Endemic equine viral disease down under 2: Arbovirus in Australia
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Introduction
From an endemic perspective, Australia experiences sporadic and unpredictable outbreaks of equine viral diseases, which may result in considerable animal morbidity and mortality and are caused by zoonotic pathogens. Of greatest importance are Hendra virus (genus *Henipavirus*) and arboviral infections, including flaviviruses in the family *Flaviviridae* (Kunjin virus, Murray Valley encephalitis virus [MVEV] and Ross River virus [RRV]), a member of the genus *Alphavirus*, family *Togaviridae*.

In a previous article Hendra virus was reviewed with respect to its clinical features, timeline, geographical distribution, reservoir hosts and control of infection through vaccination. In this, the second of two articles, a similar overview is provided of endemic arboviruses affecting horses in Australia.

Arboviruses: Clinical syndromes
Both Kunjin virus and MVEV result in clinical signs of myeloencephalitis. Signs of intra-cranial and spinal cord involvement are variable between affected horses and may include changes in mentation and behaviour; ataxia, paresis, dysmetria, muscle fasciculations, central blindness, hyper-responsiveness, cranial nerve deficits (e.g. facial and/or hypoglossal nerve dysfunction), recumbency and seizures. In contrast to several outbreaks of West Nile virus infection in North America, pyrexia is uncommon or transient and not well detected.

In addition, mortality rates in the Australian flavivirus outbreak (Table 1: approximately 10%) were lower than in North American outbreaks of WNV, where rates of 20-40% were reported.1-3

Table 1: Summary statistics of 880 arboviral infections in horses during the 2011 epidemic

<table>
<thead>
<tr>
<th>State</th>
<th>NSW</th>
<th>Vic</th>
<th>SA</th>
<th>Qld</th>
<th>WA</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>880</td>
</tr>
<tr>
<td>Neurological case proportion (%)</td>
<td>100</td>
<td>73</td>
<td>65</td>
<td>81</td>
<td>100</td>
<td>82</td>
</tr>
<tr>
<td>Fatalities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>81</td>
</tr>
<tr>
<td>Overall fatality rate (%)</td>
<td>11</td>
<td>8.7</td>
<td>5.1</td>
<td>12.7</td>
<td>20</td>
<td>9.2</td>
</tr>
<tr>
<td>Neurological case fatality rate (%)</td>
<td>11</td>
<td>10.2</td>
<td>7.9</td>
<td>15.8</td>
<td>20</td>
<td>11.3</td>
</tr>
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</table>
Clinical progression of horses infected with Kunjin virus or MVEV is variable, however many horses recover over a period of one to two weeks. However, severity of neurological deficits can fluctuate and recovery can be prolonged in some horses. Information on the rate of residual deficits is not known for Kunjin virus/MVEV: in WNV outbreaks in North America, between 10-20% of horses may have chronic gait abnormalities, behavioural changes and/or cranial nerve deficits.\textsuperscript{2,4}

While RRV is a well-recognised cause of chronic disease, including fatigue and polyarthritis in humans,\textsuperscript{5} the role of the virus in development of disease in horses is less clear. Seroconversion to RRV is not uncommon in horses and clear association with disease is limited, despite anecdotal suspicion that the virus may result in reluctance to move, ataxia and polyarthropathy.

Recently, acute serological responses to RRV have been associated with petechiation, lymphadenopathy, distal limb swelling and reluctance to move in a small number of horses, supportive of an aetiological role of the virus in this syndrome.\textsuperscript{6} It is likely that the majority of RRV infections in horses are subclinical or not associated with development of disease, possibly due to variation in virulence within RRV genotypes. In the 2011 Australian arbovirus outbreak seroconversion to RRV in horses was associated with reluctance to move, stiff gait, ataxia and peripheral oedema.

**Arboviruses: Epidemiology and reservoir hosts**

Kunjin virus is a strain of West Nile virus (WNV\textsubscript{KUN}) endemic to Australia and is maintained in bird (reservoir/amplification host)-mosquito (vector) transmission cycles in north-western Australia. Sporadic outbreaks of WNVKUN (or MVEV) infection in humans or seroconversion in sentinel chickens occur in south-east Australia; however, until recently, these viruses were rarely associated with equine disease.

In the summer and autumn of 2011, a major outbreak of arbovirus-associated neurological disease occurred in five states of Australia. A total of 982 cases of arboviral disease were reported, involving WNV\textsubscript{KUN}, MVEV and RRV, and many affected horses demonstrated signs of encephalomyelitis. Prior to the outbreak, much of south-eastern Australia had higher than average rainfall and flooding: conditions conducive to the development of mosquito and wild bird (e.g. wading birds) populations, rapid viral amplification and spread and spill over of infection into mammalian dead-end hosts.
Not surprisingly, the majority of cases were within 10km of water courses and occurred within four months of above-average rainfall. In addition to climatic conditions, the outbreak was facilitated by a horse population naïve to flavivirus infection and the emergence of a new strain of WNV$_{KUN}$. This new strain, WNV$_{NSW11}$, is thought to have arisen from point mutations at key locations on the genome which resulted in increased virulence and development of neurological disease in horses.\(^9\)

While Kunjin virus is a zoonotic pathogen, very few human cases of infection occurred during the WNV$_{NSW11}$ outbreak. In contrast, increased numbers of human RRV infections were reported in 2011, consistent with increased vector availability, abundance of natural reservoir hosts (macropods) and increased opportunity for spill-over events in human populations in regions also experiencing increases in arboviral disease in horses.\(^{10,11}\)

Given the established ability of WNV to over-winter in vector/reservoir hosts and emergence of the more virulent WNVNSW11 strain, further outbreaks of flavivirus-associated neurological disease in southern Australia are possible. However, such outbreaks may be difficult to predict and require consideration of complex interactions between host susceptibility, strain virulence, climatic conditions and host and vector population dynamics. Surveillance programmes are in place in several jurisdictions and will serve to detect changes in virus activity.