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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