Arboviruses in the Australian region, 1990 to 1998

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Abstract

Arboviruses continue to be major human pathogens in the Australian region. This report provides a summary of the activities of these viruses over the past eight years, and comments on new findings relevant to their respective ecologies. Of particular interest and concern is the propensity of these viruses to spread. The examples discussed include the initiation of dengue epidemics in north Queensland by virus imported in viraemic travellers; the spread of Japanese encephalitis virus to the Australasian region and its probable enzootic establishment in the south-west of Papua New Guinea; the potential spread of Ross River virus to other countries, as demonstrated by the 1979-80 outbreak in the South Pacific, and the recent occurrence in military personnel from the United States of America after an exercise; and the recent spread of Barmah Forest virus into Western Australia, *Comm Dis Intell* 1998;22:93-100

Introduction

Although more than 70 arboviruses have been reported from Australia, relatively few are human pathogens, and even fewer are of major concern. Those viruses which cause, or have caused, significant human disease in Australia are the flaviviruses Murray Valley encephalitis, Kunjin, Japanese encephalitis, and dengue virus types 1, 2 and 3; and the alphaviruses Ross River and Barmah Forest. All of these viruses have been responsible for human infections over the past decade. Each has its own specific ecological and epidemiological characteristics, and each has demonstrated a propensity to spread and become established in new areas, which is of growing concern. The purpose of this review is to describe the activities of these viruses and to comment on new data concerning their ecology and spread. Several recent reviews give more detailed information on the epidemiology of these viruses, including vector species, vertebrate host reservoirs, and geographic distribution.[1-4]

Dengue viruses

Dengue is not endemic in Australia. Although epidemics of dengue have been reported several

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times since 1990, these have been the result of importations by a viraemic tourist or returning resident.[1,5] The potential for local transmission of dengue is confined to an area in Queensland corresponding to the geographic range of its vector, *Aedes aegypti (Ae. aegypti)*. This extends from the islands in the Torres Strait in the north, to Mount Isa and Boulia in the west, to Roma in the south, and to Gladstone on the east coast.[6] Despite this broad geographic range, epidemic activity in the past two decades has been restricted to north Queensland from the Torres Strait south to Cairns, Townsville and Charters Towers. Outbreaks since 1990 have included:

- 1990-91, a few cases of dengue type 1 in Cairns and the Torres Strait;[1,5]
- 1992-93, a large outbreak of dengue type 2 principally in Townsville and Charters Towers with over 900 serologically confirmed cases (including the first reported case of dengue haemorrhagic fever this century) and a further 950 cases inferred on clinical grounds;[7,8]
- 1996-97, about 210 laboratory confirmed cases of dengue type 2, most of which were from the Torres Strait;[9] and
- 1997-98, approximately 165 laboratory confirmed cases of dengue type 3 and 12 of dengue type 2 from Cairns. The outbreak of dengue type 3 virus also resulted in a case of dengue haemorrhagic fever and the first case of dengue encephalopathy in Australia (J. Hanna and S. Ritchie, Queensland Tropical Public Health Unit, unpublished observiations).

Source of virus

Molecular epidemiological results have shown that the origin of the dengue 2 strain causing the 1992-93 outbreak was quite different to the origin of the 1996-97 outbreak dengue 2 strain. Nucleotide sequence data demonstrated that the 1992-93 isolates were most closely related to an isolate from Indonesia, whereas the 1996-97 isolates were most closely related to viruses which had been isolated originally from Burkino Faso. This latter finding is of interest because a large outbreak of dengue virus type 2 occurred on a number of the Pacific Islands before, during and after the 1996-97 Australian outbreak, but virus isolates obtained from the Pacific Islands were quite distinct from the Australian isolates.[9] Thus the Australian outbreak did not originate from a traveller from the Pacific.

Sequencing has shown that the dengue type 3 strain in Cairns in 1997-98 is most similar to isolates from Thailand (D. Phillips, unpublished data).

Establishment

With the increasing frequency of dengue virus introductions over the past decade, there has been some concern expressed that dengue might become endemic in the north-east of Australia. However, this is unlikely as humans are the only vertebrate hosts of the viruses and the population outside the urban areas of Cairns, Townsville and Charters Towers is sparse. Even when the vector, *Ae. aegypti*, was widespread earlier this century, there was no evidence to suggest that dengue was endemic, but rather epidemics probably arose from re-introductions via ships. Nevertheless, if vector numbers are high and re-introductions become more frequent, it might be difficult to distinguish endemicity from frequent re-introductions on epidemiological grounds without recourse to molecular techniques.

Management

Following the 1992-93 outbreak, a Dengue Fever Management Plan for north Queensland was developed by Queensland Health's Tropical Public Health Unit. The plan aims to lower the incidence of dengue in north Queensland. This is achieved by reducing vector breeding through education programs, encouraging greater awareness of the disease among general practitioners, and improved surveillance including the use of serological testing. The success of the plan has been evident in the rapid control of epidemic activity in 1996-97 and 1997-98, and by the recognition of other imported cases which were contained before they could give rise to further transmission.

Distribution risk

Early this century, *Ae. aegypti* mosquitoes were widespread in Australia, extending as far south as the Victorian border in eastern Australia and south of Perth in Western Australia,[5] but by the 1970s the distribution of *Ae. aegypti* had retreated to a small area in north Queensland. However, the pattern of epidemic dengue in north Queensland is not dissimilar to the global experience in tropical regions; that is, epidemic dengue is increasing in incidence and geographic distribution. After 25 years, epidemic dengue returned to north Queensland in 1981-82 and has increased in frequency since 1990[9]. This is associated with the increase in global travel, which has been shown to be a major factor in the emergence of infectious diseases. In parallel, *Ae. aegypti* has been spreading out from north Queensland reaching the

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Northern Territory border in the west and towards the New South Wales border in the south.

Future outlook

Continued vigilance is important to monitor both vector spread and disease importation. The spread or introduction of *Ae. aegypti* to other areas of Queensland, Northern Territory, northern New South Wales, or northern Western Australia, and importation of *Ae. albopictus*, another vector of dengue which is common in south-east Asia and Papua New Guinea, are on-going threats to public health. *Ae. aegypti* and *Ae. albopictus* have frequently been imported into Darwin in water containers on ships and in machinery,[4] and *Ae. albopictus* was imported into Perth in an international aircraft in 1975,[10] into Townsville in a cement truck from Papua New Guinea in 1997,[11] and most recently was trapped on a wharf in Cairns in 1998 (S. Ritchie, unpublished data). Quick action however, has prevented these importations from becoming established. Similarly, the importance of rapidly diagnosing imported cases in tourists or returning residents, and preventing onward transmission cannot be over emphasised.

Japanese encephalitis virus

The first reported outbreak of Japanese encephalitis (JE) in the Australasian region occurred in the Torres Strait in 1995.[12] Three cases were reported from Badu Island in the central Torres Strait, two of which were fatal. Seroepidemiological studies carried out at the time showed that the virus was relatively widespread in the central and northern islands of the Torres Strait with subclinical human cases observed on four islands, and seropositive pigs found on nine islands. It is generally believed that 1 in 30 to 1 in 300 infections with JE virus lead to clinical disease, approximately 25% of clinical cases are fatal and up to 50% have severe sequelae. The incidence of clinical disease in the Torres Strait outbreak appeared to be about 1 in 20.

Following the cases on Badu Island, inactivated vaccine was offered to all the residents of the central and northern Torres Strait islands.[13] Nearly 9,000 doses of vaccine were administered and 88% of residents who commenced the vaccine regimen received at least two doses. Subsequently, sentinel pigs on Saibai Island in the northern Torres Strait seroconverted to JE in early 1996[14] and 1997 (J. Hanna, D. Phillips, J. Lee, unpublished data), demonstrating that continuing JE virus activity was occurring in the vicinity.

Continuing activity

Further JE virus activity has recently occurred in northern Australia in early 1998 with two human cases,[15] one in an unvaccinated child from Badu Island, and the other in a fisherman from the Mitchell River area of Cape York (S. Ritchie, J. Hanna, and D. Phillips, unpublished data). The latter represents the first case of JE infection on mainland Australia. Sentinel pigs from Badu, Saibai, Mabuiag, Moa (St Pauls), Darnley and Stephen Islands were also found to have seroconverted to JE virus (J. Lee and D. Phillips, unpublished data). This suggested that JE virus activity may have been relatively widespread during the period between January and March in the central, eastern and northern Torres Strait islands. The first seropositive pigs from near Bamaga on mainland Australia were found in late March/early April, 1998. This latter observation, together with the human case from the Mitchell River area, suggests that JE virus may have become epizootic, or perhaps even enzootic, in northern Australia.

Source of virus

Ten virus isolates were obtained during the 1995 outbreak on Badu Island, two from subclinical human infections and eight from *Culex annulirostris* mosquitoes.[16] Sequencing studies have shown that these isolates were almost identical, suggesting that the outbreak originated from a single source, and that they were most closely related to a 1970 isolate from Kuala Lumpur and a 1981 isolate from Bali.[17] Isolates of JE from *Cx. annulirostris* mosquitoes trapped in Papua New Guinea in 1997, and from pig blood collected in the Torres Strait in 1998 have still to be examined for their relationship to the isolates obtained during the 1995 Badu outbreak.

Regional spread

Studies have suggested that the most likely source of the outbreak in the Torres Strait was Papua New Guinea;[17,18] it is less than 5 kilometres between some of the northern Torres Strait islands (for example, Saibai Island) and the coast of Western Province, Papua New Guinea. Seroepidemiological studies have been carried out on human and porcine sera collected from various sites in Papua New Guinea. Using a competitive ELISA test,[18] antibodies to JE have been shown to be present in sera collected in the Daru speaking area of Western Province in 1989 with a seropositivity rate of 21%. JE virus activity seems to be increasing in the Upper Fly area of Western Province, with 8% seropositivity in 1990-91 to 24% seropositivity in 1993. Antibody to JE was also detected in the Kareema region of Gulf Province and Lake Kutubu in Southern Highlands Province. Antibodies to JE could not be found in northern or eastern Papua New Guinea. Almost all pig sera collected in 1995 and 1996 from various locations in Western Province were JE antibody positive.

The first two recognised human cases of JE in Papua New Guinea occurred in 1997 in the Upper Fly area of Western Province, one of which was fatal. The patients were admitted to Rumginae Health Centre, a mission hospital near Kiunga. Subsequently a mild case of JE in a young male presenting with a severe headache and vomiting occurred in February/March 1998 (J. Oakley, S. Flew et al, personal communication). It is possible that other cases have occurred in Western Province, but because of the paucity of medical facilities and the inability to undertake laboratory diagnoses, the patients either died in the villages without medical help, or were diagnosed by local clinics as having cerebral malaria. There has been a major drought in Papua New Guinea since mid-1997 which has seen many rivers either dry into stagnant pools or have a significantly reduced water flow. This in turn has resulted in a significant increase in mosquito breeding in the Upper Fly area which may have contributed to the two human cases.

The situation in the eastern Indonesian Archipelago including Irian Jaya is less certain. A probable case of JE was reported from Irian Jaya in 1996,[19] and there have been reports of finding JE antibody in pigs from Timor and Irian Jaya.

The emergence of JE virus in the Torres Strait was unexpected, occurring over 3,000 kilometers from the nearest known focus of human infections in Bali and Lombok. How the virus spread eastwards to the Australasian region has still to be determined, but if the seropositivity of pigs in Timor and Irian Jaya is correct, it would appear that the most likely explanation is that JE has gradually extended its range eastwards during the past 15 - 20 years through bird/pig-mosquito cycles.

Future outlook

The seroepidemiological results from Papua New Guinea suggest that JE virus may now be enzootic in Western Province.[18,20] This poses a significant threat to Australia, as suitable vector mosquitoes (*Cx. annulirostris*) and vertebrate hosts (ardeid water birds) are widely spread throughout most of the Australian mainland, and wild pigs are relatively common in parts of eastern Australia, particularly in the south-west of Cape York. If JE virus spreads to Cape York or elsewhere in northern Australia, the virus could become established in small enzootic foci and could subsequently spread in mosquito-wild pig/ardeid bird cycles to more populous areas further south.[20]

Murray Valley encephalitis and Kunjin viruses

Murray Valley encephalitis (MVE) and Kunjin (KUN) viruses are best considered together as both are encephalogenic. They also share avian vertebrate hosts and mosquito vectors, although they tend to display different epidemiological patterns.[1,21] MVE virus is enzootic in northern Western Australia and in the Northern Territory, and possibly in northern Queensland where clinical disease appears to be less common. Its occurrence in southeastern Australia is very rare, and is believed to follow an unusual series of extreme weather conditions. KUN virus has a much wider distribution extending over most of tropical Australia, eastern Queensland and with occasional spread into south-east Australia. Only about 1 in 1,000 to 1 in 2,000 infections with MVE virus results in clinical disease. Of those that do, 25% are fatal and a further 25% result in permanent sequelae. The clinical disease is generally referred to as 'Australian encephalitis' and is very similar to the disease caused by JE virus.[22,23] Infection with KUN virus is generally milder, often non-encephalogenic and is not life threatening. It should be referred to as 'Kunjin virus disease' to distinguish it from encephalitis caused by MVE virus. KUN virus infections which are non-encephalogenic usually present as a febrile disease, often with polyarthralgia.

Recent activity

There have been 25 cases of encephalitis caused by MVE virus since 1990 (14 from Western Australia, 9 from the Northern Territory and 2 from Queensland), nine of which were fatal. Over the same time period, eight patients with febrile illness, some with mild encephalitis, have been diagnosed with KUN virus; three from Western Australia, three from the Northern Territory and one each from New South Wales and Victoria.[4,24] The most recent human infections due to both viruses were reported in 1998 from Western Australia; a case of severe encephalitis due to MVE virus in a 5 year old Aboriginal child from a community near Wyndham, and a case presenting with a febrile illness and polyarthralgia due to KUN virus in a 17 year old Aboriginal female from Kununurra (D. Smith and A. Broom, unpublished data).

Surveillance

Sentinel chicken flocks are employed as a means of early warning of MVE virus activity by testing the chickens for seroconversion to MVE. Sentinel flocks are maintained over the summer season in northern Victoria, southern and western New South Wales, and all year round in northern Western Australia and Northern Territory. South Australia and Queensland do not undertake surveillance for MVE virus. The results of sentinel chicken monitoring are published regularly in *Communicable Diseases Intelligence*. Significant levels of MVE virus activity have been reported from northern Western Australia and Northern Territory in most years between 1990 and 1997. Particularly high levels of MVE activity were observed in 1992-93 following very heavy rainfall and widespread flooding, and in the following year, 1993-94, possibly due to a spill-over of virus still in the environment. Virus isolation rates from mosquitoes in northern Western Australia have tended to parallel sentinel chicken seroconversions, with more than 200 isolations from the Kimberley region in 1992-93. Virus carriage rates of 1 in 100 or higher have been recorded in *Cx. annulirostris* mosquitoes.[24] No evidence of MVE activity has been observed in the chicken flocks maintained in New South Wales or Victoria since 1990, and virus has not been isolated from pools of mosquitoes trapped at various sites in the two States.

Recent research

Recent molecular epidemiological results have shown that there have been two genetic lineages of MVE virus. The first lineage included the prototype strain, the first mosquito isolate from Mitchell River Mission (Kowanyama), and some of the isolates from the 1974 outbreak in south-east Australia, after which it seems to have disappeared. The second genetic lineage included the first human case from Western Australia (1969), some of the isolates from the 1974 outbreak, and all isolates from throughout northern Australia since 1974. MVE virus has also recently been isolated from male *Ae. tremulus* mosquitoes.[25] This finding has strongly suggested that vertical transmission in the eggs of *Aedes* spp. may provide a mechanism for virus persistence in arid tropical areas, which had been suggested earlier by Marshall[21] and, more recently, as an explanation of the epidemiological patterns of human disease in the Kimberley after completion of the Ord River dam.[26]

Ross River virus

Ross River (RR) virus causes a syndrome known as epidemic polyarthritis.[27] To distinguish it from other arboviruses causing a similar syndrome, the clinical disease should be referred to as Ross River virus disease. RR virus is one of the most frequently isolated arboviruses in Australia and has been obtained from more than 30 species of mosquito belonging to six genera.[1,3,4] Similarly, RR virus disease is the most common arboviral disease in Australia. It has been reported from all States of Australia, and from Papua New Guinea and the Solomon Islands. A single major epidemic also occurred in various Pacific Islands in 1979-1980;[27] this was the largest epidemic to be

recorded, affecting more than 50,000 people, and almost certainly arose from virus imported into the region by a viraemic traveller from Australia. Although sporadic cases occur widely, particularly in coastal areas of Australia and in inland northern Australia, epidemic activity is commonly associated with heavy rainfall events and flooding, or with high tides inundating salt marshes and coastal wetlands. In general, epidemic activity is more often observed in temperate areas with sporadic cases at other times, whereas in north-eastern, tropical Australia transmission occurs throughout the year.

Recent activity

Major outbreaks caused by heavy rainfall and/or high tides occurred in:

- the south-west of Western Australia in 1991-92 and 1995-96;
- Victoria and South Australia in 1993 and 1997;
- New South Wales in 1996 and 1997; and
- Queensland in 1996.

Increased virus activity in rural areas has resulted in the 'intrusion' of RR virus into major metropolitan areas of Australia; first Perth in 1989, then Brisbane in 1992, and most recently, in Sydney and Melbourne in 1997.[4, 28] Over half (63%) of the reported cases since 1991 occurred in Queensland. The average number of cases reported nationally each year is approximately 4,800 with a maximum of 7,802 notifications in 1996 and a minimum of 2,602 notifications in 1995.[29] While distinct epidemic activity is clearly demonstrable in temperate areas, some uncertainty must exist in reporting RR virus cases in endemic situations when based on an IgM response in a single serum specimen as the IgM may represent past infection in a person who currently has another disease.

The disease

Clinical RR virus disease occurs most commonly in adults 20 to 50 years of age; clinically apparent infections are rare in children. The disease is characterised by marked arthralgia and myalgia with a true arthritis in over 40% of patients. The joints of the extremities are most commonly affected, but spinal involvement is also relatively common. Anorexia and headache may occur, and lethargy is usual.[30,31] About 50% of patients have fever or rash, which is usually maculopapular involving the trunk and limbs. While most patients are well enough to return to work within a month of onset of symptoms, a significant proportion of patients suffer residual arthralgia lasting more than a year.[30,32]

Recent research

Several major epidemiological features of RR virus infection have been elucidated since 1990, including the possible vertical transmission in desiccation-resistant mosquito (*Aedes* spp.) eggs,[33,34] and the description of distinct topotypes of RR virus (genetic variants within defined geographic regions).[35,36] The finding of RR virus in male mosquitoes[33] suggests that vertical transmission in desiccation-resistant eggs of *Aedes* spp. mosquitoes may provide a mechanism by which virus can persist for long periods in the environment. This would explain the rapid onset of cases following heavy cyclonic rainfall and flooding in arid regions. More recently, RR virus (and Sindbis virus) isolations have been made from adult *Ae. camptorhynchus* mosquitoes reared from field collected larvae.[34]

Molecular epidemiological studies of different isolates of RR virus have shown distinct topotypes, with the prototype strain and a few related strains comprising one topotype restricted to Queensland but absent since about the mid-1970s (topotype 1); a major topotype (topotype 2) in eastern Australia, Northern Territory and northern Western Australia which also gave rise to the Pacific Islands outbreak; and a Western Australian topotype (topotype 3) which was largely restricted to the southern half of Western Australia. Prior to 1996, outbreaks of RR virus disease in southern Western Australia were of topotype 3 and transmitted principally by *Ae. camptorhynchus* mosquitoes. However since 1996, topotype 2 has emerged as the cause of the largest (1995-96) observed epidemic in southern Western Australia and was transmitted by *Ae. vigilax* mosquitoes. Thus it appears that topotypes may not only vary from each other by genetic and sometimes antigenic characteristics, but also by transmissibility in different vector mosquito species.

Marsupials such as the Western grey kangaroo are believed to be the major vertebrate hosts of RR virus,[1,27] but other species may play a role. There is growing evidence to suggest that horses may act as amplifier hosts in peri-urban areas. These hosts are all sedentary species and, as birds are not involved in RR virus ecology, it is difficult to understand how single genetic types are maintained over wide areas. Recent evidence, however, has indicated that fruit bats might act as vertebrate hosts in some areas,[37] thus providing a means of virus dispersal.

International outlook

RR virus has considerable potential for spreading to other countries. As noted above, the largest recorded epidemic occurred in the Pacific Islands (especially Fiji, Samoa, Cook Islands, New Caledonia, Wallis and Futuna) in 1979-80 during which more cases were recorded than have been reported in Australia over the past decade. More recently, servicemen from the United States of America taking part in a joint exercise at Shoalwater Bay in eastern Queensland were infected, and at least one serviceman developed clinical disease on his return home.[38] While it is probable that there have been many other instances of infected people carrying RR virus to other countries, particularly in south-east Asia, the Pacific and the United States of America, without any subsequent virus transmission, this nevertheless indicates the potential for the virus to spread via viraemic humans.

Barmah Forest virus

Barmah Forest (BF) virus is an alphavirus which also causes a syndrome similar to epidemic polyarthritis.[31,39,40] To distinguish it from RR virus infection, it is recommended that the disease be referred to as Barmah Forest virus disease. The clinical features of BF virus disease are very similar to those of RR virus infection although the rash tends to be more florid. If it is vesicular, it suggests the infection is due to BF virus. True arthritis does occur but is less common than with RR virus infection. Like RR virus, BF virus may also lead to chronic illness in some patients, but little is known of the incidence or length of time that symptoms persist.[40] BF virus has been reported from all

States on mainland Australia, but has not yet been found in Tasmania or Papua New Guinea.[1,4] It is the most recently recognised of the Australian mosquito-borne human pathogens.

The virus was first isolated from mosquitoes trapped in 1974 from the Barmah Forest of northern Victoria[41] and from mosquitoes collected in south-west Queensland.[42] Although early isolations in the mid 1970s were obtained from various mosquito species across a wide geographic area including Victoria, Queensland and Northern Territory, BF virus was not associated with human infection until 1986 and with human disease until 1988. The first recognised epidemic of Barmah Forest disease occurred at Nhulunbuy in the Northern Territory in 1992 concurrently with an epidemic of RR virus.[43] Subsequent epidemics of BF virus disease have occurred in Western Australia in 1992-93[44,45] and on the southern coast of New South Wales in 1995.[46] This latter outbreak is the largest so far recorded with over 200 serologically confirmed cases. There has been a significant increase in the detection of BF virus disease from most parts of Australia over the past 5 - 7 years. This is partly due to a greater awareness of the virus by the medical profession and diagnostic laboratories, and to the availability of diagnostic reagents. It is believed that up to 10% of cases presenting as epidemic polyarthritis may be caused by BF virus, but insufficient serological data are presently available to support this.

One of the most extensive mosquito collection programs for arbovirus isolation in Australia has been undertaken in Western Australia since the early 1970s, but BF virus was only isolated for the first time from Western Australia in 1989.[47] The first isolations were from mosquitoes trapped at a remote community in the south-east Kimberley region. It was not recorded again until 1992 when the first clinical cases were observed in central and northern Western Australia.[44] Subsequently the virus spread to the south-west of Western Australia in 1993.[45] These findings suggest that the virus has only recently emerged in Western Australia.

Outbreaks of BF virus disease have sometimes been associated with concurrent outbreaks of RR virus disease, and virus isolations have been made from the same mosquito species.[3] Little is known, however, of the ecology of BF virus. It is believed that marsupials may play a role as maintenance hosts, but the genetic similarity between strains[48] suggests that a more mobile host could be involved.

Although BF virus had been classified as the sole member of the seventh serological group in the Alphaviridae, the complete nucleotide sequence of BF virus has only recently been determined, demonstrating that it is genetically distinct, but most closely related, to RR and Semliki Forest viruses.[49]

Other arboviruses

A number of other arboviruses cause occasional human infections. These include the flaviviruses Kokobera, Stratford, Alfuy and Edge Hill viruses; the alphavirus Sindbis virus; and the bunyaviruses Gan Gan and Trubanaman.[1] Of these, Kokobera, Alfuy, Edge Hill, Sindbis and Gan Gan have been associated with mild human disease, usually either polyarthralgia or febrile disease with or without a rash.[1,2,4] Recent studies with Sindbis virus have been particularly interesting in that a new genetic lineage of Sindbis has been found in the south-west of Western Australia (L. Sammels, S. Saleh, M. Poidinger, J. Mackenzie, et al unpublished data). This new lineage is quite distinct from the other recognised lineages, the African-European and Asian-Australian lineages, but is closer to the African-European lineage.[50] From a disease point of view, this is potentially important as the African-European lineage has been associated with large epidemics of febrile disease in northern Europe and southern Africa.[51] In addition, a new Sindbis-related virus has been isolated from mosquitoes in New South Wales which is genetically distinct from the other three lineages, but nothing is known of its properties or distribution at this time (S. Saleh, M. Poidinger, R. Hall, R. Russell et al, unpublished data).

There is considerable anecdotal evidence suggesting that another, unknown arbovirus may be associated with polyarthritic disease in Australia. During epidemics of RR virus disease over the past few years, a significant proportion of patients have presented with symptoms identical to RR virus infection. However, the patients have been serologically negative to RR virus and to all other Australian arboviruses known to be associated with human infection. Nevertheless this unknown virus has the same epidemiological pattern as RR virus.

Another alphavirus, Chikungunya virus is endemic in much of south-east Asia, including parts of Indonesia, and is a potential threat for introduction through a viraemic traveller. Thus possible arboviral disease should be considered in patients presenting with a febrile disease with or without polyarthralgia and/or rash and who have a recent history of travel.

Global warming, environmental factors and emergence of arboviral diseases

Considerable attention has been drawn over the past few years to the possible effects of global warming on human health. Of the infectious diseases, those most likely to be affected by global warming are diseases which are transmitted by insect vectors, and especially the mosquito-borne arboviruses. The importance of weather in the genesis of outbreaks of human arboviral disease in Australia has been widely recognised. In particular heavy rainfall and flooding may result in outbreaks of MVE. Also these and other environmental factors, such as rising sea levels may lead to greater tidal penetration of coastlines and an increased incidence of RR virus.[52-57] Models for predicting the rare occurrences of MVE outbreaks in southern Australia based on El Nino/Southern Oscillation (ENSO) meteorological conditions have been described.[58] However, arbovirus transmission cycles are complex and relatively poorly understood in Australia, particularly with regard to the environment. Thus, the public health response to the threat of increased activity of these viruses must include further research into their ecologies and to the environmental conditions that predispose to outbreaks.

Finally, over the past few years, increasing concern and attention has been directed at the problems and issues associated with new and emerging diseases. Many factors or combinations of factors can contribute to disease emergence, a significant proportion of which can influence

the incidence and spread of arboviruses. Some of these factors have been commented on above as they pertain to the Australian situation. This includes the movement of infected people as a means of introducing dengue into Australia, or exporting RR virus to the South Pacific. Also environmental changes resulting from human activities such as water entrapment and irrigated agriculture may affect the incidence of MVE and other viruses. These are described more fully elsewhere.[59] The importance of an effective surveillance and monitoring system cannot be over emphasised; it is essential not only for Australia, but as part of an international network for providing a global early warning system of the emergence of a new disease, or the spread of known disease. In conjunction with surveillance, there is also a need for rapid, widespread communication; surveillance information is only as good and as useful as the speed at which it can be disseminated. We believe Australia has an important role to play internationally in helping to promote and assist international surveillance and information exchange for arboviral diseases.

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Editor's column

With this issue, we commence a regular column from the Editor to introduce the journal contents and to provide commentary on current communicable diseases topics.

CDI is the journal of the Communicable Diseases Network Australia New Zealand. It aims to provide up to date information on the public health aspects of communicable diseases in Australia, through a combination of peer-reviewed articles, short outbreak reports, commentaries, and surveillance data and reports. Peer review was introduced at the beginning of 1996 and, since early 1997, the journal has been listed in *Index Medicus* and MEDLINE. *CDI* has a circulation of approximately 3,500 from both within Australia and overseas. Our readers include medical practitioners, microbiologists, nurses and environmental health officers.

Since October 1997 the journal has been published monthly in hard copy. It is also available on our website, the address for which is published on the back cover of *CDI*. Tabulated national notifiable diseases surveillance data are also published fortnightly on the website.

Contributions of articles, outbreak reports, commentaries, and letters are welcome and should be sent to the Editor at the address detailed on the back cover of *CDI.*

This issue of *CDI* focuses on arboviral infections in Australia. Arbovirus diseases are emerging in both Australia and the neighbouring region, with spread of these infections to areas in which they have been formerly rare or unknown. Examples are the recent occurrence of the first case of Japanese encephalitis acquired on the Australian mainland, the continuing outbreak of dengue type 3 in Cairns, and the acquisition of Ross River virus infection in western Sydney in 1997. The article by Mackenzie et al provides a useful and timely review of arboviral infections in Australia, and the neighbouring region, in recent years. Amin et al discuss the investigation of cases of RR virus infection in western Sydney in 1997. Merritt et al report on the first case of MVE acquired in Alice Springs in over 20 years. Three cases of dengue imported from Thailand are reported by Playford et al, reminding us of the need to provide good travel health advice to Australian's travelling in dengue endemic countries and of the importance of prompt notification of cases which occur in the dengue receptive area of Australia. Mein's article illustrates the complexity of diagnosis of flavivirus infection and the importance of considering the clinical, epidemiological and laboratory information for each patient before reaching a final diagnosis. For your interest, we have also reproduced an article about the recent dengue 3 outbreak in Cairns, previously published in the Tropical Public Health Unit Newsletter. Also in this issue, Holland describes the first case of infantile botulism reported in Australia since the establishment of the current NNDSS in 1992, and Ferson's letter reminds us of the need to consider hepatitis A when discussing sexually transmissible diseases.

Ross River virus infection in the north-west outskirts of the Sydney basin

Janaki Amin[1], Linda Hueston[2], Dominic E Dwyer[2], Anthony Capon[1]

Abstract

In early 1997, 69 cases of Ross River virus infection were reported in the north-western outskirts of Sydney. This represents a substantial increase over the maximum of 12 cases reported in any one year since 1991. The majority of cases (71%) are thought to have been locally acquired. This is the first reported outbreak of Ross River virus infection in this area and highlights the need for metropolitan health services to be vigilant about a disease that has primarily been associated with rural and semirural areas in New South Wales. *Comm Dis Intell* 1998;22:101-102

Introduction

Ross River virus (RR virus) is a mosquito-borne arbovirus endemic in various regions of Australia[1]. The area covered by the Western Sector Public Health Unit (WSPHU), from western metropolitan Sydney to the western and north-western rim of the Sydney basin, comprises urban, semi-rural, rural and wilderness environs. Historically, there has been no published RR virus activity in this area; WSPHU has received occasional notifications for cases who had travelled to areas of known RR virus activity. Early in 1997, staff of the WSPHU became aware of an increase in laboratory RR virus notifications. On investigation, it became apparent that some cases may have acquired the infection within the Sydney basin. A prospective descriptive investigation of RR virus notifications was then undertaken.

Methods

For the purpose of this investigation a case was defined as any person resident in the area covered by the WSPHU, with RR virus IgM positive serology, notified by a laboratory or doctor to the WSPHU between 1 January 1997 and 31 May 1997, with an onset of symptoms after 31 December 1996.

The referring doctors for cases were interviewed by telephone by the WSPHU and Department of Virology staff, and details were obtained about the presentation of symptoms, likely onset and travel history of the case. The cases were then contacted and interviewed either in person or by telephone, to clarify symptoms, date of onset and travel history. Where the case was not contactable, information obtained from the referring doctor was used for analysis.

Results

Since 1991 the maximum number of RR virus infections notified to the WSPHU in any one year had been 12. A total of 69 cases were identified during the five month study period, representing a crude incidence rate of 7.4 per 100,000 population. Histories were obtained from 68 (99%) referring doctors and 60 (87%) cases. The majority of cases, 49 (71%), recalled being bitten by mosquitoes within the Western Sector area in the 21 days prior to becoming symptomatic; 15 (22%) reported histories of travel outside the Western Sector in this time period.

Figure 1. Notifications of Ross River virus infection by week of onset, north-western Sydney, January-May 1997

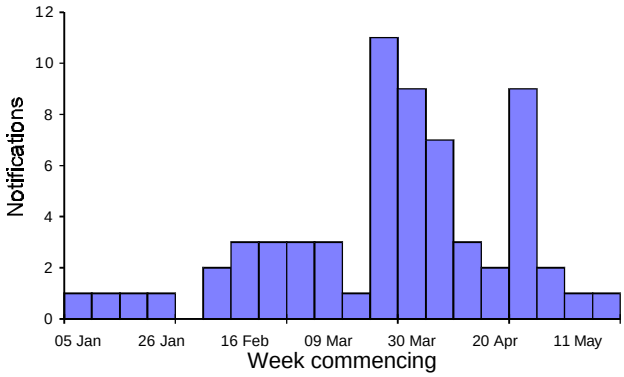
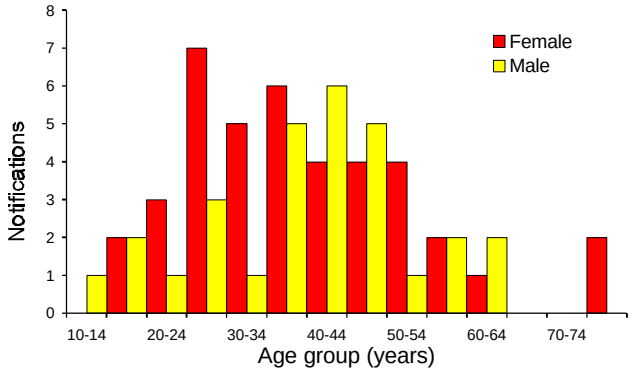


Figure 2. Notifications of Ross River virus infection by age group and sex, north-western Sydney, January-May 1997



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The onset of symptoms for most cases occurred in April 1997, with peaks in late March and early May (Figure 1). Most cases were in the 25 to 49 years age range (Figure 2). The male:female ratio was 1:1.4. All cases for whom a clinical history was available reported having joint pains; a majority (57%) also reported fatigue as a prominent symptom. Rash and fever were less commonly reported (21% each).

Discussion

The number of notifications received by the WSPHU between January and May 1997 represents a substantial increase in cases for this area. Previously, all cases for which the WSPHU have records were likely to have been acquired outside the area. In this outbreak the vast majority of infections were likely to have been locally acquired. Most cases lived in the semirural parts of the area covered by the WSPHU where conditions for RR virus activity are favourable, however a few cases with no travel history did occur within the Sydney metropolitan area. These findings were limited by the reliance of defining a case on a single IgM result. The high false positive rate of some IgM assays,[2] and the persistence of virus specific IgM,[3] made diagnosis and time, and therefore place, of infection difficult to ascertain. However, the substantial increase in notifications compared to previous years seems to indicate that there was an increase in local RR virus activity.

The potential for RR virus activity in semi-rural areas to spread into major metropolitan centres has been seen in outbreaks in Perth and south-west Western Australia. [4,5] Lindsay et al., postulated that in the Perth outbreak, transmission within urban areas was facilitated by the movement of infected humans.[4] The incidence of infection in the metropolitan area in this western Sydney outbreak is unlikely to have been high enough for human-mosquito-human transmission to occur, and more likely to have arisen from animal host-mosquito-human transmission in the local area or unreported travel through endemic areas.

A smaller proportion of cases experienced fever and/or rash than has been documented in some previous studies.[1,3] There were anecdotal reports of people believing they could not have RR virus infection if they did not have a fever and rash. Future health promotion messages regarding RR virus therefore should state that fever and rash are not always seen. Health promotion interventions should commence by early summer; however, considering the March and May peaks of onset in this outbreak, it may be worthwhile to also target interventions to coincide with events such as school holidays and public holidays, when people spend long periods of time outdoors and are therefore more likely to be bitten by mosquitoes. Health promotion interventions (including initiatives such as minimising mosquito breeding sites and education programs) and mosquito species and population studies, are currently being undertaken in western Sydney. The interventions were targeted for the 1997/98 mosquito season. Only four cases with onset between January 1998 and April 1998 have been notified to the WSPHU to date.

This is the first reported outbreak of RR virus in western Sydney. The reason why an outbreak occurred in this area in 1997 is difficult to determine. A combination of rains in late January and early February, the presence of appropriate mosquito vectors and macropod hosts, the large number of horses on farms in the area (possibly acting as amplifying hosts), and the increasing urbanisation around the natural and artificial waterways are likely to have contributed to this outbreak. This outbreak highlights the need for public health staff to be vigilant regarding RR virus, even in areas with no previous documented history of RR virus activity.

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A presumptive case of fatal Murray Valley Encephalitis acquired in Alice Springs

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Abstract

A presumptive case of Murray Valley Encephalitis (MVE) acquired in Alice Springs in March 1997 is reported. The patient subsequently died in Mackay. The diagnosis of Murray Valley Encephalitis was supported by the detection of flavivirus IgM in cerebrospinal fluid. Low titres of IgM specific to Murray Valley Encephalitis and Alfuy were detected in a single serum sample. The patient's travel movements indicate that his infection was acquired in the Alice Springs vicinity. This conclusion was further supported by the detection of Murray Valley Encephalitis activity in sentinel animals in the area and by the presence of large numbers of the principal mosquito vector of Murray Valley Encephalitis in the Northern Territory. *Comm Dis Intell* 1998;22:103-104

Introduction

Murray Valley Encephalitis (MVE) is an arboviral disease endemic to northern areas of the Northern Territory and Western Australia. The last Australia-wide epidemic in 1974 resulted in 58 cases.[1] Since then notifications have only occurred in northern Australia with two confirmed cases in Western Australia in early 1997 and a further case in early 1998.

Case Report

A 60 year old male was admitted to the Mackay Base Hospital on 3 April 1997 with severe headache, fever and confusion. He described a two week history of myalgia and arthralgia that had begun while he was in Alice Springs. He had then flown to Mackay with an overnight stop in Cairns. Soon after admission he had a generalised fit and was transferred to the Intensive Care Unit. The initial results included a normal CT brain scan and a marked monocyte response (white blood cells, WBC, 540/mm^3) in his cerebrospinal fluid (CSF). CSF was also submitted for Gram stain, bacterial culture, and antigen studies for *N. meningitidis*, *H. influenzae*, and *S. pneumoniae*, all of which were negative. A presumptive diagnosis of herpes encephalitis was supported by the detection of herpes simplex virus (HSV1) antigen in a lesion on his lip and he was commenced on intravenous Acyclovir. However, polymerase chain reaction (PCR) testing for HSV antigen in CSF was subsequently negative. Serological studies for IgG and IgM to Ross River virus, Barmah Forest virus, MVE, Kunjin, Alfuy, Kokobera, Stratford and Edge Hill were all negative in blood collected on 3 April. An EEG on 7 April detected changes suggestive of a diffuse encephalopathy.

The patient's condition initially improved, but deteriorated on 10 April when he developed a dense right hemiplegia, increased confusion and a productive cough. He was intubated and managed for aspiration pneumonia. A repeat lumbar puncture demonstrated a persistent monocytosis (WBC 30/mm^3). CSF was submitted for Ziehl-Neelsen stain, cryptococcal antigen studies, and complement fixation for measles, HSV and varicella zoster virus; all CSF tests were again negative.

A CT brain scan on 11 April detected a focal low density region in the left internal capsule consistent with a left middle cerebral artery infarct. When the scan was repeated on 16 April this area was considerably larger and was consistent with a massive infarct involving the left middle and anterior cerebral arteries. He continued to deteriorate and died on 25 April. A post-mortem examination was not performed.

Subsequent investigations

Flavivirus IgM was subsequently detected by enzyme immunoassay (EIA) in serum collected on 9 April, and haemagglutination inhibition assays of serum fractions demonstrated low titres of MVE specific and Alfuy specific IgM (1:40 for both). Specific IgM to Kunjin virus was not detected.

Arboviral serology was then performed on the CSF sample from 3 April. Flavivirus IgM was detected, however there was insufficient sample for virus specific assays. Virus isolation was unsuccessful from both the CSF and the serum samples.

The patient was a resident of Alice Springs and had not travelled elsewhere in the Northern Territory for many months prior to his illness. He worked at a site 8 km south of Alice Springs in the vicinity of known mosquito breeding grounds, and fellow workers at this site had recently complained of mosquito problems (O. Harris, personal communication). He also reported receiving numerous mosquito bites while watching a football match in Alice Springs in the days prior to his illness but there were no other complaints about mosquitoes at this match or at any other locations within the town area.

As the incubation period of mosquito-borne arboviral encephalitides, including MVE, is 5-15 days,[2] it is likely

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that his infection was acquired in the vicinity of Alice Springs. MVE is endemic in the 'Top End' of Northern Territory but had not been detected south of Tennant Creek since 1974.[3] A sentinel chicken flock was established on the southern outskirts of Alice Springs in November 1996, and although no evidence of MVE activity was detected when the flock was bled on 17 March, seven of nine chickens had seroconverted to MVE virus when bled on 22 April.[4] Subsequent investigations on sera from sentinel cattle provided further evidence of flavivirus activity in the Alice Springs region during this period. No cattle were positive when tested in January and February, one had positive flavivirus serology by ELISA on 17 March and five had seroconverted to MVE virus by 15 May.

The suspected principal vector of MVE in the Northern Territory is the mosquito *Culex annulirostris*.[3] Following heavy rains across much of the Northern Territory in early 1997, mosquito surveillance detected high numbers of *Cx. annulirostris* in Alice Springs. Numbers began to rise in early February and reached a peak of up to 1,100 per CO_2 baited trap per night by late February. Numbers remained high until mid-March then steadily declined to below 100 per trap by May.

The Territory Health Services issued a MVE virus alert for the entire Northern Territory, including Alice Springs, in early May immediately following the detection of MVE activity in sentinel chickens in Tennant Creek. Positive serum samples from the Alice Springs sentinel flock had not been confirmed at that time.

Discussion

Infection with MVE virus is the most likely explanation of this patient's illness and death, however this diagnosis could not be confirmed and there were a number of atypical features. The detection of specific IgM in CSF is one of the laboratory diagnostic criteria published by the Centers for Disease Control and Prevention, Atlanta, for arboviral encephalitis.[5] In this case flavivirus IgM was detected in CSF but there was insufficient sample for virus specific assays. The only flaviviral antibodies detected in serum were low titres of IgM to MVE and Alfuy in a single sample. Taken in conjunction with the CSF result, this supports the likelihood of MVE infection, as Alfuy is not a recognised cause of human encephalitis. MVE antibodies were not detected in serum taken on the day of admission at which time the patient had been unwell for two weeks. IgM antibodies would often be present in serum by this stage, but their appearance may occasionally be delayed.

The patient presented with an acute encephalitic illness consistent with MVE. However, the extensive cerebral infarct that subsequently developed was unusual and we are not aware of other case reports of cerebral infarcts in adults with MVE.

The patient's movements suggest that the infection was acquired in the Alice Springs vicinity and this is supported by the detection of MVE activity in sentinel chickens and cattle, and the presence of high numbers of the vector *Cx. annulirostris* in the region.

In this instance, the detection of seroconversions in sentinel animals was such that human transmission appears to have occurred before a MVE virus alert could be issued. This emphasises the need for resources to allow more frequent bleeding and testing of sentinel animals so that warnings can be issued as early as possible.

This presumptive case of MVE is the first recognised as acquired in Alice Springs since 1974[3] and it is a reminder that MVE may be present in central Australia following favourable environmental conditions. This patient's subsequent travel also reinforces the importance of interstate coordination of arboviral surveillance.

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Dengue or Kokobera? A case report from the Top End of the Northern Territory

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Abstract

In early April 1998, the Centre for Disease Control in Darwin was notified of a possible case of dengue which appeared to have been acquired in the Northern Territory. Because dengue is not endemic to the Northern Territory, locally acquired infection has significant public health implications, particularly for vector identification and control to limit the spread of infection. Dengue IgM serology was positive on two occasions, but the illness was eventually presumptively identified as Kokobera infection. This case illustrates the complexity of interpreting flavivirus serology. Determining the cause of infection requires consideration of the clinical illness, the incubation period, the laboratory results and vector presence. Waiting for confirmation of results, before the institution of the public health measures necessary for a true case of dengue, was ultimately justified in this case. This is a valid approach in the Northern Territory, but may not be applicable to areas of Australia with established vectors for dengue. *Comm Dis Intell* 1998;22:105-107

Introduction

Dengue fever is a flavivirus infection transmitted by the mosquito *Aedes aegypti.* After an incubation period of 7-10 days, a flu-like illness develops with high fevers, chills, myalgia and headaches. Distinctive features include retro-orbital headache and bone pain ('breakbone fever'). Following repeat infection with a heterologous serotype it can be a severe, occasionally fatal illness, causing haemorrhage and shock. The last documented cases of dengue fever in Darwin occurred in 1955. Surveys since 1974 have found no *Ae. aegypti* mosquitoes in the Northern Territory.[1] Proven locally acquired dengue in 1998 would necessitate an expensive program of enhanced human and entomological surveillance, Northern Territory quarantine, and vector control measures.

Case study

On 2 April 1998, the Centre for Disease Control in Darwin received a notification from a local doctor of a suspected case of dengue in a Darwin resident. A 20 year old male had presented to his general practitioner on 17 March 1998 complaining of a two day history of fevers, chills, myalgia, pharyngitis and headache. The illness was short lived; his temperature returned to normal after three days, but he had persistent myalgia and remained tired for a week. He made a complete recovery.

The patient gave a history of recent travel to New South Wales and Queensland, from which he had returned 22 days prior to the onset of symptoms. He denied travelling further north up the Eastern seaboard than suburban Brisbane during this trip. He had not been overseas since 1989 and had not been north of Rockhampton since 1993. Extensive questioning failed to reveal any other recent source of exposure to the vector.

Diagnosis

Dengue serology was ordered because of his travel history. However, on epidemiologic grounds, the illness was most likely to have been locally acquired in the Northern Territory. The clinical illness was not consistent with classic dengue, as there was no bone pain or retro-orbital headache. As the diagnosis was not confirmed, it was decided to repeat the serology results, to ascertain whether there was a fourfold rise in total antibody, prior to implementing a full scale search for a possible vector.

On both 17 March and 2 April 1998, the patient's screening flavivirus IgG by haemagglutination inhibition test showed a titre of 1:160, with a positive dengue IgM and negative Murray Valley encephalitis and Kunjin IgMs by immunofluorescence. However, given the highly variable persistence of flavivirus IgM[2], it was considered that this could have been evidence of old infection, either from 1993 in Queensland or (as an unlikely possibility) from India before 1989. There was no fourfold titre rise in total antibody to support an acute infection, and it was unlikely that, at presentation to his doctor on day two of the illness, the IgM would be already positive.

A more likely possibility was that his test results were due to another flavivirus infection giving a false positive dengue result, as has been documented previously[3]. No serum was left from the first bleed to undertake polymerase chain reaction testing or virus culture. In order to exclude other flaviviruses, the remaining second, and a third specimen were sent to Queensland Health Scientific Services for further testing (Table 1).

The twofold rise in Kokobera IgG titre alone was not significant. However, the presence of moderate levels of Kokobera IgG and Kokobera specific IgM indicated probable Kokobera infection. Virus neutralisation tests were not undertaken.

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Table 1. Results of flavivirus testing from the case in Northern Territory[1]

Test	Date of specimen collection	
	2 April 1998	20 April 1998
Enzyme immunoassay		
Flavivirus IgG	Detected	Detected
Dengue IgM	Not detected	Not detected
Encephalitic flavivirus IgM[2]	Not detected	Not Detected
Non-encephalitic Australian flavivirus IgM[3]	Detected	Detected
Ross River IgG, IgM		Not detected
Barmah Forest IgG, IgM		Not detected
Haemagglutination inhibition (HAI)		
Murray Valley encephalitis	40	20
Dengue 1	80	80
Dengue 2	40	20
Dengue 3	20	20
Dengue 4	20	20
Alfuy	160	80
Kunjin	40	80
Kokobera	80	160
Stratford	80	160
Japanese encephalitis	80	80
Ultra-centrifugation and HAI		
Kokobera IgM		Detected
Stratford IgM		Not detected

1. From the Queensland Health Scientific Services
2. Japanese encephalitis, Murray Valley encephalitis, Kunjin
3. Kokobera, Stratford

Vector monitoring

A vector survey is costly because *Ae. aegypti* is not readily caught in the usual CO_2 baited traps, and time consuming house to house searches of water containers with larvae and adult biting catches are required. While the serological results were pending, overall mosquito activity was monitored by three CO_2 traps set around the patient's house on two occasions in mid April, and the results of ongoing ovitrap surveillance for exotic mosquito species were reviewed. No *Ae. aegypti* or *Ae. albopictus* (another recognised vector of dengue) were detected, and the overall numbers of adult mosquitoes caught at the residence were low.

Discussion

On the basis of these results a presumptive diagnosis of Kokobera infection was made. This flavivirus is known to cause occasional human infection[4] and the clinical illness may resemble dengue, although it has more often been associated with arthralgia.[5] Kokobera has been isolated from *Culex annulirostris* mosquitoes in the Northern Territory[6] and these were the predominant mosquitoes trapped around the patient's house. In addition, there have been ten Kokobera isolates from *Culex annulirostris* during recent mosquito surveys in northern Queensland (D. Phillips, personal communication).

Specific flavivirus serology results, particularly IgM results, are unreliable. They may be elevated for a period of some years following infection, or falsely elevated because of cross reactivity with related but distinctly different flavivirus, or other arbovirus infections, each with very different public health implications. If significant public health action is dependent on a flavivirus result, every effort should be made to confirm the diagnosis, rather than rely on a positive IgM result alone. A fourfold rise in antibody level over the acute phase of illness, with sera tested in parallel to ensure a consistent reading under identical conditions, is required for diagnosis. It is, therefore, very important to obtain repeat blood samples. This approach is suitable in the Northern Territory, but may not be applicable to areas of Australia with established vectors for dengue, where immediate public health action is required. In these areas, other tests, such as polymerase chain reaction, or viral culture may be used to establish the diagnosis quickly.

Conclusions

Because of the high rate of cross reactivity in flavivirus serology, a positive screening test should be interpreted with caution. Specific tests for other flavivirus infections such as Kokobera are not routinely requested. If the patient had had a travel history consistent with vector contact in Queensland, he would have been notified as a case of dengue. However, if he had not travelled to Queensland dengue serology would not have been requested in the first place. This case is a reminder to consider a wide range of diagnostic possibilities when determining the cause of an arboviral infection.

This case also reinforces the importance of ensuring that all factors; laboratory tests, clinical symptoms and epidemiologic data, are consistent before making a diagnosis that has considerable public health implications. This case of 'dengue' was suspect because the clinical illness was inconsistent and there was no entomological evidence that the vectors were present in Darwin. The assumption that this was not dengue was borne out by reference laboratory testing. In the Northern Territory it justified the approach of waiting for the results before vector surveys and control strategies, including human health service alerts, were implemented.

Acknowledgements

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Three cases of dengue 1 virus infection from islands in the Gulf of Thailand

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Abstract

Three Australian tourists who recently travelled to islands in the Gulf of Thailand developed febrile illnesses associated with myalgias, thrombocytopenia, and atypical lymphocytosis. Dengue 1 virus was isolated from all three patients. The patients' clinical features and serological and virological investigations are presented. These cases highlight the need for awareness of dengue amongst travellers and the preventive precautions required when visiting endemic regions. After the urgent exclusion of malaria, dengue should be considered in the the differential diagnosis of febrile persons who have recently returned from endemic regions. *Comm Dis Intell* 1998;22:107-109

Introduction

Dengue fever is endemic throughout southeast Asia. Over the past three years, increased dengue activity has been reported from Malaysia, where over 19,500 cases of predominantly dengue 1 and 2 were notified during 1997,[1] Indonesia,[2] Cambodia,[3] India[4] and the western Pacific.[3,5,6,7] Although the north and central areas of Queensland, which correspond to the distribution of *Aedes aegypti*,[8] are potentially receptive to the establishment of endemic dengue, the virus is not endemic in Queensland. Epidemics are assumed to have arisen from viraemic travellers.[9] Recent outbreaks in Queensland have included an outbreak of dengue 2 in Cairns, commencing in December 1996, and resulting in 201 confirmed cases,[10] and an outbreak of dengue 3, which commenced in December 1997 and has resulted in 165 confirmed cases up to 25 May 1998 (J. Hanna and S. Ritchie, personal communication). Sequencing data of the dengue 3 isolates has shown that the most likely source of the virus was Thailand (D. Phillips, unpublished data)

This report presents three cases of dengue 1 in Australian tourists who recently travelled to islands in the Gulf of Thailand, and discusses the implications of these cases for travellers to endemic areas and for dengue control in Australia.

Case 1

A 57 year old male developed a febrile illness associated with myalgias on 17 October 1997, three days after returning from Ko Chang. He had spent one week on the

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island and sustained numerous mosquito-bites. Over the preceding 6 months he had travelled through southern Spain and northern India without medical problems, apart from self-limited diarrhoea in India. Upon presentation to hospital on the 20 October 1997, investigations revealed mild leucopenia (total white cell count 2.3 $\times 10^9$/L, neutrophil count 1.47 $\times 10^9$/L). Four days later significant thrombocytopenia (platelet count 56 $\times 10^9$/L) and mild atypical lymphocytosis (5% of 2.7 $\times 10^9$/L) developed.

Arboviral serology was performed on serum collected on 21 October and 24 October. Flavivirus IgG was reactive by enzyme immunoassay (EIA) on both specimens. Dengue IgM by EIA was initially nonreactive, but reactive on the second specimen. Cross reactive IgM antibody to the four dengue serotypes was detected following ultra-centrifugation separation and haemagglutination inhibition assay (UC/HI) of the second specimen. Dengue 1 virus was isolated from both specimens.

Case 2

A 39 year old male developed a febrile illness associated with myalgias, bone pain, and vague generalised abdominal pain on the 17 October 1997, five days after arriving on Ko Pha-ngan. He sustained numerous mosquito-bites whilst on the island. For the preceding three weeks he had trekked in the Himalayan area of Nepal without medical problems. He presented to hospital in Australia four days after the onset of symptoms. He developed marked leucopenia (total white cell count 1.4 $\times 10^9$/L, neutrophil count 0.58 $\times 10^9$/L) and thrombocytopenia (platelet count 51$\times 10^9$/L). Seven days after the onset of symptoms, his fevers and symptoms subsided, with the subsequent development of atypical lymphocytosis (10% of 2.1 $\times 10^9$/L). Resolution of cytopenias occurred by day 12.

Flavivirus IgG and dengue IgM was detected from serum collected on 23 October. Specific IgM antibodies to dengue 1, but not types 2, 3, or 4, were detected by UC/HI. Dengue 1 virus was isolated from this serum.

Case 3

A 31 year old female, the partner of Case 2 who also visited Ko Pha-ngan, developed a similar febrile illness on the 22 October 1997, approximately five days after that of her partner. She noted a transient erythematous rash over the trunk. Although her fevers and other symptoms settled within five days, she presented to hospital on day 7 of the illness with a faint petechial rash over her ankles and feet. Investigations revealed mild thrombocytopenia (platelet count 81 $\times 10^9$/L), and atypical lymphocytosis (10% of 4.4 $\times 10^9$/L).

Flavivirus IgG was nonreactive and dengue IgM was reactive by EIA. Cross reactive IgM antibodies to all four dengue serotypes were detected by UC/HI. Dengue 1 virus was isolated from this serum.

Discussion

These three recent cases of dengue highlight several important points.

In 1996, 43 cases of dengue were reported in Australia,[11] including both imported and locally acquired dengue, while in 1997 approximately 171 cases were reported.[12] However, given that the south-east Asian region, including the Gulf of Thailand islands where the three patients visited, are both popular destinations for western tourists and areas of dengue endemicity, it is surprising that dengue is not more frequently diagnosed in travellers returning to Australia.

Those visiting endemic regions should routinely be given pre-travel advice regarding dengue. This is particularly important for those travelling to south-east Asia during the late wet season, September to November, which is the peak time for dengue transmission. Emphasis should be given to the importance of avoiding both day-time active mosquitoes that transmit dengue, as well as night-time active mosquitoes that transmit malaria.

Medical officers to whom returned travellers present should be aware of dengue. In addition to malaria, typhoid, HIV, and rickettsial infections, dengue and other arboviral infections should be considered in febrile returned travellers. The incubation period of dengue fever ranges from 3-14 days, usually 5-7 days, and is typically followed by an abrupt onset of fevers, malaise, retrorbital headaches, myalgias, and bone and joint pains. Other specific symptoms include: a bitter, often metallic, taste; itchy skin or sensation of pins and needles in the skin; and vomiting and diarrhoea. The diagnosis may be suggested clinically, although at a minimum, blood films should routinely be performed to exclude malaria. A variety of haematological abnormalities may be encountered, including leucopenia, thrombocytopenia, and atypical lymphocytosis.[13]

Although serology is commonly used to confirm dengue infection, cross-reactive antibodies may prevent identification of the infecting serotype, as occurred in Cases 1 and 3. The definitive diagnosis of dengue infection requires either isolation of virus, or detection of viral RNA by polymerase chain reaction (PCR) in acute-phase serum specimens. These services are provided by reference laboratories.

The period of viraemia extends from shortly before until the end of the febrile stage of the illness,[14] and cases in potentially receptive areas of Australia should avoid being bitten by day-time active mosquitoes, in order to prevent outbreaks of dengue.

The pathogenesis of dengue haemorrhagic fever relates to sequential infection with heterologous dengue serotypes occurring months to years apart,[15] and patients, particularly children, diagnosed with dengue should be counselled regarding the potential risk of dengue haemorrhagic fever if revisiting areas of dengue endemicity.

Although it is considered unlikely that dengue will become endemic in Australia,[16] large outbreaks can result from imported cases in the dengue receptive areas. Doctors should be alert to the possibility of dengue in travellers from dengue endemic areas in order to diagnose cases early. Prompt notification of suspected cases occurring in the dengue receptive areas is vital to allow rapid public health action to limit the spread of the virus.

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Dengue 3 in Cairns: the story so far

Adapted from the Tropical Public Health Unit - Newsletter No. 24, May 1998; with the permission of the Tropical Public Health Unit, Queensland Health, PO Box 1103, Cairns, Queensland 4870

In early December last year, the Tropical Public Health Unit (TPHU) was notified of an adult resident of the Atherton Tablelands who had a positive screening-test for dengue. The patient had a non-specific illness that might have been dengue but he had not recently travelled overseas. Because the screening-test gives a number of false-positive results, it is TPHU policy to delay an investigation in these circumstances while a more reliable test is undertaken in Brisbane.

Before this result became available however, the patient subsequently informed the TPHU that he had had substantial contact with a travellers' guesthouse on The Esplanade, Cairns North, and several other people at the guesthouse had developed a similar febrile illness. Blood samples were quickly collected from as many of these other individuals as possible and sent to Brisbane for urgent testing; several of these samples tested positive for dengue 3. Because some of these positive people were staff at the guesthouse and had not recently travelled overseas, it was obvious that they had acquired dengue at the guesthouse. Therefore the outbreak was first confirmed on December 12, eight days after the original notification and 16 days after the onset of his symptoms.

Mosquito investigations were commenced immediately. Numerous adult *Aedes aegypti* were found on the premises including several blood-fed females in the rooms of ill people. The interior of the hostel was sprayed with a commercial pyrethroid aerosol spray. Several pot-plant containers containing *Ae. aegypti* larvae were also found and emptied. Properties within several hundred metres of the guesthouse were also surveyed, containers emptied and (with permission) interiors sprayed.

Although we were not able to identify the traveller who imported the dengue 3 virus from overseas into Cairns, the virus has a very similar nucleotide sequence to a dengue 3 virus isolated from a traveller who returned to Australia from Thailand in 1993. This suggests that the current virus was imported from southeast Asia.

A travellers' guesthouse obviously caters to lots of travellers, and we are aware of eight overseas travellers (two of whom contacted TPHU from Spain and Canada via the Internet!) and two interstate visitors who contracted the virus whilst staying in the guesthouse. Because travellers travel, several turned up in other locations in 'dengue-receptive' North Queensland whilst still infectious to *Ae. aegypti* mosquitoes: Innisfail, Mission Beach, Townsville, Magnetic Island and Proserpine. Fortunately no local transmission occurred in these locations, but the inevitable soon happened: spread to other suburbs in Cairns.

In early February TPHU recognised that local transmission of dengue 3 was occurring in Parramatta Park. This is an older, more central suburb with many old Queenslander cottages on small properties. Most of these residences are not screened, and many properties were found to be effectively breeding large numbers of *Ae. aegypti* mosquitoes in rubbish and garden containers in the backyards. The Parramatta Park outbreak was explosive with 60 confirmed cases occurring in February; a considerable number of these cases were either working from home or unemployed and therefore spending long hours at home exposed to infectious mosquitoes.

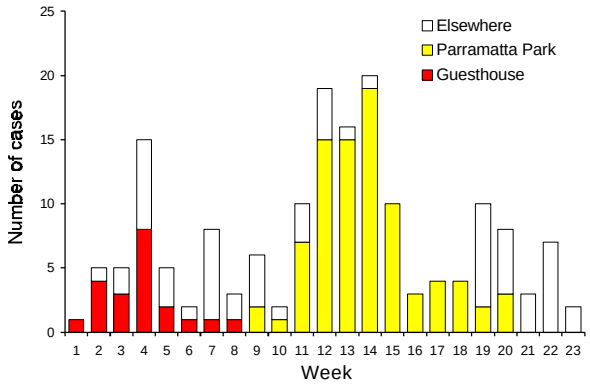
Not many travellers stay in Parramatta Park and therefore it was unlikely that the virus would be taken to other travellers' destinations in North Queensland. However residents of Parramatta Park work, visit and convalesce from dengue in other suburbs of Cairns. Therefore further spread within Cairns was inevitable: Westcourt, Earlville had small numbers of cases in late February. There have been more recent, and also small, foci in Stratford and Holloways Beach; it seems that spread has occurred from the latter suburb to a resort Barrier Reef island and to Machans Beach. Fortunately the virus has not spread to the Torres Strait, where a large outbreak of dengue type 2 occurred a year ago.

The outbreak of dengue 3 in Cairns is now in its sixth month. To 25 May 1998, 165 cases have been confirmed. Of these, 31 (19%) have been hospitalised; although some of these only required an overnight stay for IV fluids, two required ICU care. There has been one case of dengue haemorrhagic fever (in an elderly male who fortunately

only had mild haemorrhage) and one case of dengue encephalopathy (a male in his 20's who collapsed at work, had several seizures and became increasingly unrousable). Clearly those affected in this outbreak are more ill than those affected in last year's dengue 2 outbreak in the Torres Strait.

The outbreak seems to be slowing down, and we hope it will soon be over. That the number of cases has been held down to less than 22 so far is a credit to the hard work of the Entomological and Environmental Health staff of TPHU and the Cairns City Council.

Figure 1. Notifications of dengue 3, from December 1997, Cairns, by location and week



A case of infant botulism in South Australia

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This report documents the first case of infant botulism recorded in South Australia since 1990.

On 25 May 1998 a case of infant botulism was notified to the Communicable Disease Control Branch. The 6 month old female, from a northern country area of South Australia, was admitted to hospital with paralysis and was diagnosed initially on clinical grounds. She had decreased spontaneous movement, reduced anti-gravity movements, no head movements, and gag and cough reflexes were absent. She was intubated, ventilated and given general supportive treatment.

The baby had become unwell the day before with lethargy and difficulty feeding, and was described by the mother as 'being uncomfortable'. The mother also reported constipation occurring some days before. The diagnosis was confirmed serologically and by tests in mice.

The baby was breast fed on demand and solid foods had been introduced over the previous 6 weeks. Foods consumed included commercially prepared apricot and rice, pear, mango and apple, and pumpkin either from jars or tins, sweet biscuits and a baby rice cereal. The infant also ate home prepared chicken and vegetable, and toast with Vegemite. No honey or corn syrup was consumed. The family has a dog and keeps chickens although the baby did not have contact with the animals.

Infant botulism results from spore ingestion and subsequent vegetative growth, and in-vivo toxin production in the intestine by *Clostridium botulinum*. The syndrome affects infants almost exclusively, but can affect adults who have altered gastro-intestinal anatomy and microflora. The illness typically begins with constipation followed by lethargy, listlessness, poor feeding, ptosis, difficulty swallowing, loss of head control, hypotonic extending to generalised weakness (the 'floppy baby') and, in some cases respiratory insufficiency and arrest.

There are many sources of spores, including foods and dust. Honey and corn syrup have been implicated in infant botulism. Environment and food sampling in isolated cases, such as the one reported here, is unrewarding because of the ubiquitous nature of the organism.

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Editorial note

This is the first notification of a case infant botulism in Australia since botulism became a nationally notified disease in 1992. Infant botulism generally occurs between the ages of 2 weeks and 1 year, with 94% of cases occurring at or before the age of 6 months. Clinical severity can range from mild illness with gradual onset to severe respiratory insufficiency and death. Case fatality rates in countries with good paediatric intensive care units are less than 1%. Excretion of *C. botulinum* toxin and organisms can occur in the faeces for extended periods (weeks to months) but no instance of secondary person-to-person transmission has been documented.

An antitoxin is available but is not recommended in the treatment of infant botulism.

Current issues in immunisation

An occasional report series from the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS)

National immunisation coverage - interpreting the first three quarterly reports from the ACIR

Peter B McIntyre[1], Timothy C Heath[1], Edward D O'Brien[2], Brynley P Hull[3]

The methodology for calculating immunisation coverage from information in the Australian Childhood Immunisation Register (ACIR) has recently been described in *Communicable Diseases Intelligence,*[1] and the third quarterly report of national immunisation coverage appears in this issue (page 122). The purpose of this report is to outline some of the limitations of these data and to emphasise the important messages from them.

Immunisation coverage estimates from the ACIR compared with the ABS

The Australian Bureau of Statistics (ABS) immunisation survey[2] measured immunisation coverage by a very different method to the ACIR.[1] The ABS survey was conducted by face-to-face interview of a random sample of Australian households, representative of the resident population. Although immunisation status was measured by parental report, which tends to overestimate immunisation,[3] parents of 61% of children referred to immunisation records.[2] By contrast, the ACIR measures coverage from information submitted by providers; the greatest problem with this method is failure to report.

Table 1 compares the third quarterly coverage estimates from the ACIR for completion of the immunisation schedule at 12 months of age, with the estimates from the 1995 ABS survey for children of the same age. In most States, the ABS estimate of Diptheria-Tetanus-Pertussis (DTP) and Oral Polio (OPV) coverage was substantially higher than the ACIR's, and it is likely that the true coverage lies somewhere between these figures. A notable exception was Queensland, where DTP coverage on the ACIR was 2% higher than estimated by the ABS. As Queensland's reporting system (VIVAS) is linked to vaccine supply, encounters are more likely to be reported to the ACIR from providers using VIVAS than from providers using the standard encounter form.[4] It is likely that the ACIR coverage estimates for Queensland are closer to true coverage in that State than those for other jurisdictions.

Estimates of Hib coverage from the ACIR are 10% higher than the ABS estimates. This reflects the marked improvement in immunisation coverage between the ABS survey in 1995 (which took place less than two years after Hib was introduced into the schedule) and the period covered by the third quarterly report. It also confirms that the ACIR is able to detect large changes in immunisation coverage, such as occur with the introduction of new vaccines into the childhood schedule.

The Northern Territory is the only jurisdiction where Hib coverage, as measured by the ACIR, substantially exceeds that of DTP. There are a number of possible factors contributing to this. Firstly, the Northern Territory uses the PRP-OMP conjugate Hib vaccine, which requires only 2 doses for the primary course for all children. This may lead to both a truly higher completion rate and a spurious increase, as it is known that third dose vaccinations are less frequently reported to the ACIR.[4] Secondly, Hib immunisation has been more actively promoted in the Northern Territory than in other jurisdictions.

Evidence for under-reporting to the ACIR

In 1996, a consultancy group (Human Capital Alliance) conducted an evaluation of the ACIR.[4] This included cross-checking of parent-held immunisation records amongst a sample of children recorded by the ACIR as being at least 30 days overdue in September 1996.[3] This comparison showed that 27% of third dose DTP encounters were discrepant due to missing data on the ACIR,[4] confirming underestimation of coverage, but not quantifying it.

The degree by which coverage is underestimated is likely to vary by State or Territory and by the pattern of immunisation provision. Published data are available from two jurisdictions with a high proportion of providers in the public sector - the Northern Territory and Victoria. In the Northern Territory, a recent audit of immunisation coverage registers found that second dose Hib and third dose DTP coverage were 94% and 89% respectively,[5] compared with ACIR estimates of 59% and 67%. Reasons for underestimation in the Northern Territory include:

- limited use of Medicare numbers as the unique identifier in Northern Territory data, which makes matching of vaccination encounters to Medicare data problematic;
- delays in data transmission that have resulted in the exclusion of a significant proportion of Northern Territory data from the quarterly ACIR reports; and until recently,

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- reluctance by some health services to participate in the ACIR.

In Victoria, a pilot study of home immunisation, conducted in November 1996 in economically disadvantaged local government areas in Melbourne, estimated that 93% of children were up to date with immunisation at 9 or 16 months of age, compared with 84% recorded by the ACIR.[6] The degree of under reporting may be greater in States with a higher proportion of general practitioner immunisation, such as New South Wales and Western Australia, and lower in areas with centralised reporting to the ACIR, such as Queensland and the Australian Capital Territory. An independent evaluation of children recorded as being overdue by the ACIR in New South Wales, based on Public Health Units, was completed in 1997. This study should provide insights into ACIR reporting in a large area with predominantly general practitioner based immunisation delivery. A similar evaluation is also planned for Western Australia in 1998.

Future developments and conclusions

Current ACIR estimates of immunisation coverage in Australia for the vaccines scheduled in the first 12 months of life are minimum estimates or a worst case scenario. The ACIR underestimates immunisation coverage because of under reporting of vaccination encounters. Delays in reporting encounters, data transfer, and data entry are less influential causes of underestimation, because the method used to calculate national immunisation coverage allows at least 6 months after the recommended age of vaccination for reports to be entered into the ACIR.[1] Between the first and third quarterly cohorts, there has been a small but definite increase nationally from 75 - 77% in the proportion of children fully immunised with a primary course of DTP, Hib, and OPV vaccines. While there may have been a real improvement in immunisation coverage, it is likely that this largely represents improved reporting to the ACIR.

The introduction of additional financial incentives for general practitioner immunisation can be expected to further improve the accuracy of the ACIR estimates of coverage, and its usefulness for monitoring Australia's progress towards national immunisation targets. Despite its limitations, ACIR data are providing valuable insights into the patterns of immunisation in Australia and with improving participation, ACIR's value as a planning tool will be further enhanced.

Table 1. Percentage of children fully immunised, by State and Territory and assessment method, assessed at 1 year of age

	Vaccine					
	DTP		OPV		Hib	
State	ACIR[1] (%)	ABS[2] (%)	ACIR (%)	ABS (%)	ACIR (%)	ABS (%)
Australian Capital Territory	83	87	82	87	81	69
New South Wales	78	87	77	88	77	63
Northern Territory	59	85	59	70	67	70
Queensland	81	79	82	83	82	52
South Australia	81	86	81	85	81	57
Tasmania	81	87	82	91	81	63
Victoria	82	90	82	88	82	66
Western Australia	72	87	72	84	72	71
Australia	79	86	79	86	79	62

1. Australian Childhood Immunisation Register: assessment date 30/9/97 for cohort of children born between 1/7/96-30/9/96.
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The NCIRS was established by the National Centre for Disease Control, Commonwealth Department of Health and Family Services. The Centre analyses, interprets, and evaluates national surveillance data on immunisation coverage and vaccine preventable diseases. NCIRS also identifies research priorities, and initiates and coordinates research on immunisation issues and the epidemiology of vaccine preventable diseases in Australia.

Letter to the Editor

Hepatitis A - the neglected sexually transmissible disease

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To the Editor: Dore and Kaldor provide valuable guidance on the directions a coordinated national system for surveillance of sexually transmissible diseases (STD) might take.[1] They did not attempt to provide an outline of what diseases might fall into this group, or what data should be collected for each disease, although a recent review of a decade of STD surveillance in Victoria examined data on syphilis, gonorrhoea, chlamydia, genital herpes, genital warts, hepatitis B, chancroid, lymphogranuloma venereum and donovanosis. [2]

However, it would seem timely to draw attention again to hepatitis A as a sexually transmissible disease, at least in some urban populations. The epicentre of a prolonged epidemic of hepatitis A in 1991-92 among homosexual men, was the gay community of the inner Sydney suburbs. A crude incidence of 136 cases per 100,000 population was recorded for 1991 in eastern Sydney, compared to 13 per 100,000 for Australia as a whole.[3] The same epidemic was observed in Melbourne[4] and simultaneously in other cities around the world.[5] A similar, though smaller, epidemic occurred in 1995-96 in Sydney.

At the onset of the 1991-92 epidemic, the then Eastern Sydney Public Health Unit established a standalone hepatitis A database where risk factor information, in particular sexual preference, was kept in de-identified format. Maintenance of the database through subsequent epidemics in other risk groups [6,7] has been invaluable in helping direct prevention activities, including education regarding hygiene and appropriate use of normal human immunoglobulin and hepatitis A vaccine. Analysis of the data revealed that during the 1991-92 and 1995-96 epidemics, adult males comprised almost 90% of all cases. This lead to a peak incidence among young males, approaching 500 cases per 100,000 (or 0.5%) per year. Information on sexual preference was recorded in 75% of adult males, and of these, men who have sex with men contributed 80-90% of cases. In Australian cities, hepatitis A appears to behave as a sexually transmissible disease, and its proper surveillance requires collection of comprehensive risk factor data including sexual preference.

References

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2. Crofts N, Gertig DM, Stevenson E, et al. Surveillance for sexually transmissible diseases in Victoria, 1983 to 1992. *Aust J Public Health* 1994;18:433-9.
3. Stokes M-L, Ferson MJ, Young LC. Outbreak of hepatitis A among homosexual men in Sydney. *Am J Public Health* 1997; 87:-39-41.
4. Stewart T, Crofts N. An outbreak of hepatitis A among homosexual men in Melbourne. *Med J Aust* 1993;158:519-21.
5. CDC. Hepatitis A among homosexual men - United States, Canada and Australia. *MMWR* 1992; 41:155,161-4.
6. Ferson MJ, Young LC, Robertson PW, Whybin LR. Hepatitis A outbreak in a preschool in Eastern Sydney. *Comm Dis Intell* 1994;18:82-83.
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Notice to readers

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Communicable Diseases Surveillance

Highlights

Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. The Australian Sentinel Practice Research Network (ASPREN) is a general practitioner-based sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases' whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Vaccine preventable diseases

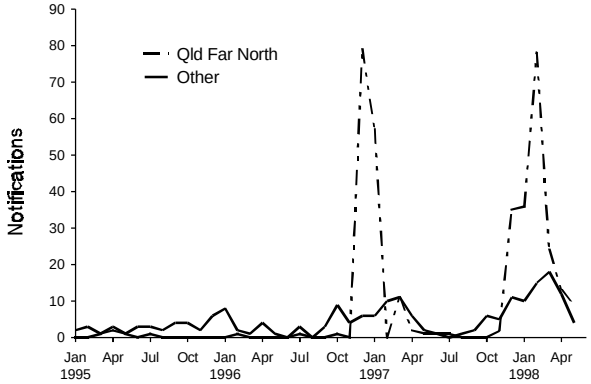
The number of pertussis notifications continued to fall with the number of cases having date of onset in April 1998 being lower than in any month since July 1996. In each of the previous four years (1994-1997), the number of notifications had been at a minimum in April, with an increase in the later months of the year. It will be interesting to observe the notifications in the next couple of months to see whether the downward trend continues, perhaps indicating an end to the current epidemic, or whether the number increases as in previous years.

Haemophilus influenzae type b (Hib) notifications remain low with 14 notifications so far in 1998 compared to 20 in the same period in 1997. Similarly, measles notifications continue to be as low as they were in 1997 with 190 notifications in 1998, compared to 183 in the same period in 1997.

Arboviruses

A further 44 notifications of dengue have been recorded for the current reporting period. The total number of cases with onset in 1998 was 219, of which 180 (82%) were from Queensland. This reflects the outbreak of dengue 3 in Cairns reported on page109 (Figure 1). The figure also shows the 1996-97 Torres Strait outbreak of dengue type 2 reported previously (*Comm Dis Intell* 1997;21:33). A small number of cases confirmed elsewhere in Australia appear to be related to the Cairns outbreak, but in most cases the disease was acquired overseas.

Figure 1. Notifications of dengue, 1995 to 1998, by month of onset, and area of residence



The number of new notifications for Barmah Forest virus infection and Ross River virus infection has also declined markedly over the last month (Figures 2 and 3).

Figure 2. Notifications of Barmah Forest virus infection, 1995 to 1998, by month of onset

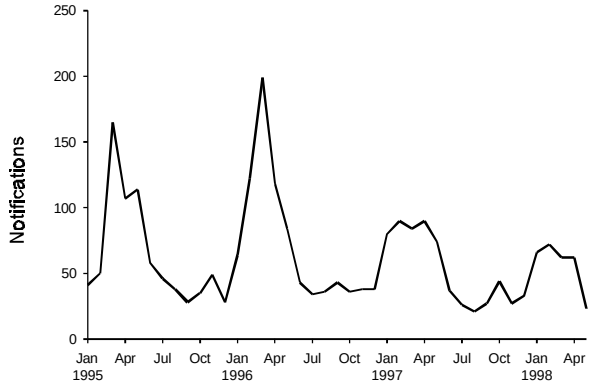
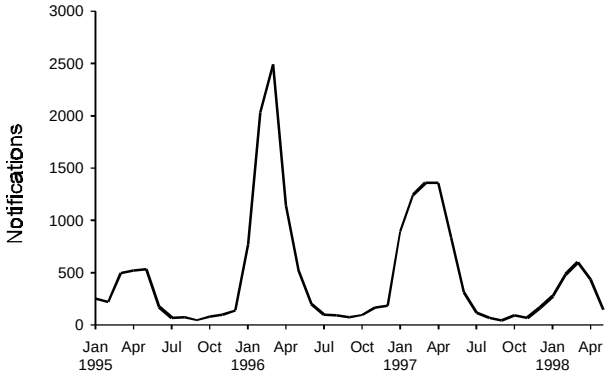


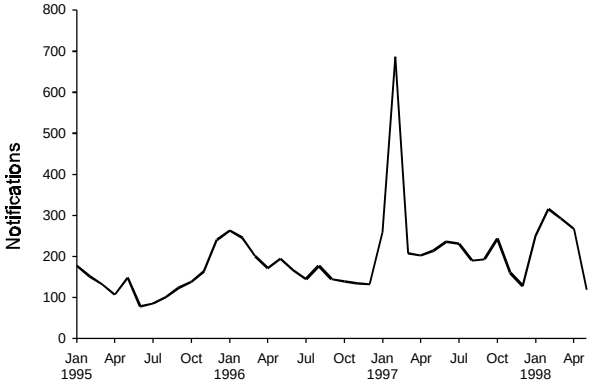
Figure 3. Notifications of Ross River virus infection, 1995 to 1998, by month of onset



Hepatitis A

The number of notifications for hepatitis A has declined since February (Figure 4). Of the 296 cases reported for the current period, 152 (51%) were in the age range 15-34 years; 192 of the 296 notifications (65%) were in males, the male:female (1.8:1 overall) being reflected in all age groups.

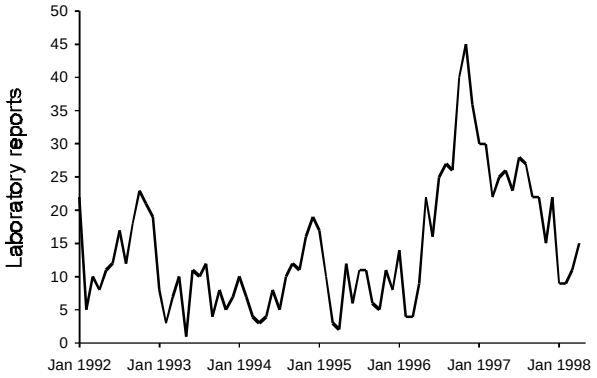
Figure 4. Notifications of hepatitis A, 1995 to 1998, by month of onset



Parvovirus

The number of laboratory reports of parvovirus has risen in recent weeks after falling markedly late last year (Figure 5). A total of 47 reports has been received for the year to date, most of which (43%) were for females in the 25-44 years age group.

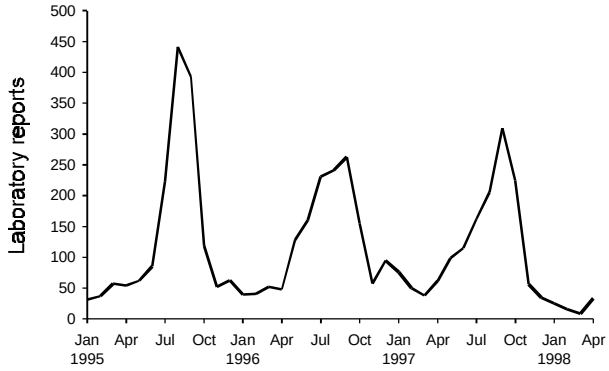
Figure 5. Laboratory reports of parvovirus, 1992 to 1998, by month of specimen collection



Rotavirus

Laboratory reports of rotavirus remain low for the time of year (Figure 6). We can expect a rise in the number of reports in the coming winter months.

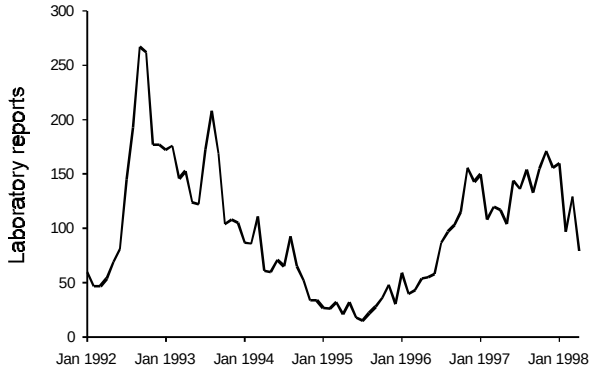
Figure 6. Laboratory reports of rotavirus, 1995 to 1998, by month of specimen collection



Mycoplasma pneumoniae

The LabVISE scheme has recorded a decline in the number of reports of *Mycoplasma pneumoniae* in recent weeks (Figure 7). This follows a sustained rise in reporting throughout 1997.

Figure 7. Laboratory reports of *Mycoplasma pneumoniae*, 1992 to 1998, by month of specimen collection



Tables

There were 6,054 notifications to the National Notifiable Diseases Surveillance System (NNDSS) for this four week period, 29 April to 26 May 1998 (Tables 1, 2 and 3). The numbers of reports for selected diseases have been compared with historical data for corresponding periods in the previous three years (Figure 8).

There were 1,294 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) this four week period, 23 April to 20 May 1998 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 17 to 20 ending 24 May 1998 are included in this issue of *CDI* (Table 6).

Table 1. Notifications of other diseases received by State and Territory health authorities in the period 29 April to 26 May 1998

Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
Arbovirus infection (NEC)[3]	0	1	4	7	0	0	2	0	14	10	52	88
Barmah Forest virus infection	0	2	0	70	0	0	0	2	74	94	309	420
Campylobacteriosis[4]	26	-	16	277	121	16	1	100	557	880	3,279	4,697
Chlamydial infection (NEC)[5]	34	NN	41	429	0	18	7	179	708	656	3,674	3,336
Dengue	0	1	1	38	0	2	0	2	44	11	270	188
Donovanosis	0	NN	2	0	NN	0	0	0	2	3	16	13
Gonococcal infection[6]	4	71	88	125	0	2	29	95	414	394	2,074	1,640
Hepatitis A	7	75	7	177	7	0	11	12	296	190	1,295	1,524
Hepatitis B incident	0	0	3	5	0	0	0	0	8	23	64	100
Hepatitis C incident[7]	1	1	0	-	0	0	-	-	2	2	20	6
Hepatitis C unspecified	26	NN	15	308	NN	30	0	82	461	1,109	2,254	3,889
Hepatitis (NEC)	0	0	0	0	0	0	0	NN	0	1	7	11
Legionellosis	0	1	0	14	5	0	3	0	23	16	103	72
Leptospirosis	0	2	0	23	1	0	0	2	28	8	73	50
Listeriosis	0	0	0	1	0	0	0	2	3	4	25	42
Malaria	4	6	0	0	0	0	5	5	20	118	211	352
Meningococcal infection	1	9	0	5	0	0	2	3	20	25	93	129
Ornithosis	0	NN	0	0	0	0	4	0	4	10	13	32
Q Fever	0	7	0	28	5	0	4	0	44	70	206	242
Ross River virus infection	1	13	4	451	3	3	1	14	490	1,217	2,056	5,430
Salmonellosis (NEC)	4	57	29	330	60	10	38	48	576	494	3,585	3,841
Shigellosis[4]	0	-	5	32	3	0	5	14	59	58	300	393
Syphilis[8]	0	28	24	48	0	0	0	2	102	92	495	510
Tuberculosis	3	15	0	19	1	0	8	2	48	86	333	428
Typhoid[9]	0	1	0	0	0	0	0	2	3	5	39	41
Yersiniosis (NEC)[4]	0	-	0	17	4	1	2	0	24	18	125	132

1. For HIV and AIDS, see Tables 7 and 8. For rarely notified diseases, see Table 3.
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. NT: includes Barmah Forest virus.
4. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
5. WA: genital only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. Qld, Vic and WA incident cases of Hepatitis C are not separately reported.
8. Includes congenital syphilis
9. NSW, Qld, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified

\- Elsewhere Classified.

Table 2. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period

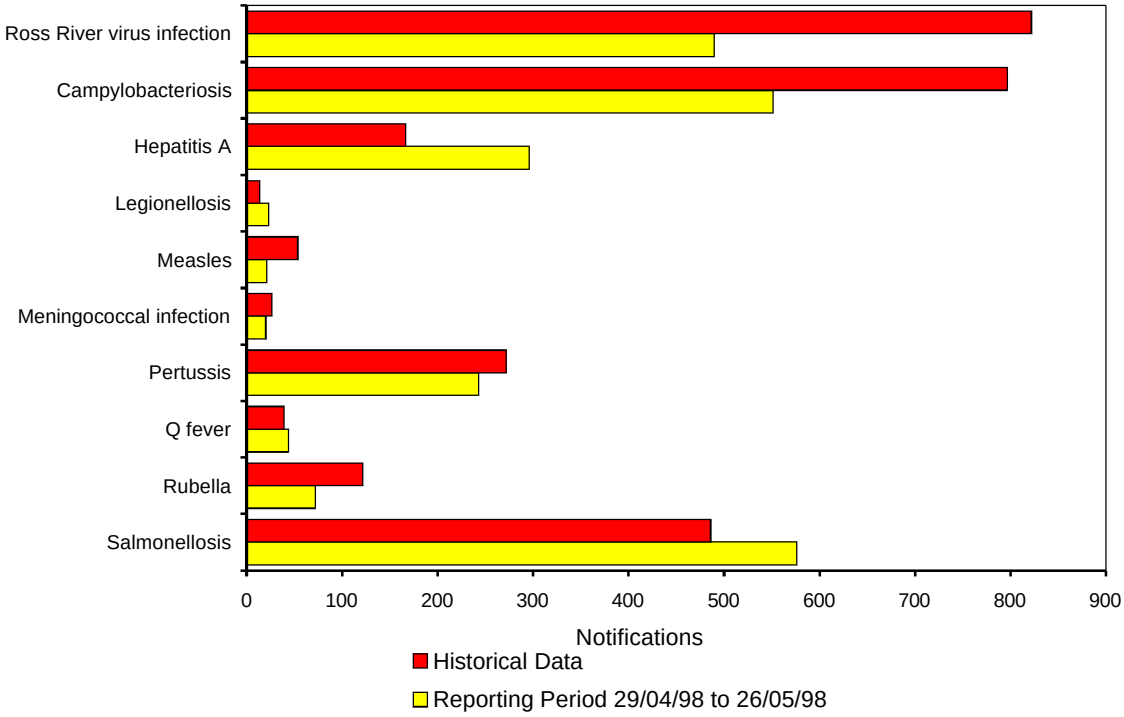
Disease[1,2]	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1998	This period 1997	Year to date 1998	Year to date 1997
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0
H. influenzae type b infection	0	2	0	2	0	0	2	0	6	5	14	20
Measles	6	4	0	4	0	4	6	3	27	38	190	183
Mumps	1	1	0	2	0	0	2	0	6	22	68	85
Pertussis	6	63	0	111	41	3	36	19	279	389	2,728	3,078
Rubella[3]	2	3	0	47	1	2	9	8	72	93	292	633
Tetanus	0	0	0	0	0	0	0	0	0	1	0	4

NN. Not Notifiable

1. No notifications of poliomyelitis have been reported since 1986.
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. Includes congenital rubella

Please note some data for NNDSS not available, see note next page.

Figure 8. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in the corresponding 4 week periods of the last 3 years and the 2 week periods immediately preceding and following those.

Table 3. Notifications of rare[1] diseases received by State and Territory health authorities in the period 29 April to 26 May 1998

Disease[2]	Total this period	Reporting States or Territories	Total notifications 1998
Brucellosis	4	ACT, Qld	19
Cholera			2
Hydatid Infection	4	Qld, Vic, WA	15
Leprosy	1	NSW	2

1. Fewer than 60 cases of each of these diseases were notified each year during the period 1988 to 1998.
2. No notifications have been received during 1998 for the following rare diseases: botulism, lymphogranuloma venereum, plague, rabies, yellow fever, or other viral haemorrhagic fevers.

Please note:

For the National Notifiable Diseases Surveillance System (Tables 1, 2 and 3, and Figure 8):

- *sexually transmissible diseases notifications for 1997 and 1998 from South Australia are not available; and*
- *notifications for the period 13 to 26 May 1998 from Victoria are not available*.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 23 April to 20 May 1998, and total reports for the year

	State or Territory[1]								Total this period	Total reported in *CDI* in 1998
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Measles, mumps, rubella										
Measles virus					1			1	2	35
Mumps virus								3	3	13
Rubella virus				5				2	7	61
Hepatitis viruses										
Hepatitis A virus	1	2	2	17	9			6	37	210
Hepatitis D virus					1				1	3
Hepatitis E virus								1	1	2
Arboviruses										
Ross River virus			3	24	2			9	38	508
Barmah Forest virus			3					3	6	21
Dengue not typed			1					5	6	19
Kunjin virus								1	1	3
Flavivirus (unspecified)				3					3	37
Adenoviruses										
Adenovirus type 1					3		1		4	9
Adenovirus type 2					3				3	11
Adenovirus type 3					4		1		5	17
Adenovirus type 7					4				4	11
Adenovirus type 37							1		1	1
Adenovirus not typed/pending		16			54			4	74	308
Herpes viruses										
Cytomegalovirus		2		8	7	2	3	8	30	355
Varicella-zoster virus		10		17	30	2	18	30	107	573
Epstein-Barr virus		5	1	10	78	2	9	26	131	772
Other DNA viruses										
Parvovirus					4			4	8	67
Picorna virus family										
Coxsackievirus B6					1				1	1
Echovirus type 11		6							6	16
Echovirus type 18		5							5	5
Rhinovirus (all types)	1	8			9		2	11	31	188
Enterovirus not typed/pending		9		5			1	27	42	197
Ortho/paramyxoviruses										
Influenza A virus		29		1	45			9	84	230
Influenza B virus					18			3	21	75
Parainfluenza virus type 1	1	5			16		1	3	26	150
Parainfluenza virus type 2		1			5				6	20
Parainfluenza virus type 3		2			1			12	15	189
Parainfluenza virus typing pending						1			1	2
Respiratory syncytial virus		15		7	22	1	2	5	52	316
Other RNA viruses										
Rotavirus		3		1	9	2		10	25	150

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 23 April to 20 May 1998, and total reports for the year, continued

	State or Territory[1]								Total this period	Total reported in *CDI* in 1998
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Other										
Chlamydia trachomatis not typed	20	35	52	37	74	10		129	357	1,756
Chlamydia species		6							6	23
Mycoplasma pneumoniae		7		16	29		3	2	57	639
Coxiella burnetii (Q fever)		2		7	2			2	13	52
Rickettsia spp - other								4	4	6
Salmonella species								1	1	6
Bordetella pertussis	1			9			17	32	59	648
Legionella pneumophila					1				1	4
Legionella longbeachae					3				3	19
Cryptococcus species	2								2	11
Leptospira hardjo								1	1	2
TOTAL	26	168	62	167	435	20	60	356	1,294	7,758

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.

Table 5. Virology and serology laboratory reports by contributing laboratories for the reporting period 23 April to 20 May 1998

State or Territory	Laboratory	Reports
Australian Capital Territory	Woden Valley Hospital, Canberra	30
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	102
	New Children's Hospital, Westmead	18
	Royal Prince Alfred Hospital, Camperdown	38
Queensland	Queensland Medical Laboratory, West End	175
South Australia	Institute of Medical and Veterinary Science, Adelaide	435
Tasmania	Northern Tasmanian Pathology Service, Launceston	4
	Royal Hobart Hospital, Hobart	17
Victoria	Royal Children's Hospital, Melbourne	30
	Victorian Infectious Diseases Reference Laboratory, Fairfield	28
Western Australia	PathCentre Virology, Perth	321
	Western Diagnostic Pathology	96
TOTAL		1,294

Table 6. Australian Sentinel Practice Research Network reports, weeks 17 to 20, 1998

Week number	17		18		19		20	
Week ending on	3 May 1998		10 May 1998		17 May 1998		24 May 1998	
Doctors reporting	45		50		46		51	
Total encounters	6,061		6,535		6,650		6,861	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	30	4.9	19	2.9	55	8.3	43	6.3
Rubella	1	0.2	1	0.2	1	0.2	2	0.3
Measles	0	0.0	0	0.0	1	0.2	0	0.0
Chickenpox	7	1.2	11	1.7	12	1.8	9	1.3
Pertussis	0	0.0	0	0.0	3	0.5	0	0.0
HIV testing (patient initiated)	5	0.8	16	2.4	13	2.0	10	1.5
HIV testing (doctor initiated)	2	0.3	4	0.6	4	0.6	0	0.0
Td (ADT) vaccine	43	7.1	41	6.3	36	5.4	33	4.8
Pertussis vaccination	28	4.6	40	6.1	36	5.4	30	4.4
Reaction to pertussis vaccine	4	0.7	3	0.5	1	0.2	0	0.0
Ross River virus infection	0	0.0	0	0.0	2	0.3	1	0.1
Gastroenteritis	60	9.9	86	13.2	73	11.0	91	13.3

NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of more than 40 communicable diseases or disease groups endorsed by the National Health and Medical Research Council (NHMRC). Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see CDI 1998;22:4-5.

LabVISE is a sentinel reporting scheme. Twenty-one laboratories contribute data on the laboratory identification of viruses and other organisms. Data are collated and published in Communicable Diseases Intelligence every four weeks. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see CDI 1998;22:8.

ASPREN currently comprises about 100 general practitioners from throughout the country. Up to 9,000 consultations are reported each week, with special attention to 12 conditions chosen for sentinel surveillance. CDI reports the consultation rates for all of these. For further information, including case definitions, see CDI 1998;22:5-6.

Additional Reports

National Influenza Surveillance, 1998

Three types of data are included in National Influenza Surveillance, 1998. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network, Department of Human Services (Victoria), Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health (Northern Territory); laboratory surveillance data from the Communicable Diseases Intelligence Virology and Serology Laboratory Reporting Scheme, LabVISE, and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. For further information about these schemes, see CDI 1998; 22:83.

Sentinel General Practitioner Surveillance

Consultation rates for influenza like illness recorded by ASPREN remained below 9 per 1,000 consultations (Figure 9). The rates for the Northern Territory Tropical Influenza surveillance have shown a modest decline since the beginning of the year to levels below 3 per 1,000 consultations in the last month. These are comparable to those reported by the Victorian scheme. The New South Wales scheme reported the highest levels of influenza activity for the month of May, with consultation rates between 8 and 12 per 1,000 encounters.

Laboratory Surveillance

For the year to date there have been 208 laboratory reports of influenza. Of these, 160 (77%) were influenza A and 48 (23%) influenza B (Figure 10). More influenza A has been reported for the 25 to 44 year old age group than

in the previous month, and influenza B reports continued to be low in children less than 5 years of age.

Figure 9. Sentinel general practitioner influenza consultation rates, 1998, by scheme and week

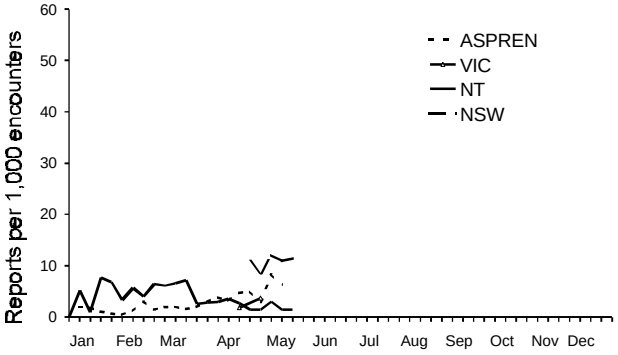
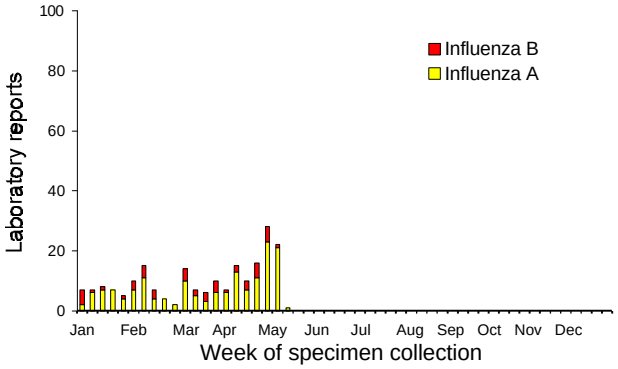


Figure 10. Laboratory reports of influenza, 1998, by type and week of specimen collection



The WHO Collaborating Centre for Influenza Reference and Research has received 36 isolates of influenza A and 6 of influenza B for the year to date. All the influenza A viruses were H3N2 strains related to A/Sydney /5/97. Analysis of type B isolates is pending.

Absenteeism surveillance

Rates of absenteeism for Australia Post employees for three consecutive days of each week have been reported for the four weeks preceding May 27. These rates have remained stable at a level of 0.25% to 0.27% nationally.

HIV and AIDS Surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (ACT, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648 Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for December 1997, as reported to 31 March 1998, are included in this issue of CDI (Tables 7 and 8).

Childhood immunisation coverage

Table 9 provides the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 July and 30 September 1996 according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in CDI 1998;22:36-37.

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 December 1997, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 1997	This period 1996	Year to date 1997	Year to date 1996
HIV diagnoses	Female	0	0	0	0	1	0	1	1	3	4	75	69
	Male	0	26	0	10	2	0	15	2	55	64	702	852
	Sex not reported	0	3	0	0	0	0	0	0	3	0	17	5
	Total[1]	0	29	0	10	3	0	16	3	61	68	795	927
AIDS diagnoses	Female	0	0	0	1	0	0	1	0	2	3	25	32
	Male	0	10	0	3	0	0	4	0	17	37	286	609
	Total[1]	0	10	0	4	0	0	5	0	19	40	311	641
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	13	17
	Male	0	4	0	1	0	0	2	0	7	26	204	480
	Total[1]	0	4	0	1	0	0	2	0	7	27	218	497

1. Persons whose sex was reported as transgender are included in the totals.

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 December 1997, by sex and State or Territory

		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	20	540	7	119	51	4	191	82	1,014
	Male	178	10,163	93	1,766	620	75	3,674	843	17,412
	Sex not reported	0	260	0	0	0	0	29	1	290
	Total[1]	198	10,983	100	1,891	671	79	3,904	929	18,755
AIDS diagnoses	Female	7	157	0	43	19	2	63	23	314
	Male	80	4,325	30	752	318	41	1,515	334	7,395
	Total[1]	87	4,493	30	797	337	43	1,585	359	7,731
AIDS deaths	Female	2	112	0	28	14	2	43	15	216
	Male	52	3,032	23	523	214	26	1,198	241	5,309
	Total[1]	54	3,151	23	553	228	28	1,247	257	5,541

1. Persons whose sex was reported as transgender are included in the totals.

Table 9. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 July 1996 to 30 September 1996; assessment date 30 September 1997.

	State or Territory								
	ACT	NSW	NT[1]	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,123	22,756	886	12,461	4,844	1,737	15,869	6,519	66,195
Vaccine									
DTP (%)	82.7	77.6	59.1	81.5	80.9	80.7	81.6	72.0	78.9
OPV (%)	82.3	77.2	59.4	81.9	80.8	81.6	81.7	72.3	78.9
Hib (%)	81.3	76.8	66.6	82.5	80.8	80.9	81.7	72.4	79.0
Fully Immunised (%)	80.6	74.7	55.0	79.4	78.9	79.2	79.9	70.5	76.7
Change in fully immunised since last quarter (%)	+3.2	+1.5	-6.7	-1.1	+2.0	+3.2	-0.1	+3.6	+0.9

1. Some data from the Northern Territory were not included on the ACIR at the time of these calculations. Northern Territory calculations, using a local database, indicate that the proportions of children immunised at 12 months of age are as follows : DTP - 80.0%, Polio 79.8%, Hib 86.0%, fully immunised - 77.0%.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Family Services. For further information on these figures or data on the ACIR please contact the Immunisation Section of the HIC: Telephone 02 6203 6185.

Dengue overseas

Source: World Health Organization and the Pacific Public Health Network

Many parts of south-east Asia (Malaysia, Taiwan [China], Cambodia, Viet Nam, Thailand, Philippines, Indonesia, Myanmar), the western Pacific (Guam, Cook Islands, Fiji, New Caledonia, Kiribati) and Latin America (Brazil, Venezuela, Columbia) have been experiencing unusually high levels of dengue/dengue haemorrhagic fever activity. Although there is often a seasonal increase in dengue in some of these places at this time of the year, the level of activity in 1998 is considerably higher than in previous years. Changes in weather patterns as a result of the El Nino phenomenon are thought to be a major contributing factor.

Unless more effective measures are taken to control the main vector, *Aedes aegypti*, in these and other countries/areas, dengue will continue to be a growing problem in tropical and subtropical regions of the world. Essential elements of an effective program are integrated mosquito control with community and intersectoral involvement, vector surveillance for monitoring and evaluation, emergency preparedness, capacity building and training, and applied research.

Viet Nam. A total of 16,647 cases of dengue/dengue haemorraghic fever with 55 deaths (case fatality rate = 0.3%) has been reported since the beginning of 1998. The incidence has more than doubled compared with the same period last year. As the traditional peak season for dengue (June to November) has only just started a major epidemic is expected to occur. While dengue 2 virus was the most prevalent strain in 1997, early data suggests that dengue 3 virus predominates this year.

Malaysia. Since the beginning of 1998 there has been a total of 5,337 cases (including 194 cases of dengue haemorraghic fever) and five deaths reported The number of cases is similar to that reported for the same period last year.

Indonesia. There has been a rapid increase recently in dengue/dengue haemorrhagic fever cases and all provinces of the country are now affected. As of 5 May a total of 32,665 cases with 774 deaths had been reported. This number of cases is considerably higher than for the same period last year. It is expected that cases will continue to increase during the peak season of May to July.

Brazil. This year Brazil is experiencing the highest levels of dengue transmission in its history. A total of 234,828 cases was reported during the first four months of 1998, compared with 159,965 cases during the same period in 1997. There have been 60 cases of dengue haemorrhagic fever reported and eight deaths. Both dengue 1 and dengue 2 viruses are circulating.

Tonga. Since February 1998 the Ministry of Health in Tonga has reported a total of 438 suspected cases of dengue. Of these 220 were serologically tested and 70 confirmed. Included was a six year old child with confirmed dengue who died. There is little evidence of dengue in outer islands. The number of cases has fallen in recent weeks. The virus has been identified as dengue virus type 2.

Overseas briefs

Source: World Health Organization (WHO)

Crimean-Congo haemorrhagic fever

Pakistan. Crimean-Congo haemorrhagic fever has been diagnosed in four patients, including two deaths, in a village in the Kohlu area of Baluchistan Province of Pakistan in February 1998. All cases belonged to the same family of herders living in close contact with their sheep. Specimens were collected from the two surviving patients, from other members of the family who developed fever without haemorrhagic symptoms, and from many of the contacts. Blood specimens from cases and contacts were shipped to the WHO Collaborating Centre for Reference and Research on Special Pathogens at the Centre for Applied Medical Research, Public Health Laboratory Service, Porton Down, United Kingdom for testing. All had IgG and IgM antibody to Crimean-Congo haemorrhagic fever virus indicating recent infection. Further virological testing is in progress at Porton Down.

Afghanistan. Crimean-Congo haemorrhagic fever has been diagnosed in a village in the district of Rustaq, Province of Takar in mid-March 1998. A total of 19 cases have been reported of which 12 were fatal. Rustaq district, in north-east Afghanistan, was severely affected by the earthquake in early February and access was further complicated by melting snow. However, representatives from United Nations and non-governmental organisations were on site to provide assistance and notified WHO of the outbreak. The WHO country office organised the investigation and management of the cases with the International Federation of Red Cross and Red Crescent Societies and Médecins Sans Frontières. Blood specimens were shipped to the WHO Collaborating Centre for Reference and Research on Special Pathogens at the Centre for Applied Medical Research, Public Health Laboratory Service, Porton Down, United Kingdom. Serological testing (IgG/IgM antibody) provided evidence of Crimean-Congo haemorrhagic fever virus infection. Further virological testing is in progress at Porton Down.

Cholera in Africa

Rwanda. A cholera outbreak which began in late February 1998 in Cyangugu Prefecture close to the border with the Democratic Republic of the Congo is continuing with an increased number of cases reported recently.

Democratic Republic of the Congo. The Democratic Republic of the Congo has reported a total of 9,605 cases with 746 deaths (case fatality rate 8%) since January 1998. Major outbreaks have occurred in the Bunia area (Orientale Province, ex Haut-Zaire province) which is 1,600 km east of Kinshasa, and in Bukavu (Sud-Kivu Province) which is close to the Rwandan border. Cholera has also been reported from other areas in the country. The WHO country office, as well as major nongovernement organisations, are providing support to the health authorities to control the outbreak.

Burundi. From 1 April to 18 May 1998, 77 cases of cholera and 3 deaths had been reported in Bujumbura Province, Burundi**.** The districts of Buyenzi, Bwiza, Cibitoke and Musaga were the most affected. Health education and activities to improve sanitation are being carried out by the Ministry of Health.

Uganda. The dramatic cholera outbreak which started in late 1997 is still affecting the country with over 20,000 cases and over 1,000 deaths reported since the beginning of 1998.

The current cholera outbreak affecting the Great Lakes region confirms earlier forecasts of a potential spread from eastern African countries which were affected by major outbreaks last year, to countries in the central and southern part of Africa.

Malaria in the United Republic of Tanzania.

Following reports of an outbreak in Sumbawanga District, in the south-eastern part of the country, the Regional Health Authority sent an investigating team to the affected areas; Matai, Sopa and Katete wards. It has been confirmed that the outbreak was caused by a severe form of falciparum malaria. The whole country is experiencing increased numbers of malaria cases and deaths following an abnormally long rainy season. Similar outbreaks have been reported in several other districts (Tanga, Muleba, Korogwe, Handeni and Lushoto) in the last 12 months. Malaria is endemic in the whole country and increased transmission often occurs at this time of year.

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