



Epidemic viral diseases of wildlife – sudden death in tammar wallabies, blind kangaroos, herpesviruses in pilchards – what next?

In Australia the impact of European settlement on the indigenous human population and on flora and fauna is inevitably the subject of ongoing speculation. Major changes have occurred as a result of urban and rural developments and the introduction of agricultural practices which collectively impact on the environment and ecosystems especially through land clearing, water use and modification of water courses and water catchments. From both a human and animal health perspective, the changes as viewed by the general public are perhaps not always apparent but the impacts are no less significant. A range of microbial pathogens, parasites and other pests have been introduced to populations that often have not encountered these challenges before. Our indigenous wildlife populations have not been

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immune from these threats. And, if we include aquatic as well as terrestrial species of 'wildlife', and venture to our immediate coastline, in recent years there has been profound evidence of the impact that follows the introduction of an exotic pathogen.

Sometimes an epidemic has arisen following the movement of an insect vector beyond its usual range resulting in the incursion of an otherwise endemic

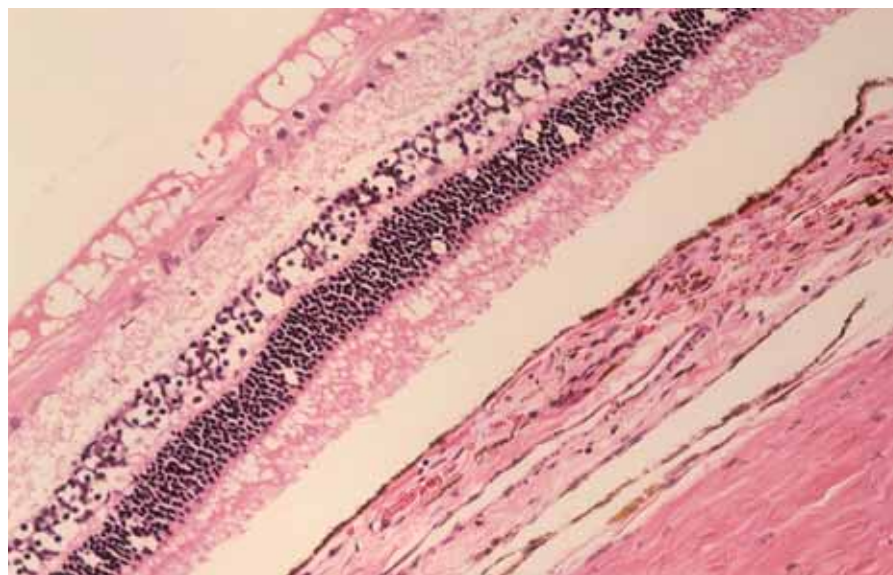
pathogen into a susceptible population. In other cases, disease outbreaks have resulted from the movement of indigenous fauna into new environments. In some instances, there have been devastating epidemics resulting from the introduction of an exotic pathogen to a wildlife population. In the last decade there has been an example of each of these events in Australian wildlife populations.

Tammar Sudden Death Syndrome (TSDS)

Tammar wallabies (*Macropus eugenii*) are small, nocturnal macropods that originally ranged throughout southwestern Western Australia, southern South Australia and offshore islands of these states¹. Free-ranging tammar wallabies are now geographically restricted to small areas of Western Australia and several offshore islands². Tammars are also maintained at a number of mainland captive breeding centres, zoological parks and research centres where the tammar wallaby is an important model species used for the study of marsupial biology, behaviour, and physiology.

In October 1998, a research facility in Sydney observed a sudden increase in deaths in its collection of tammar wallabies. Over a 6 week period more than 85 of the 234 tammar wallabies held at the facility died. Sudden deaths of tammar wallabies were also observed in 6 other research facilities and zoological gardens in New South Wales between October and December 1998 and in one

Figure 1. Histological section of a normal retina of a red kangaroo (Photo courtesy L. Reddacliffe, Elizabeth Macarthur Agricultural Institute).





research facility in Queensland in March 1999. One hundred and twenty tammar wallaby deaths were confirmed during this period though population census suggests that approximately 230 tammar wallabies may have died. The majority of animals died without premonitory signs. In all cases, sick animals died in less than 12 hours of being noticed to be ill.

Gross post mortem findings consisted of extensive congestion and haemorrhage in a range of thoracic and abdominal organs, major muscle groups and in subcutaneous tissues³. Viruses of the Orbivirus genus (family Reoviridae), probably from the Eubenangee serogroup, were isolated from tissue samples collected from a number of tammar wallabies from a research facility in Sydney. An apparently identical Orbivirus was also isolated from a tammar wallaby that died suddenly in the Queensland research facility. In many respects this disease resembles peracute bluetongue, another orbivirus infection of sheep.

Orbiviruses are arthropod borne viruses, usually spread by biting midges (*Culicoides* spp). The epidemics of TSDS occurred in the summer months

following periods of very heavy rain and when insects were abundant. As TSDS has never been described south of Sydney, it is probable that the southern limit of the vector is in the vicinity of Sydney. Therefore, it could be argued that these epidemics may have been the result of human intervention through the movement of a susceptible macropod species into a geographical area that is well north of its usual habitat and into the potential range of a pathogenic orbivirus.

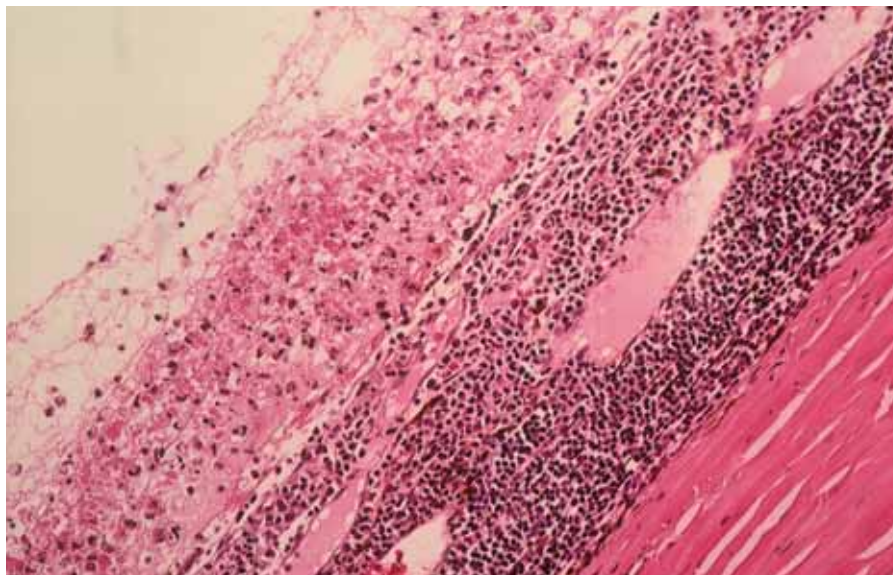
Kangaroo blindness

Between 1994 and 1996, a disease epidemic in kangaroos swept across the continent from the east, extending to the south and across to the west coast. During the autumn of 1994, large numbers of kangaroos were noticed in western New South Wales, South Australia and north-western Victoria exhibiting abnormal behaviour that was consistent with blindness. They were regularly observed beside roadways and in open areas close to humans and showed no apparent fear of humans or other animals. There were numerous accounts of kangaroos running directly into tree trunks, bushes, the side of motor vehicles and even people. The affected species

were mostly western grey kangaroos (*Macropus fuliginosus*), but eastern grey (*Macropus giganteus*), red kangaroos (*Macropus rufa*), and even euros (*Macropus robustus*), were affected. The outbreak resumed in the following summer between March and June 1995 in western New South Wales, South Australia, north-western Victoria and, later, in December 1995 to April 1996 in Western Australia⁴. Investigations involving several laboratories identified² arthropod-borne orbiviruses, Wallal and Warrego as possible causes^{5,6}. Subsequent transmission studies suggest that Wallal virus is the probable cause of this syndrome⁷. Clinical and histopathological examinations showed severe degeneration and inflammation in the eyes (Figures 1 & 2), with lesions predominantly in the retina. Wallal virus antigen was detected in the cells of the retina by immunofluorescence conducted on histological sections.

The strain of Wallal virus isolated from blind kangaroos was shown to be a variant but retrospective testing of fixed tissue blocks from 20 years previously identified a similar strain. The outbreak is thought to be due to the southern and then western movement of a pathogenic *Culicoides* borne virus into a susceptible population. It is likely that the virus has been endemic in the north rather than a new introduction to Australia.

Figure 2. Degenerative and inflammatory changes in the retina of a red kangaroo following experimental infection with Wallal virus (Photo courtesy L. Reddacliffe, Elizabeth Macarthur Agricultural Institute).



Pilchard Herpesvirus Infection

In 1998-9 massive numbers of dead pilchards, estimated at up to 65,000 tonnes nationally, were washed onto beaches along the entire southern coast of the continent and on the east coast as far north as Sydney. Subsequent investigations identified a herpesvirus infection of gill tissue as the cause of death of these fish^{8,9}. There has been intense debate and speculation that the causative virus was introduced in imported pilchards used to feed tuna being farmed in sea cages in South



Australia. Whether this epidemic was really due to the accidental introduction of an exotic pathogen into the fish populations in Australian waters will never be known, but the impact on the local fish population was devastating. There were also profound effects on a range of other aquatic and avian species that have pilchards in their diet.

Discussion

It is quite likely that human intervention has contributed to some or all of the epidemics that have been described. There is speculation that further incidents are likely to occur through the increasing movements of animals and man and, indirectly, through events such as climate change. The reality of permanent climate change can be debated, but large changes in temperature and rainfall patterns are not needed to bring about dramatic changes in the distribution of arthropod vectors of viruses. For example, a one degree rise in temperature could change the southern limit of some midges from central NSW to southern Victoria, resulting in a devastating spread of vector borne viruses into fully susceptible populations of wildlife and domestic animals¹⁰.

Of course, potential impacts are not limited to mammals and fish. North America has witnessed the effects of the introduction of an exotic virus, West Nile, on the wild bird population as well as on humans and horses. Whether a highly pathogenic strain of influenza virus would have any effect on our wild bird population is unclear, but the risks are high for all species. Finally, when pathogens of human and farm animals become established in wildlife populations, there is a likelihood of wildlife reservoirs becoming a source for future epidemics in susceptible species.

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