disease symptoms between the US and Europe (3). EM is seen in 50-70% of US patients and in about 20% of European cases. In the US, arthritis is much more common in untreated patients than neurological signs, while in a German study 56% of patients had neurological signs and 13% arthritis - essentially the reverse of the US experience. Acrodermatitis chronica atrophicans is rare in the US, while about 10% of untreated European patients present with this symptom.

Laboratory diagnosis

Laboratory diagnosis in the US is carried out using an Indirect Fluorescent Antibody Test (IFAT) or Enzyme-linked Immunosorbent Assay (ELISA). Diagnosis may, however, be complicated by false positive reactions in the IFAT due to cross reactions with treponemes or leptospires (both are in the same phylum as borrelia), or with certain autoantibodies such as rheumatoid factor.

False negative results may be seen in patients early in the course of the disease, or in those seen later but in whom early antibiotic treatment has abrogated the humoral response. Interlaboratory variation in test results has also complicated diagnosis in the United States. In addition, symptoms sometimes persist in patients treated with antibiotics and who remain seronegative.

Treatment

Treatment of stage 1 of Lyme disease is generally oral tetracycline (or doxycycline) or an oral penicillin (eg. Penicillin V or amoxycillin). Erythromycin is used as a second line drug for those cases where a penicillin or tetracycline is contraindicated. Patients with stage 2 or 3 disease usually respond to parenteral beta-lactam antibiotics (eg. penicillin, ceftriaxone or cefotaxime.)

The Australian experience

A clinical syndrome with some of the features of Lyme disease was first described in Australia in 1982 (4). The patient was a 21-year-old labourer who was bitten on the leg by an unidentified 'insect'. The patient presented with EM which subsequently ran a relapsing course, with occasional secondary lesions developing on the face or shoulders. The patient also experienced recurrent oligoarthritis (left knee and left hip). Later, a marked behavioural change, headaches, memory loss, and urinary retention were observed. A diagnosis of mild, resolving meningoencephalitis was made. An ECG-documented supraventricular tachycardia, without AV block was also detected. Treatment was with IV penicillin and corticosteroids early in the course of the disease, neither of which appeared to have any effect on the course of the disease.

Details of 3 further cases of a Lyme disease-like syndrome in Australia were reported in 1986 (5,6). Only one patient recalled having been bitten by a tick. Symptoms in these patients included EM (3/3), lethargy (2/3), and arthralgia (1/3), fever (1/3) headache (2/3) and sore throat (1/3).

One patient was treated with IV penicillin, which resulted in resolution of the symptoms. A second was treated with oral tetracycline, and although the rash cleared, it recurred some weeks later. This was treated with oral penicillin, and the rash resolved completely. The third patient was treated with doxycycline, and the symptoms subsequently cleared.

An assessment of the epidemiology of Lyme disease in Australia is complicated by the fact that the aetiological agent of the clinical disease(s) in Australia has not yet been identified. Serological tests in use in this country are based on antigens from North American or European strains of *B. burgdorferi*, and their specificity for local strains of the causative organism are unknown. Furthermore, the vector(s) of the disease in Australia is unknown, although the common bush or scrub tick, *Ixodes holocyclus*

has been proposed as a candidate. This tick is found along the coastal plain from Queensland to eastern Victoria, and there is a single record from eastern Tasmania (see map 1). Although it is believed that the tick's preferred host is the bandicoot, it will feed on any warm blooded animal. At this stage, however, this species of tick can not be assumed to be involved in transmission of the disease in Australia.

Map 1. Locations (dots) of cases diagnosed as Lyme disease in NSW and the distribution (shaded area) of *Ixodes holocyclus* in eastern Australia. Map courtesy of Dr R Munro, Liverpool Hospital, NSW.

Laboratory (serological) tests for Lyme disease are considered difficult to interpret because of cross reactivity with other spirochaetes. An Indirect Fluorescent Antibody Test (IFAT) and an Enzyme-Linked Immunosorbent Assay (ELISA) have been developed by some Australian laboratories based on a North American strain of Borrelia burgdorferi. In addition, several commercial test kits are available, but because of questions about the aetiology of the Australian syndrome, their sensitivity and specificity are uncertain.

CDI has received results from three laboratories which have serological tests for Lyme disease. Westmead Hospital, Westmead, NSW has developed its own IFAT and ELISA. Testing of clinical material at that hospital was begun in March 1988. From then until April 1990 a total of 926 specimens from 809 patients was tested (see table 1). All specimens were tested by both IFAT and ELISA.

Table 1. Lyme disease serology March 1988 - March 1990
Westmead Hospital

TIME PERIOD	NO. SPECIMENS TESTED
3/88 to 12/88	351
Calendar 1989	413
1/90 to 3/90	162
Total	926

On the basis of serological criteria determined by the laboratory, 19 (2.1%) were considered doubtful positives, 27 (2.1%) were possible positives and 28 (3.0%) were probable positives.

In addition to testing clinical specimens, this laboratory has, since 1988, tested samples of blood obtained from blood banks. Half of these came from the Sydney area, and the other half from the south coast of NSW.
A low level of seropositivity was found, with no significant difference in prevalence between the Sydney and south coast specimens.

Serological testing for Lyme disease commenced in Queensland in 1987 with a culture of Borrelia burgdorferi obtained from the USA. This culture has been repeatedly subcultured and is used as the source of antigen for the Indirect Fluorescent Antibody (IFAT) test performed in the State Health Department's Serology section.
Results obtained to June 1989 are summarised in Table 2 over

Table 2. Lyme disease serology 1986 - June 1989 at the State Health Laboratories, Brisbane

TIME PERIOD	NO. TESTED	% POSITIVE*	NO. POSITIVE
1986/87	126	22.0%	28+
1987/88	641	20.0%	128+
1988/89	480	6.3%	30
Total	1247		

* ie. IFAT titre of 1:64 or greater
\+ Approximate numbers

Eight of the patients diagnosed in 1987/88 also had IgM - specific antibody. Five of these were from north Queensland and two from interstate. Information provided for these patients showed that two had contact with ticks, three had a history of arthritis and one had a history of dementia.

Of the 30 positive specimens tested in 1988/89, four also had IgM-specific antibodies present, without cross-reactivity to other spirochetal tests performed in the State Health Laboratory. Three of these persons were from North Queensland and the fourth patient was a child from the Gold Coast for whom no clinical information was received. Two of the North Queensland patients had a history including fever and arthritis and the third presented with a history of depression.

The State Health Laboratory in WA has an ELISA test for Lyme disease. Thus far, positive serology has been returned on three patients. One was a woman who had almost certainly acquired the infection in Europe. The second patient was a 2.5 year old girl. Serum has been sent to the Centres for Disease Control, Atlanta, for confirmation but no result had been returned at the time of writing (June 1990). The third patient was an adult male about whom there are no other details.

There would appear to be no serological data to June 1990 indicating that Lyme disease is present in Victoria, South Australia, Tasmania, the Northern Territory or the Australian Capital Territory. However, enquiries made in those States and Territories were not exhaustive, and it is possible that some cases have been diagnosed.
Recently a case of Lyme disease was diagnosed by a laboratory in the UK in a patient who had recently visited Papua New Guinea. The patient was a 10 year old male (7). No other details are available, but it should be noted that Lyme disease is known to occur in the UK.

SUMMARY AND CDI EDITORIAL COMMENT

A clinical syndrome resembling the Lyme disease or Lyme borreliosis of North America and Europe has been described in Australia. Most cases have been reported from the coastal regions of New South Wales and Queensland, although two locally acquired cases have been reported from Western Australia, and possibly one from Papua New Guinea.

Neither the aetiological agent(s) nor its vector(s) has been described in Australia, although serological testing of patients has indicated that the aetiological agent may be related to, if not identical with, Borrelia burgdorferi. The common bush or scrub tick, Ixodes holocyclus has been postulated as a possible vector, based on ecological data; it would, however, be inappropriate with current knowledge to draw firm conclusions as to its role, if any, in transmission of this disease.

Relatively few laboratories in Australia routinely test for Lyme disease. This, together with uncertainty about the aetiology of the disease in this country means that knowledge of its epidemiology is limited at best. CDI would welcome reports from medical practitioners and laboratories on patients presenting with a clinical syndrome resembling Lyme disease or who return positive serology. Data such as clinical presentation, age/sex of patient, history of tick or other 'insect' bite, time and place of acquisition of the infection, type of test performed, titre, and whether a commercial or in-house test would provide valuable epidemiological information. Alternatively, blood (10 mL, clotted tube) could be sent for serology to :-

Mr D Dickeson
Bacteriology Unit
Department of Infectious Diseases and Microbiology
Westmead Hospital
Westmead NSW 2145

A research program is also being undertaken at the same hospital in an attempt to isolate the aetiological agent and identify the vector(s). Medical practitioners in New South Wales, Victoria, South Australia, Western Australia, Tasmania and the Australian Capital Territory who remove ticks or other 'insects' from patients in whom they suspect Lyme disease are encouraged to forward the specimen to :-

Dr R C Russell
Head, Medical Entomology Unit
Department of Infectious Diseases and Microbiology
Westmead Hospital
WESTMEAD NSW 2145

Tick specimens collected from patients in Queensland and the Northern Territory should be forwarded to :-

Mr R Silcock
Serology Section
State Health Laboratories
63 George Street
BRISBANE QLD 4000

The specimen should be sent, if possible in the live state, in a screw-capped glass or plastic vial containing some loose, dry packing material such as facial tissue, with one drop of water added. Ticks can survive for many months without nutrients at room temperature. The water is added to increase humidity in the tube and assists in maintaining viability of the specimen. It is important that only one drop is added. Excess water will encourage fungal growth which will destroy the insect.

Acknowledgements

CDI wishes to thank the following people for the provision of data for this article:-

Dr R Munro, Liverpool Hospital, Liverpool NSW
Dr R Russell, Westmead Hospital, Westmead NSW
Mr D Dickeson, Westmead Hospital, Westmead NSW
Mr R Silcock, State Health Laboratories, Brisbane Qld
Dr J Gill, Health Department of Western Australia, Perth WA

References

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4. Stewart A, Glass J, Patel A et al. Lyme arthritis in the Hunter Valley. Med J Aust. 1982; 1: 139

5. McCrossin I. Lyme disease on the NSW south coast. Med J Aust. 1986; 144 724-5

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7. Communicable Disease Report UK 1990 90/19 P5.

MENINGOCOCCAL MENINGITIS IN THE NORTHERN TERRITORY

(Contributed by the Northern Territory Department of Health and Community Services, Nganampa Health Council and Central Australian Aboriginal Congress, Alice Springs).

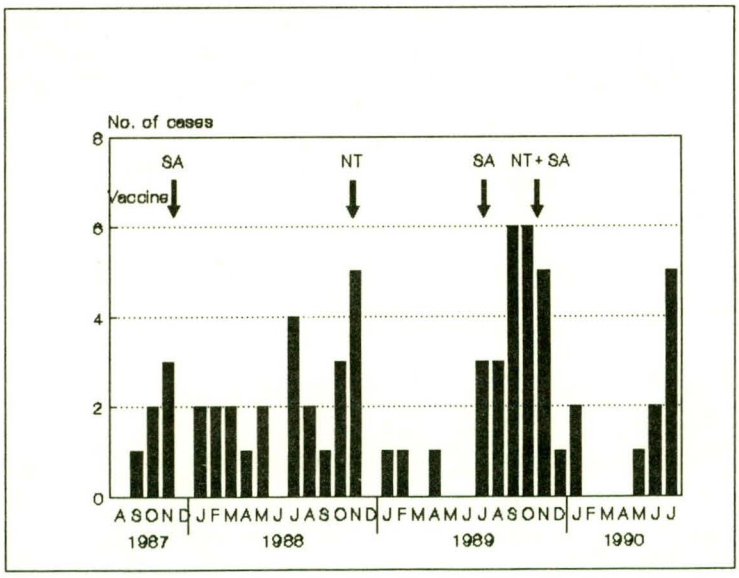
Introduction

The initial outbreak of 15 cases of meningococcal meningitis in 1987/88 in central Australia (CA) was described in CDI 88/16. Cases are still being seen in this region in 1990. Chemoprophylaxis to close contacts of index cases and a vaccination program of children between 1 and 15 years of age have been effective in preventing a larger outbreak of the disease. There have been more than the usual number of sporadic cases in the top end of the Northern Territory (NT).

The outbreak and vaccination programme 1987-90

One to three cases of meningococcal disease have been reported annually from CA since the outbreak of meningitis between 1971 and 1973. The earliest cases in the current outbreak were seen at the end of 1987 among Aboriginal communities bordering on the NT, i.e. in Western Australia (not included in this paper) and in the Nganampa Health Areas of South Australia (north-western SA). The 1987 vaccination programme for the 628 children between 3 and 15 years of age in the Nganampa areas was described in CDI 88/16. Thereafter, cases occurred among Aboriginal people in the southern region of the NT, peaking in October/November 1988 (Figure 1).

Figure 1. Incidence of Meningitis

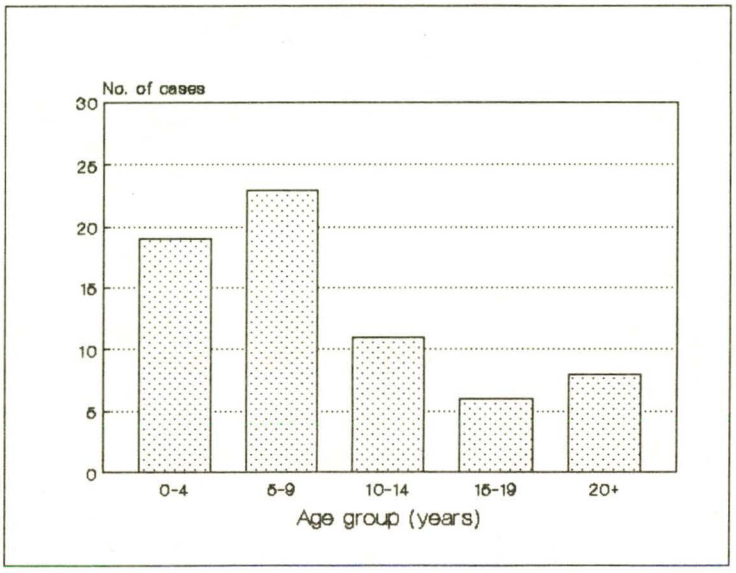


Vaccination with the bivalent "Mencevax AC" (SmithKline Biologicals) was offered to about 4 300 Aboriginal children between 2 and 15 years of age in the southern region of the NT.

When a second wave of cases were seen in the Nganampa areas in mid 1989, catch-up vaccination was offered to children over 2 years of age not previously vaccinated. Later that year, cases occurred in children under the age of 2 years in both regions, and therefore the age of catch-up vaccination of Aboriginal children in both regions was lowered to 1 year. After a 3 month lull, more cases were seen between May and July 1990.

There were a total of 69 cases with meningococcal meningitis since October 1987. Only two cases were Europeans, (not detailed further in this paper - both were adults with serogroup C infections), while the remaining 67 were in Aboriginal people. The age distribution of the cases is shown in Figure 2.

Figure 2. Age distribution of meningococcal meningitis cases

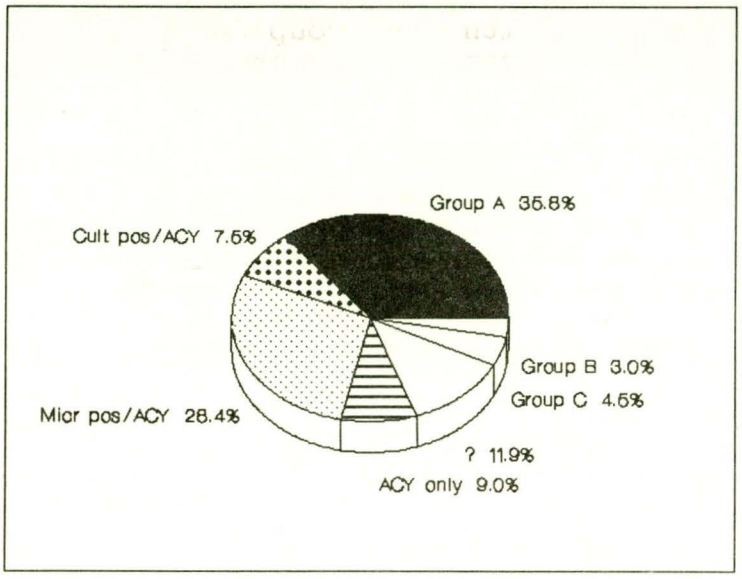


There were 3 deaths, all in 1988: a child died while being treated for pneumonia in a rural community, an adult was found dead at home after a one day illness, and an adult with chronic renal and cardiac disease died in hospital. None had the clinical features of meningococcal septicemia.

Meningococcal serogroups

Serogrouping was performed at either the Microbiology laboratory of the Adelaide Childrens' Hospital or the Microbiological Diagnostic Unit at Melbourne University. The results of serogrouping the culture positive cases, and of the microscopy and latex (A, C, Y, W135) tests in the culture negative cases are shown in Figure 3. All the culture negative cases received parenteral antibiotics before admission to hospital. Of the organisms grown on culture, 22 were serogroup A; three were serogroup C and two serogroup B.

Figure 3. Meningococcal serogroups (67 cases)



Vaccine efficacy

The calculation of vaccine efficacy is complicated by several factors:

1. vaccination having occurred on four separate occasions between 1987 and 1989 (Figure 2),
2. uncertainty on the exact number of the denominator population eligible for vaccination and on the vaccination status of the 4 children who developed meningitis, and
3. an inability to identify the specific organism and/or serogrouping in 9 of the 18 cases who had been vaccinated previously.

Based on the best estimate of the denominator population, and assuming the least favourable outcome of vaccination, i.e. that all 18 cases had serogroup A meningococcal meningitis and had been vaccinated previously; the vaccine efficacy is 92.7% (95% confidence intervals 82.7% - 96.9%). This estimate does not allow for differences in age-specific efficacy rates (efficacy is lower in children < 4 years of age) and for the varying intervals between time of vaccination and occurrence of disease (efficacy wanes progressively with time, and quicker in children < 4 years of age). The overall efficacy is higher than that reported from larger studies in the 'meningitis belt' of Africa.

Chemoprophylaxis

Rifampicin was routinely offered to close contacts, and also to the index cases before discharge from hospital. There were 3 secondary cases among known contacts of an index case; two children had not received Rifampicin, and the third case developed in an adult contact 3 weeks after she had taken the two-day course of Rifampicin.

Meningococcal disease in the Top End

Five (5) cases have been seen in the Katherine Region since 1987. In October 1989 a 6 year old Aboriginal child died with the clinical features of septicemia (serogroup C). Two Aboriginal children were diagnosed with serogroup C meningitis in March 1990. Two European children had meningitis; one died in hospital in April 1990, and the second case occurred in May 1990. The CSF gram stain and culture results were both negative, but the latex (A, C, Y, W135) result was positive in both cases.

There were 3 European children with meningococcal meningitis (serogroup B, C and WB5) and one with meningcoccal septicemia (serogroup C) in Darwin between June 1989 and January 1990; further cases have not been seen since then.

Conclusion

The control measures have contained, but not arrested the serogroup A outbreak which began at the end of 1987 in central Australia. A high level of clinical protection has been provided by the vaccine. In the Top End of the Northern Territory, a higher incidence than usual was observed of sporadic serogroup C disease since October 1989.

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Similarly, readers sited relatively close together (eg same department, branch, etc) and thus capable of locally distributing copies by hand, are asked to give consideration to having their total requests bulk mailed.

Please direct notifications to:

CDI mailing list
Communicable Diseases Section
Commonwealth Department of
Community Services and Health
GPO Box 9848
CANBERRA ACT 2601

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

PERIOD 16/8/90 TO 29/8/90

1. CODE 018 - MICROBIOL DIAG UNIT, UNI MELB (VIC)
2. CODE 019 - FAIRFIELD HOSP (VIC)
3. CODE 065 - STATE HEALTH LAB (WA)
4. CODE 066 - PRINCESS MARGARET HOSP (WA)
5. CODE 110 - INST OF MED & VET SCIENCE (SA)
6. CODE 111 - ROYAL CHILDRENS HOSP (VIC)
7. CODE 112 - INST CLINICAL PATH & MED RES (NSW)
8. CODE 113 - PRINCE HENRY/PRINCE OF WALES HOSP (NSW)
9. CODE 114 - ROYAL ALEXAND RA CHILDRENS HOSP (NSW)
10. CODE 115 - STATE HEALTH LAB (QLD)
11. CODE 116 - WODEN VALLEY HOSP (ACT)

		019	065	066	110	111	112	113	114	115	116	TOTAL
0100	ADENOVIRUS NOT TYPED	0	0	2	4	0	9	4	0	8	0	27
0101	ADENOVIRUS TYPE 1	1	0	0	1	0	0	0	0	0	0	2
0102	ADENOVIRUS TYPE 2	3	0	0	0	0	1	1	0	0	0	5
0103	ADENOVIRUS TYPE 3	1	0	0	2	0	1	0	1	0	0	5
0104	ADENOVIRUS TYPE 4	2	0	0	0	0	0	0	0	0	0	2
0105	ADENOVIRUS TYPE 5	0	0	0	0	0	1	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	0	0	0	0	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	2	0	0	0	0	2
0128	ADENOVIRUS TYPE 28	0	0	0	1	0	0	0	0	0	0	1
0137	ADENOVIRUS TYPE 37	0	0	0	2	0	0	0	0	0	0	2
0142	ADENOVIRUS TYPE 42	1	0	0	0	0	0	0	0	0	0	1
0145	ADENOVIRUS TYPE 45	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	0	0	0	3	0	3	0	0	0	7
0201	INFLUENZA A VIRUS	2	0	8	0	0	1	0	0	0	1	12
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	0	0	0	0	0	0	3	0	3
0203	INFLUENZA B VIRUS	1	0	0	0	0	1	0	0	0	1	3
0301	PARAINFLUENZA VIRUS TYPE 1	2	0	0	4	2	2	0	0	0	0	10
0302	PARAINFLUENZA VIRUS TYPE 2	1	0	0	0	1	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	1	0	0	0	1	4	1	0	0	0	7
0400	RESPIRATORY SYNCYTIAL VIRUS (R	57	2	61	54	57	9	4	6	23	5	278
0500	RHINOVIRUS (ALL TYPES)	10	0	0	0	7	2	1	0	0	0	20
0600	MYCOPLASMA PNEUMONIAE	2	0	0	3	1	3	0	0	0	1	10
0700	ORNITHOSIS-PSITTACOSIS	1	1	0	0	0	0	0	0	0	0	2
0809	COXSACKIEVIRUS A9	0	0	0	0	0	2	0	0	0	0	2
0816	COXSACKIEVIRUS A16	2	0	0	0	0	0	0	0	0	0	2
0903	COXSACKIEVIRUS B3	0	0	0	1	0	1	0	0	0	0	2
1000	ECHOVIRUS NOT TYPED	0	0	0	1	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	0	0	0	0	1	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	0	0	2	0	1	0	0	3
1009	ECHOVIRUS TYPE 9	0	0	0	0	0	1	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	0	0	0	1	0	0	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	0	0	0	0	1	0	1	0	0	2
1018	ECHOVIRUS TYPE 18	1	0	0	0	0	0	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	0	0	0	0	0	0	1	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	4	0	0	0	4
1101	POLIOVIRUS TYPE 1	2	0	0	0	0	0	0	1	0	0	3
1102	POLIOVIRUS TYPE 2	0	0	0	1	0	0	0	1	0	0	2
1103	POLIOVIRUS TYPE 3	0	0	0	0	0	2	0	1	0	0	3
1200	MUMPS VIRUS	1	0	0	0	0	2	0	0	0	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	0	0	2	0	1	0	0	0	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	0	3	0	0	33	0	0	0	9	46
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	5	7	0	16	2	0	3	2	0	0	35
1303	VARICELLA-ZOSTER VIRUS	6	3	0	0	0	2	0	0	1	0	12
1306	HERPES SIMPLEX TYPE 1	46	30	0	6	2	3	0	1	20	0	108
1307	HERPES SIMPLEX TYPE 2	48	58	0	10	0	11	0	0	21	0	148
1366	HERPES VIRUS TYPE 6	0	0	0	0	0	0	0	1	0	0	1
1399	HERPES VIRUS TYPING PENDING	0	6	0	0	7	0	0	0	1	0	14
1401	COXIELLA BURNETII	0	1	0	0	0	1	0	0	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	3	0	1	0	0	6	0	5	0	15
1521	MEASLES VIRUS	6	1	0	0	1	1	0	0	0	2	11
1522	RUBELLA VIRUS	3	0	0	0	0	0	0	0	0	0	3
1532	HEPATITIS B ANTIGEN	12	24	0	8	2	44	1	1	38	0	130
1535	HEPATITIS A ANTIBODY	3	7	0	3	0	0	0	0	0	0	13
1536	HEPATITIS C VIRUS	0	6	0	0	0	1	0	1	0	1	9
1541	CHLAMYDIA A - C. TRACHOMATIS	0	60	0	42	0	22	2	0	0	3	129
1556	CMV - CYTOMEGALOVIRUS	43	3	6	2	7	4	2	2	10	0	79
1563	CORONAVIRUS	0	0	0	0	0	2	0	0	0	0	2
1564	ROTAVIRUS	8	0	18	16	0	15	13	3	0	0	73
1566	NORWALK AGENT	0	0	0	0	0	1	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	2	0	0	0	0	4	0	0	0	0	6
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	2	0	2	2	0	0	6
9992	ROSS RIVER VIRUS	2	0	0	0	0	0	0	0	0	0	2
9993	ASTROVIRUS	0	0	0	0	0	3	0	0	0	0	3
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	2	0	1	0	0	3
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	1	0	0	0	0	0	0	1
TOTAL		279	212	98	180	97	198	48	27	130	23	1292

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 16/8/90 TO 29/8/90

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	13	0	8	2	4	0	27
0101	ADENOVIRUS TYPE 1	0	1	0	0	1	0	2
0102	ADENOVIRUS TYPE 2	2	3	0	0	0	0	5
0103	ADENOVIRUS TYPE 3	2	1	0	0	2	0	5
0104	ADENOVIRUS TYPE 4	0	2	0	0	0	0	2
0105	ADENOVIRUS TYPE 5	1	0	0	0	0	0	1
0110	ADENOVIRUS TYPE 10	1	0	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	2	0	0	0	0	0	2
0128	ADENOVIRUS TYPE 28	0	0	0	0	1	0	1
0137	ADENOVIRUS TYPE 37	0	0	0	0	2	0	2
0142	ADENOVIRUS TYPE 42	0	1	0	0	0	0	1
0145	ADENOVIRUS TYPE 45	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	3	4	0	0	0	0	7
0201	INFLUENZA A VIRUS	1	2	0	8	0	1	12
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	0	3	0	0	0	3
0203	INFLUENZA B VIRUS	1	1	0	0	0	1	3
0301	PARAINFLUENZA VIRUS TYPE 1	2	4	0	0	4	0	10
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	5	2	0	0	0	0	7
0400	RESPIRATORY SYNCYTIAL VIRUS (R	19	114	23	63	54	5	278
0500	RHINOVIRUS (ALL TYPES)	3	17	0	0	0	0	20
0600	MYCOPLASMA PNEUMONIAE	3	3	0	0	3	1	10
0700	ORNITHOSIS-PSITTACOSIS	0	1	0	1	0	0	2
0809	COXSACKIEVIRUS A9	2	0	0	0	0	0	2
0816	COXSACKIEVIRUS A16	0	2	0	0	0	0	2
0903	COXSACKIEVIRUS B3	1	0	0	0	1	0	2
1000	ECHOVIRUS NOT TYPED	0	0	0	0	1	0	1
1004	ECHOVIRUS TYPE 4	1	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	3	0	0	0	0	0	3
1009	ECHOVIRUS TYPE 9	1	0	0	0	0	0	1
1011	ECHOVIRUS TYPE 11	0	0	0	0	1	0	1
1014	ECHOVIRUS TYPE 14	2	0	0	0	0	0	2
1018	ECHOVIRUS TYPE 18	0	1	0	0	0	0	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	1	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	4	0	0	0	0	0	4
1101	POLIOVIRUS TYPE 1	1	2	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	1	0	0	0	1	0	2
1103	POLIOVIRUS TYPE 3	3	0	0	0	0	0	3
1200	MUMPS VIRUS	2	1	0	0	0	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	1	3	0	0	0	0	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	33	1	0	3	0	9	46
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	5	7	0	7	16	0	35
1303	VARICELLA-ZOSTER VIRUS	2	6	1	3	0	0	12
1306	HERPES SIMPLEX TYPE 1	4	48	20	30	6	0	108
1307	HERPES SIMPLEX TYPE 2	11	48	21	58	10	0	148
1366	HERPES VIRUS TYPE 6	1	0	0	0	0	0	1
1399	HERPES VIRUS TYPING PENDING	0	7	1	6	0	0	14
1401	COXIELLA BURNETII	1	0	0	1	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	6	0	5	3	1	0	15
1521	MEASLES VIRUS	1	7	0	1	0	2	11
1522	RUBELLA VIRUS	0	3	0	0	0	0	3
1532	HEPATITIS B ANTIGEN	46	14	38	24	8	0	130
1535	HEPATITIS A ANTIBODY	0	3	0	7	3	0	13
1536	HEPATITIS C VIRUS	2	0	0	6	0	1	9
1541	CHLAMYDIA A - C. TRACHOMATIS	24	0	0	60	42	3	129
1556	CMV - CYTOMEGALOVIRUS	8	50	10	9	2	0	79
1563	CORONAVIRUS	2	0	0	0	0	0	2
1564	ROTAVIRUS	31	8	0	18	16	0	73
1566	NORWALK AGENT	1	0	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	4	2	0	0	0	0	6
1599	ENTEROVIRUS TYPING PENDING	4	2	0	0	0	0	6
9992	ROSS RIVER VIRUS	0	2	0	0	0	0	2
9993	ASTROVIRUS	3	0	0	0	0	0	3
9994	SMALL VIRUS (LIKE) PARTICLE	3	0	0	0	0	0	3
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	0	0	0	0	1	0	1
TOTAL		273	376	130	310	180	23	1292

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

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 16/8/90 TO 29/8/90

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	14	0	0	0	1	26
0101	ADENOVIRUS TYPE 1	0	1	0	0	0	0	0	0	0	0	0	1
0102	ADENOVIRUS TYPE 2	1	2	0	0	0	0	0	0	0	0	1	4
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	2	0	0	0	0	3
0105	ADENOVIRUS TYPE 5	0	0	0	0	0	0	1	0	0	0	0	1
0111	ADENOVIRUS TYPE 11	0	0	0	0	0	0	2	0	0	0	0	2
0128	ADENOVIRUS TYPE 28	0	0	0	0	0	0	1	0	0	0	0	1
0137	ADENOVIRUS TYPE 37	0	1	0	0	0	0	0	0	0	0	0	1
0145	ADENOVIRUS TYPE 45	0	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	4	0	0	0	0	0	0	0	0	0	4
0201	INFLUENZA A VIRUS	0	8	0	0	0	2	0	0	0	0	0	10
0202	INFLUENZA A VIRUS SUBTYPE H3N2	0	2	0	0	1	0	0	0	0	0	0	3
0203	INFLUENZA B VIRUS	1	1	0	0	0	0	0	0	0	0	0	2
0301	PARAINFLUENZA VIRUS TYPE 1	1	9	0	0	0	0	0	0	0	0	0	10
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	1	6	0	0	0	0	0	0	0	0	0	7
0400	RESPIRATORY SYNCYTIAL VIRUS (R	6	260	0	0	1	0	1	0	0	0	0	268
0500	RHINOVIRUS (ALL TYPES)	1	18	0	0	0	0	0	0	0	0	0	19
0600	MYCOPLASMA PNEUMONIAE	1	9	0	0	0	0	0	0	0	0	0	10
0700	ORNITHOSIS-PSITTACOSIS	0	2	0	0	0	0	0	0	0	0	0	2
0809	COXSACKIEVIRUS A9	1	0	0	0	0	0	1	0	0	0	0	2
0816	COXSACKIEVIRUS A16	0	0	0	0	0	0	1	0	0	0	1	2
0903	COXSACKIEVIRUS B3	0	1	0	0	0	0	0	0	0	0	0	1
1004	ECHOVIRUS TYPE 4	0	0	0	0	0	0	1	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	1	0	0	1	0	0	1	0	0	0	0	3
1009	ECHOVIRUS TYPE 9	0	0	0	0	0	0	0	0	0	0	1	1
1011	ECHOVIRUS TYPE 11	0	1	0	0	0	0	0	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	1	0	0	1	0	0	0	0	0	0	0	2
1018	ECHOVIRUS TYPE 18	0	0	0	0	0	0	0	0	0	0	1	1
1028	ECHOVIRUS TYPE 28 = RHINO VIRU	0	1	0	0	0	0	0	0	0	0	0	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	0	3	0	0	0	0	3
1101	POLIOVIRUS TYPE 1	0	2	0	0	0	0	0	0	0	0	0	2
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	1	0	0	0	0	1
1103	POLIOVIRUS TYPE 3	1	1	0	0	0	0	0	0	0	0	0	2
1200	MUMPS VIRUS	3	0	0	0	0	0	0	0	0	0	0	3
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	1	0	0	1	0	0	0	0	1	4
1301	HERPES SIMPLEX VIRUS - NOT TYP	8	1	1	0	0	0	0	0	0	0	18	28
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	3	0	1	0	0	0	0	2	0	0	0	6
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	0	0	0	11	11
1306	HERPES SIMPLEX TYPE 1	0	6	0	0	0	0	0	0	1	0	68	75
1307	HERPES SIMPLEX TYPE 2	2	0	0	0	0	0	0	0	0	0	76	78
1366	HERPES VIRUS TYPE 6	0	0	0	0	0	0	0	0	0	0	1	1
1399	HERPES VIRUS TYPING PENDING	0	2	0	0	0	0	0	0	0	0	12	14
1401	COXIELLA BURNETII	1	0	0	0	0	0	0	1	0	0	0	2
1502	PICORNIA VIRUS - NOT TYPED = E	0	6	0	0	0	0	8	0	0	0	0	14
1521	MEASLES VIRUS	0	0	0	0	0	0	0	0	0	0	8	8
1532	HEPATITIS B ANTIGEN	51	0	0	0	0	0	1	72	0	0	0	124
1535	HEPATITIS A ANTIBODY	3	0	0	0	0	0	0	7	0	0	0	10
1536	HEPATITIS C VIRUS	2	0	0	0	0	0	0	7	0	0	0	9
1541	CHLAMYDIA A - C. TRACHOMATIS	14	1	0	0	0	0	0	0	0	3	1	19
1556	CMV - CYTOMEGALOVIRUS	4	22	0	0	0	1	1	5	0	4	1	38
1563	CORONAVIRUS	0	0	0	0	0	0	2	0	0	0	0	2
1564	ROTAVIRUS	2	0	0	0	0	0	71	0	0	0	0	73
1566	NORWALK AGENT	0	0	0	0	0	0	1	0	0	0	0	1
1571	ENTEROVIRUS TYPE 71 (BCR)	2	2	0	0	0	0	0	0	0	0	2	6
1599	ENTEROVIRUS TYPING PENDING	0	2	0	0	0	0	2	0	0	0	0	4
9992	ROSS RIVER VIRUS	0	0	0	0	0	0	0	0	0	0	1	1
9993	ASTROVIRUS	0	0	0	0	0	0	3	0	0	0	0	3
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	0	3	0	0	0	0	3
9998	ARBOVIRUS GROUP B.(UNSPECIFIED	1	0	0	0	0	0	0	0	0	0	0	1
TOTAL		114	384	3	2	2	4	122	94	1	7	205	938

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 16/8/90 TO 29/8/90

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	0	1	0	0	0	0	0	0	0	0	1
0101	ADENOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	0	1
0102	ADENOVIRUS TYPE 2	0	0	0	0	0	0	0	0	1	0	1
0103	ADENOVIRUS TYPE 3	2	0	0	0	0	0	0	0	0	0	2
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	1	0	0	2
0110	ADENOVIRUS TYPE 10	0	0	0	0	0	0	0	0	1	0	1
0137	ADENOVIRUS TYPE 37	0	1	0	0	0	0	0	0	0	0	1
0142	ADENOVIRUS TYPE 42	1	0	0	0	0	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	1	0	0	0	0	0	1	1	0	3
0201	INFLUENZA A VIRUS	0	0	0	0	0	0	1	1	0	0	2
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	1	0	0	0	1
0400	RESPIRATORY SYNCYTIAL VIRUS (R	0	0	0	0	0	0	0	2	8	0	10
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	0	0	0	1	1
0903	COXSACKIEVIRUS B3	0	0	0	0	0	0	0	0	1	0	1
1000	ECHOVIRUS NOT TYPED	0	0	0	0	0	0	0	0	0	1	1
1100	POLIOVIRUS NOT TYPED	0	0	0	0	0	1	0	0	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	0	0	1	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	1	0	0	0	0	1
1103	POLIOVIRUS TYPE 3	0	0	0	0	0	1	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	16	0	0	0	0	0	0	2	0	18
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	1	20	2	0	0	1	1	4	0	29
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	0	1	0	1
1306	HERPES SIMPLEX TYPE 1	8	23	0	0	0	0	0	0	2	0	33
1307	HERPES SIMPLEX TYPE 2	1	69	0	0	0	0	0	0	0	0	70
1502	PICORNIA VIRUS - NOT TYPED = E	1	0	0	0	0	0	0	0	0	0	1
1521	MEASLES VIRUS	0	0	0	0	0	0	0	1	2	0	3
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	0	3	0	3
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	0	6	0	6
1535	HEPATITIS A ANTIBODY	0	0	0	0	0	0	0	1	2	0	3
1541	CHLAMYDIA A - C. TRACHOMATIS	1	109	0	0	0	0	0	0	0	0	110
1556	CMV - CYTOMEGALOVIRUS	3	1	0	1	0	4	1	2	29	0	41
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	0	0	2	0	2
9992	ROSS RIVER VIRUS	0	0	0	0	1	0	0	0	0	0	1
TOTAL		18	222	20	3	1	7	4	10	66	3	354