The effect of climate change on Australian arboviruses

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Since it was first raised more than 20 years ago [1], there has been increasing concern about the potential impacts of climate change on mosquito-borne viral diseases in Australia. This has generated a number of predictions and projections on the effect of global warming on the incidence and spread of Australian arboviruses. These have been discussed recently by Russell [2] and Jacobs and colleagues [3] and thus need not be repeated in detail here. In addition, it was also the topic of a previous 'In Focus' article [4]. This paper will briefly focus on the complex interplay between climate-associated factors which may affect the incidence and spread of mosquito-borne viruses, and show how these factors may influence the ecologies of the major Australian arboviruses.

The influence of environmental factors on arbovirus ecology

The arthropod-vertebrate host transmission cycle is a highly complex, dynamic and finely balanced interaction between the virus, its vertebrate host(s), vector(s), and the environment [5, 6]. The relationships between environmental factors and arboviral ecologies are many and well established, often complex and inter-related, and affect adult and larval mosquitoes as well as the vertebrate hosts. Thus climate (long-term weather patterns) and weather (moisture, humidity, precipitation, temperature, sun-light, wind velocity, atmospheric pressure and other factors) impact on breeding, developmental rate, nutritional status, survival, host-seeking behaviour, diapause, and many other aspects of the biology of the vector [7-9]. Environmental factors also affect food supplies for both adult (plant nectar) and larval (organic matter or other mosquito larvae) mosquitoes, and play a role in wind-borne vector dispersal, and therefore in disease spread. The environment also impacts on the vertebrate host in many different ways, including the growth of herbage as a food supply, breeding, immune status, and influencing migration as examples [10]. In addition, it also impacts on human behaviour and demographics which affects the likelihood of human exposure to arboviruses [11].

The Australian arboviruses of importance to human health

Only a few of the known arboviruses cause human disease [12, 13]. These are shown in Table 1. The most important of these are the Flaviviruses Murray Valley encephalitis virus (MVEV), West Nile virus Kunjin clade (WNV-KUN), Japanese encephalitis virus (JEV), and the four subtypes of dengue virus (DENV) and, of the Alphaviruses, Ross River (RRV) and Barmah Forest viruses (BFV). The major vectors and vertebrate hosts, where known, are also shown in Table 1. However, it should be noted that a number of other mosquito vectors have been implicated in transmission cycles, either because of virus isolation or, in a few cases, from transmission studies carried under laboratory conditions. Similarly, various other vertebrate hosts may also be involved in the transmission cycles of most of the viruses listed in Table 1, with the exception of the four DENV which have highly specific transmission cycles in mainland Australia with humans as their vertebrate hosts and *Aedes aegypti* mosquitoes their major vector. Thus, climatic effects may vary depending on breeding preferences and host preferences of different mosquito species. Additional information about the possible vectors and vertebrate hosts can be found in a number of recent reviews [12-15].

The ecologies of the major arboviruses, and the role of climatic factors in their transmission cycles

MVEV

MVEV is endemic across most of northern Australia and Papua New Guinea (PNG), with the highest incidence of virus activity in the Kimberley region of Western Australia (WA) and the north of the Northern Territory [4]. It also occurs at frequent intervals in the Pilbara region of WA and in the north of Queensland, and occasionally spreads south into the Gascoyne of WA and central Australia near Alice Springs. Very rarely, the virus emerges to cause epidemics of disease in south-eastern Australia, the last two major epidemics being in 1951 and 1974. Prior to the early

Table 1. Arboviruses infecting humans in Australasia.

Virus	Serological group	Disease	Vector	Vertebrate hosts
Family Flaviviridae – genus Flavivirus				
Murray valley encephalitis virus (MVE)*	JE† sero-group	Encephalitis	*Culex annulirostris*	Waterbirds, esp. *Ardeid* sp.
West Nile virus (Kunjin clade) (WNV-KUN)	JE sero-group	Encephalitis, usually mild	*Cx. annulirostris*	Waterbirds, esp. *Ardeid* sp.
Alfuy virus (ALFV)	JE sero-group	Asymptomatic infections$^{\Delta}$	*Cx. annulirostris*	Various bird species
Japanese encephalitis virus (JEV)	JE sero-group	Encephalitis	*Cx. annulirostris*	Waterbirds esp. *Ardeid* sp., domestic pigs
Kokobera virus (KOKV)	Kokobera sero-group	Mild polyarticular disease	*Cx. annulirostris*/ *Aedes vigilax*	Macropods, horses
Stratford virus (STRV)	Kokobera sero-group	Asymptomatic infection	*Ae. vigilax*	Macropods??
Sepik virus (SEPV)	Yellow fever sero-group	Mild febrile disease	*Mansonia* sp.	Unknown
Edge Hill virus (EHV)	Yellow fever sero-group	Mild arthralgia, fatigue	*Cx. annulirostris*	Macropods??
Dengue virus type 1 (DENV-1)	Dengue sero-group	Dengue fever, dengue HF/SS#	*Ae. aegypti*	Humans
Dengue virus type 2 (DENV-2)	Dengue sero-group	Dengue fever, dengue HF/SS	*Ae. aegypti*	Humans
Dengue virus type 3 (DENV-3)	Dengue sero-group	Dengue fever, dengue HF/SS	*Ae. aegypti*	Humans
Dengue virus type 4 (DENV-4)	Dengue sero-group	Dengue fever, dengue HF/SS	*Ae. aegypti*	Humans
Family Togaviridae – genus Alphavirus √				
Ross River virus (RRV)		Polyarthritis with rash, fever	*Ae. vigilax* / *Ae. camptorhynchus*/ *Cx. annulirostris*	Macropods, horses, other small animals
Barmah Forest virus (BFV)		Polyarthritis with rash, fever	*Ae. vigilax* / *Ae. camptorhynchus*/ *Cx. annulirostris*	Small native mammals??
Sindbis virus (SINV)		Rare human disease; fever, rash	*Cx. annulirostris*	Various avian sp.
Family Bunyaviridae – genus unassigned				
Gan Gan virus (GGV)		Acute polyarthritis-like illness	*Ae. vigilax* / *Cx. annulirostris*	Unknown
Trubanaman virus (TRUV)		Asymptomatic infections	*Anopheles annulipes* / *Cx. annulirostris*	Unknown

* Acronyms are those assigned by the International Committee for the Taxonomy of Viruses (ICTV)
† JE, Japanese encephalitis
Δ Asymptomatic infections are believed to occur from neutralising antibody data
\# HF – haemorrhagic fever; SS – shock syndrome
√ Alphaviruses are no longer classified by antigenic groups

1970s, it is probable that epizootic cycles of MVEV were confined to small pockets and driven by local, wet season flooding, but human cases were rarely seen. The development of Lake Argyle, Lake Kununurra and the large irrigation area in the Kimberley region of WA in the early 1970s is believed to have had a profound affect on the ecology of the region, with year round water and wetlands making continuous transmission cycles of arboviruses possible around Kununurra [16]. This in turn is believed to have led to seeding of virus in desiccation-resistant *Aedes* sp. eggs, gradually extending south and east of the Kununurra area [16]. Epidemic activity of MVEV is clearly driven by rainfall and flooding, but heavy rainfall and flooding may not in themselves be sufficient to initiate an epidemic [17]. It had been suggested that climatic conditions, including rainfall and flooding in central Australia, could be responsible for the movement of MVEV from northern Australia to south-eastern Australia [18, 19].

WNV-KUN

WNV-KUN has a different pattern of activity to MVEV despite sharing vertebrate hosts and vectors. It is also enzootic across northern Australia, but tends to occur much further south and may sometimes emerge in south-eastern Australia in years in which there is no evidence of MVEV. Nevertheless, it is probable that WNV-KUN activity is also driven by heavy rainfall and flooding.

DENV1-4

As described above, humans are the vertebrate hosts for DENV1-4 in Australia, and *Ae. aegypti* the major vector, although there is growing concern about a second major vector species, *Ae. albopictus,* which is now found in the islands of the Torres Strait [2]. DENV1-4 are not endemic to northern Australia; epidemic activity is initiated by importation through the arrival of infected travellers. Cases of dengue fever have generally been restricted to urban areas in the north-east of Queensland, although the geographic range of the vector, *Ae. aegypti,* extends further south to Gladstone, and in the west to Mount Isa [20]. This geographic distribution of *Ae. aegypti* is not believed to be governed by climate in eastern Australia [2] and, as the vector is a container breeder around human habitation, they may not be as susceptible to climatic factors as other species [21, 22].

JEV

JEV is not currently in mainland Australia, but threatens to spread from the Torres Strait and PNG, where it is believed to be enzootic, into north Queensland [23]. It first appeared in the Torres Strait in 1995, where it was responsible for three cases of encephalitis, two of whom died [24]. In 1998, two further cases of encephalitis were reported, one from the Torres Strait and the other on mainland Australia [25]. This latter case, together with evidence in pigs and/or mosquitoes in two areas of Cape York, were the first time JEV had occurred naturally in mainland Australia. JEV has been evident in the Torres Strait every year from 1995 to 2006, with the single exception of 1999, and was observed in mainland Australia in 1998 and 2006. It was suggested that the spread of JEV into Cape York in 1998 was due to cyclonic winds carrying the virus from PNG [26]. In addition, it is interesting to note that there had been a severe drought at that time in PNG which may have significantly increased the amount of virus in the environment [23]. The major mosquito vectors for JEV in Australia and the Torres Strait are *Cx. annulirostris* and *Cx. gelidus,* and the vertebrate hosts are ardeid birds, and possibly feral pigs.

Ross River and Barmah Forest viruses

RRV and BFV are enzootic throughout mainland Australia [4, 13, 14], and although both have also been responsible for disease in Tasmania, it is probable that only RRV is endemic [13]. RRV is the most important cause of arboviral disease in Australia, with nearly 5000 cases of epidemic polyarthritis reported annually, most of them from Queensland [2-4, 13, 27, 28]. BFV causes a similar disease [27], although the incidence of infection is only a quarter of that due to RRV. The major vertebrate hosts for RRV are macropods [4, 28], but a variety of other species may also be important, including horses and small marsupials in urban and peri-urban situations [28, 29].

The ecology of BFV, however, is much less well understood. The major vectors for both viruses are the southern and northern saltmarsh mosquitoes, *Ae. camptorhynchus* and *Ae. vigilax* respectively, the fresh-water breeding *Cx. annulirostris,* and the floodwater *Aedes* species in inland areas, but the ecology of RRV is more complex than this, with virus being isolated from over 30 mosquito species, many of which are probably important in specific ecological systems and geographic areas [28]. Epidemic activity is driven by various climatic factors, especially tides and rainfall, but forecasting has proved difficult as the climatic events tend to be localised [5]. The importance of tidal inundation of saltmarsh was shown clearly in the 1988-89 RRV outbreak in Western Australia. The same area also demonstrated the following year that, even when conditions are right for mosquito breeding, weather conditions have to be conducive to marsupial breeding to recruit young non-immune vertebrate hosts into the transmission cycle [30]. There are many examples of the importance of rainfall in generating outbreaks of RRV, perhaps most clearly seen following heavy rainfall and flooding in arid areas, and are an important generator of outbreaks country-wide [3, 28, 30, 31].

Climate change and Australian arboviruses

Various models of the effect of climate change on rainfall, sea level heights, humidity, sea surface temperature and wind to Australia have been predicted by CSIRO [32, 33] and the Intergovernmental Panel on Climate Change (IPCC 2007) [34] under different scenarios of greenhouse gas emissions. These predictions encompass a

number of implicit concerns for arboviral disease ecologies. Thus, under a best estimate, sea level rises are predicted to rise up to 80cm by the end of the century, although there is some evidence that this is already happening at an even faster rate, and temperatures are predicted to rise by about 1°C by 2030 (relative to 1990).

Rainfall is predicted to decrease throughout most of the Continent in winter and spring, and in much of western and central Australia in summer and autumn, but may increase slightly in summer in northern and eastern Australia. Increased cyclonic events may occur in northern Australia and be more intense, resulting in heavy rainfall events and extensive flooding. Humidity levels will also change, with lower humidity in central and southern Western Australia and Victoria in summer and autumn but will remain much the same elsewhere, and it will decrease over most of Australia in winter and spring with the exception of the north-east of the continent.

These and earlier predictions of global warming have been used by various authors to estimate possible changes in the spread and incidence of arboviral diseases in Australia. Some of these predictions are more general and descriptive, whereas others propose models to estimate potential risk. Most of the models relate to DENV1-4 and RRV.

The predictions for the arboviral encephalitides MVEV and WNV-KUN are based in part on an expected increase and intensity of cyclonic rainfall events and flooding [35], although these factors may also have a negative effect through flushing away mosquito larvae. Thus, with these heavy, sporadic rainfall events in northern and central Australia following cyclonic activity, it was suggested that there might be an increased chance of MVEV moving southwards towards the populous areas of southeastern Australia. JEV also has the potential for being brought into Australia more frequently through cyclone-generated, wind-blown mosquitoes [26].

There have been a number of predictions for DENV1-4 spread in Australia. These have been discussed by Russell and colleagues [2, 22]. The importance of the historical distribution of dengue needs to be taken into account as a major factor in any model, as does the urban nature of the vector, and its current distribution which has been maintained for a number of years. This experience strongly indicates that much of Australia is receptive to incursions of *Ae aegypti,* as demonstrated recently by the infestations in Tennant Creek and Groote Eylandt, but if climate change is an important factor, one has to ask why the vector hasn't spread already following major annual monsoonal rainfall, cyclonic events, and flooding.

The effect of climate change on RRV and BFV is more complex and difficult to predict [3, 5]. The expectation of higher sea levels will undoubtedly have a significant effect on the generation of outbreaks in coastal areas as wetlands and salt-marshes become more frequently inundated by tides, but the risk will depend very much on local coastal topography. Rainfall will also be crucial in many areas, both inland and coastal. A number of different predictions and models have been developed based on various climatic variables, either individually or in concert, including tides, temperature, rainfall and humidity. These have recently been reviewed [3], but the major message from these various studies is that, although useful, the predictions are only valid in localised or regional foci [3, 5, 36]. Thus they may also be useful for public health warnings and risk reduction in local areas and regions, but they may have limited value in terms of national or state-wide forecasting.

Overall, the conclusions reached by Russell [2] best describe the probable effects of climate change on Australian arboviruses – that endemic arboviruses may possibly increase in some areas, but they are likely to decrease in other areas and that, with respect to exotic virus diseases such as dengue, we are unlikely to see a significant effect in the distribution of transmission.

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