Neuro-angiostrongylosis in wild Black and Grey-headed flying foxes (*Pteropus* spp)

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Objective To identify nematodes seen in histological sections of brains of flying foxes (fruit bats) and describe the associated clinical disease and pathology.

Proceedures Gross and histological examination of brains from 86 free-living flying foxes with neurological disease was done as part of an ongoing surveillance program for Australian bat lyssavirus. Worms were recovered, or if seen in histological sections, extracted by maceration of half the brain and identified by microscopic examination. Histological archives were also reviewed.

Results There was histological evidence of angiostrongylosis in 16 of 86 recently submitted flying foxes with neurological disease and in one archival case from 1992. In 10 flying foxes, worms were definitively identified as *Angiostrongylus cantonensis* fifth-stage larvae. A worm fragment and third stage larvae were identified as *Angiostrongylus* sp, presumably *A cantonensis*, in a further three cases. The clinical picture was dominated by paresis, particularly of the hindlimbs, and depression, with flying foxes surviving up to 22 days in the care of wildlife volunteers. Brains containing fifth-stage larvae showed a moderate to severe eosinophilic and granulomatous meningoencephalitis (n = 14), whereas there was virtually no inflammation of the brains of bats which died when infected with only smaller, third-stage larvae (n = 3). There was no histological evidence of pulmonary involvement.

Conclusion This is the first report of the recovery and identification of *A cantonensis* from free-living Australian wildlife. While angiostrongylosis is a common cause of paresis in flying foxes, the initial clinical course cannot be differentiated from Australian bat lyssavirus infection, and wildlife carers should be urged not to attempt to rehabilitate flying foxes with neurological disease.
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CNS	Central nervous system
HE	Haematoxylin and eosin

The rat lungworm, *Angiostrongylus cantonensis*, is a metastrongylid nematode that inhabits the right ventricle and pulmonary arteries of various rats, being restricted in Australia to the two introduced species, *Rattus norvegicus* and *R rattus*. Since its lifecycle was elucidated in Brisbane almost 50 years ago,[1] it has been incriminated in infections in a wide range of mammalian hosts,[2] almost all presenting with disease of the CNS. Originally, cases were reported in coastal Queensland and north-eastern New South Wales, although, over the last decade, the parasite has been recognised in Sydney in dogs, rats, zoo primates and native mammals.[2]

Rats, and abnormal definitive hosts, acquire the infection by eating third stage larvae in the tissues of the intermediate hosts, slugs and snails, which have become infected by eating first-stage larvae expelled in rat faeces. Molluscivorous paratenic hosts (such as land planarians, freshwater crustacea) are sometimes infected, although their importance in Australia is unknown. Once in the definitive host, infective larvae undergo an obligatory migration through the CNS, where they grow and moult twice to attain the immature adult stage. They then invade the venous system to return to the right ventricular outflow, where, in suitable hosts, they copulate and produce eggs. Clinical disease arises from mechanical damage and associated inflammation in nervous tissue, blood vessels and the subarachnoid space.

Recently, the parasite was reported for the first time in flying foxes (fruit bats), in captive *Pteropus poliocephalus* in Sydney.[3] We have been aware for at least 9 years of the sporadic finding of nematodes in histological sections of brain from neurologically-affected captive and wild flying foxes in south-east Queensland and northern New South Wales, although the identity of these parasites had never been established. Here, we now report the first confirmed *A cantonensis* infections among wild Black and Grey-headed flying foxes (*Pteropus alecto* and *P poliocephalus*, respectively) from this region.

Materials and methods

From November 1997 to November 2000, inclusive, 86 wild flying foxes with neurological signs were necropsied at the Queensland Department of Primary Industries Animal Research Institute, Yeerongpilly, as part of an ongoing surveillance program for Australian bat lyssavirus infection. They comprised 51 *P alecto*, 24 *P poliocephalus*, 9 *P scapulatus* (Little Red flying fox) and 2 *P conspicillatus* (Spectacled flying fox). For lyssavirus detection, impression smears of fresh brain were fixed in acetone and subjected to a direct fluorescent antibody test using the Centocor® fluorescein-labelled anti-rabies reagent (Centocor Inc. Malvern, PA, USA). Half the brain was then stored at -70°C, whereas the other half was fixed immediately in 10% buffered neutral formalin and processed for routine histology.

A worm recovered from the brain surface of one of the 86 flying foxes was fixed in 10% buffered neutral formalin for identification. Nematodes were seen histologically in brain sections of another 15 of the 86 flying foxes. The frozen half-brains of these 15 bats were subsequently fixed at room temperature in 10% buffered neutral formalin, then gently macerated under stereomicroscopical guidance. All worms found were mounted in chlorlactophenol on a glass slide under a coverslip, examined by compound microscopy, drawn using a camera lucida attachment and measured. Formalin-fixed lungs, available from all except bat 7, were processed so that HE-stained

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sections through the hilar regions could be examined microscopically for nematodes or eggs within the major pulmonary arterial branches.

Histological sections of the brain and lung of an additional specimen (bat 17) were retrieved from the archives of the Veterinary Pathology laboratory of The University of Queensland Veterinary School; fresh brain material was not available for lyssavirus testing, nor fixed tissue for worm extraction.

Results

The case details and clinical histories of 17 cases of neuroangiostrongylosis in flying foxes are shown in Table 1.

Bats 1-16 were negative for lyssaviruses by direct fluorescent antibody test; bat 17 was not tested.

Gross pathological findings

Ten of the 16 bats submitted to the Animal Research Institute, and bat 17, were well-nourished, with ample fat reserves. The conditions of bats 7 and 8 were poor, while those of bats 1, 2, 3 and 4 were not recorded. Bat 6, maintained in care for 22 days, had considerable amounts of abdominal fat, but wasting of the pectoral muscles and brown discoloration of the meninges. Bat 3 had a retroperitoneal haemorrhage at the level of the left adrenal gland. Although no spinal fracture was detected grossly, the spinal column was removed for radiological examination, which revealed displacement of the caudal endplate of the third lumbar vertebra. Petechial hemorrhages were found on the cerebral cortices of bats 11, 12 and 13, whereas the meninges of bat 11 were congested, and the ventral meninges of bats 13 and 15 were cloudy. One nematode was recovered from the dorsal surface of the cerebellum and brain stem of bat 15.

Histopathology

Eosinophilic and granulomatous meningoencephalitis, and one or more sections of nematodes, as shown in Figures 1 and 2, were found in brain sections from bats 1, 2, 5 to 13, 15, 16 and 17 (n = 14). The meningitis was typically most severe around the brain stem and cerebellum, with macrophages predominating in the infiltrate, often with large numbers of lymphocytes and eosinophils, some plasma cells, and occasionally, multinucleated giant cells. In all but one of the recent cases there were perivascular cuffs of macrophages, lymphocytes and eosinophils. These perivascular cuffs were particularly prominent in bats 1, 2, 7, and 8. Foci and tracts of tissue disruption, gliosis, and/or haemorrhage, presumably the result of larval migration, were seen in sections from 10 of the recent cases and were most severe in bats 5, 6, 11, and 13. Nematode sections were seen most frequently in the subarachnoid space of the cerebral sulci and/or cerebellar folds, but some were located within the cerebral cortex, thalamus, cerebellum, brain stem and lateral ventricles. Bats 3, 4, and 14 (n = 3) were distinct in that they displayed virtually no inflammation, and their brain parenchyma appeared unremarkable except for the occasional presence of very small nematodes, as shown in Figure 2 (insert).

Neither nematodes nor their eggs were found in any lung sections.

Identification of worms

Worms recovered from bats 2, 5, 6, 9 to 13, 15 and 16 (n = 10) were identified as fifth-stage (L5) larvae ('immature adults')

Table 1 Case details and clinical histories of 17 flying foxes with neuroangiostrongylosis.

Bat	Description	Month/year, place found and clinical history
1	Black adult M	11/1997 Cleveland. Found on ground, aged, debilitated, persistent hind limb paresis, passive disposition, killed 7 days later.
2	Black adult M	11/1997 Brisbane. Rescued from wire fence, paresis, poor hind limb reflexes, killed 3 days later.
3	Black adult M	11/1997 Nambour. Found under palm tree, flaccid paralysis all limbs with absent grip and placing reflexes, full bladder, incontinent, killed within 24 hours.
4	Black adult M	11/1997 Deagon. Docile in tree on the foot path. Next day, 'frothing' at mouth, head tilt, unequal pupils, killed.
5	Grey adult M	05/1998 Ashgrove. Found on ground, put in tree, didn't move, rescued next day. No improvement over 7 days, killed.
6	Black juvenile M	06/1998 Bundamba. Hanging low in tree, docile, paresis, initially able to hang. In care 22 days, became recumbent, cranial nerves normal, poor hind limb reflexes, killed.
7	Grey juvenile F	12/1998 Nerang. Found weak and uncoordinated on ground by dogs, killed by dog owner.
8	Black juvenile F	10/1999 Brisbane. Found in tree, crashed when attempted to fly, dehydrated, generalized paresis, died same day.
9	Black adult F	10/1999 Everton Park. Seen in tree, next day on ground, ascending hind limb paresis. Day 3, delivered stillborn pup. Persistent paresis, then day 16 head tilt, head tremor, nystagmus, pallor, killed.
10	Grey adult M	10/1999 Broadbeach. Found on ground, hind limb paresis, depressed intermittent shiver ing, no improvement over 8 days, killed.
11	Black juvenile F	05/2000 Clontarf. Captive born, released. 2 to 3 weeks later found depressed in tree near cage, easily captured, movements stiff and slow. Deteriorated over 24 hours, nystagmus, died.
12	Black adult F	05/2000 Southport. Found on ground, depressed, wide based stance, drooped wings, ate well, day 3 recumbent, killed.
13	Black juvenile M	06/2000 Hope Is. Found on ground, deterio rated over 7 days, profoundly depressed, generalised paresis, nystagmus killed.
14	Black adult F	06/2000 Southport. Seen on ground under palm tree, rescued next day, depressed, unable to hang, hind limb paresis, killed.
15	Grey adult	F 11/2000 Miami. Found low in bush, would not fly, docile. Hung with wide based stance, wings held loosely. By day 9 inappetent, depressed, killed
16	Black adult M	11/2000 Palm Beach. Found low on fence, rescued 3 days later, moribund, recumbent, no voluntary limb movement, killed.
17	Black adult M	04/1992 location not recorded. Unable to hang, tremors, Treated for suspected Pb poisoning. Day 15, depressed, respiratory distress, died.

Black = *Pteropus alecto* (Black flying fox).
Grey = *Pteropus poliocephalus* (Grey-headed flying fox).
M = male, F = Female

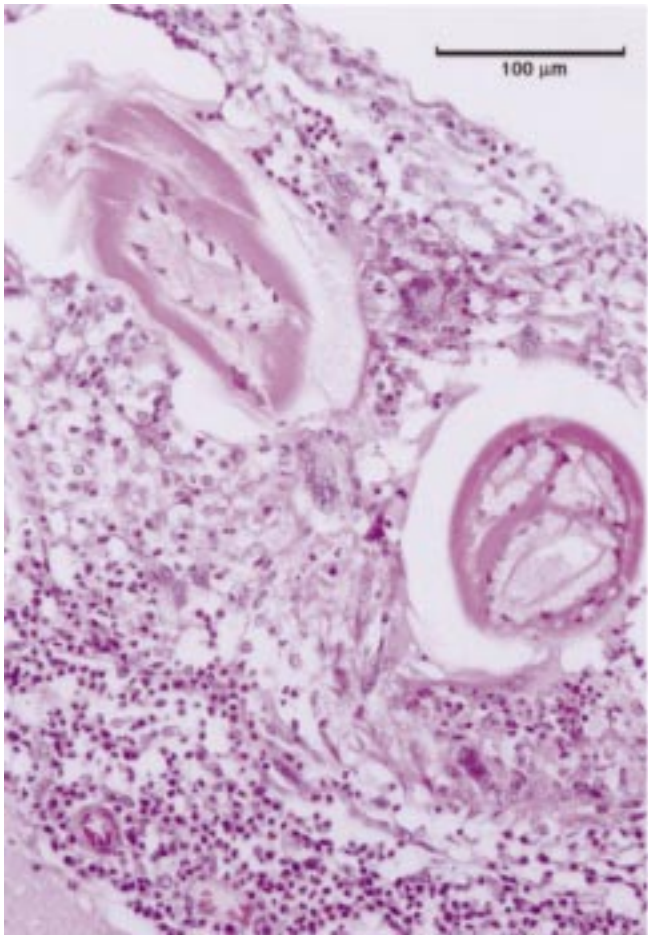

Figure 1. Female *Angiostrongylus cantonensis* (L5, maximum diameter 102 mm) from bat 6 within a focus of granulomatous meningitis containing macrophages, lymphocytes, eosinophils, and multinucleated giant cells.

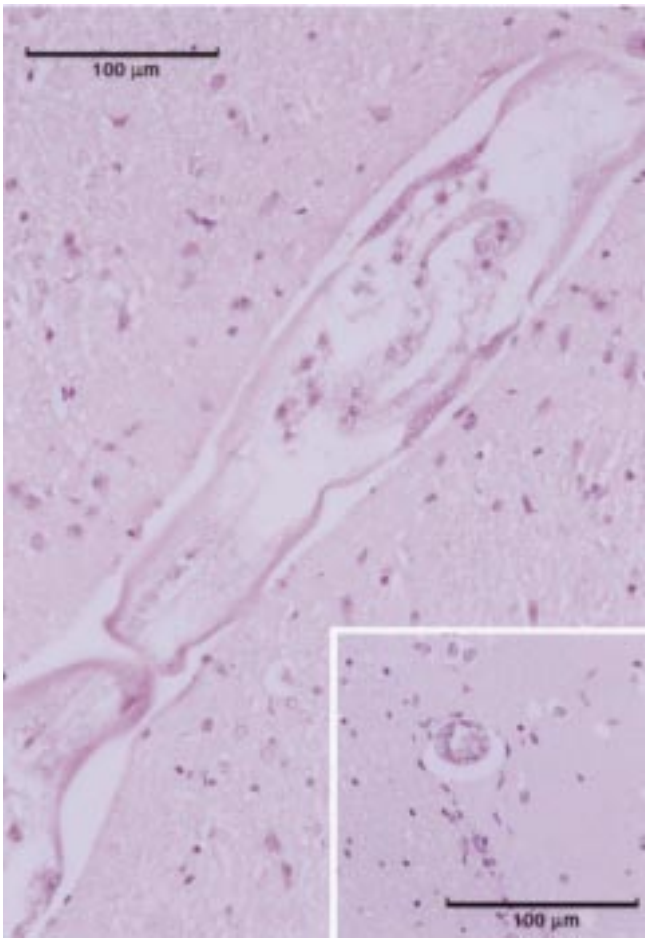

Figure 2. *Angiostrongylus cantonensis* (L5, maximum diameter 96 mm) migrating through the thalamus of bat 11, as indicated by the absence of an adjacent inflammatory reaction. Severe granulomatous meningitis was seen elsewhere in this section in association with other worms. Insert is *Angiostrongylus* sp (L3, maximum diameter 29 mm) in the frontal cortex of bat 14; early stage infection as indicated by immaturity of larva and lack of inflammatory response in the meninges and brain.

of *A cantonensis*, as distinct from *A mackerrasae*, by the relatively long spicules in the males (0.65 to 1.46 mm) and absence of a caudal mucron in the females. A fragment of an adult worm consistent with *Angiostrongylus* was recovered from bat 8. Multiple third-stage larvae (L3) 420 to 565 µm in length were recovered from bats 3 and 4; their identification as *Angiostrongylus* L3 was based on the overall size and shape, internal morphology and dimensions, the buccal details (including a 'double spear') and tail shape.[1,4] Worms were not recovered from the brains of bats 1, 7, or 14, whereas the brain of bat 17 was not available for examination. The number and lengths of worms recovered from each bat are shown in Table 2. The duration of infection was estimated by comparing the size of nematodes with published maximum growth rates in experimental rats.[1] In retrospect, some of this data is known to be derived from *A mackerrasae*, although the growth of *A cantonensis* in comparable stages is almost identical.[4,5] It was assumed that growth rates in bats, presumably abnormal hosts, was less than or equal to the rate found in rats, the natural hosts.

Discussion

Previous reports of infection with *A cantonensis* were in humans, captive wildlife, or domestic animals.[2] This is the first report of *A cantonensis* as a cause of disease in free-living populations of Australian native wildlife in which the nematode has been recovered and identified. Between November 1997 and November 2000, of 86 flying foxes with CNS disease, 16 were diagnosed with neuro-angiostrongylosis (19%), indicating this to be a common cause of neurological disease in flying foxes. Histological evidence of infection in another flying fox was also found in archival material from 1992. All the flying foxes with angiostrongylosis presented in south-east Queensland. Nematodes were recovered from the brains of 10 of the 16 recent cases, and morphologically confirmed as *A cantonensis.* The adult worm fragment and third stage larvae recovered from a further three cases, and histological sections of nematodes in the remaining three, had features consistent with those of *Angiostrongylus* spp, presumably *A cantonensis.* To date, only the introduced parasite, *A cantonensis*, not its native cogener, *A mackerrasae*, has been identified as a cause of disease in humans and animals.[2]

The number of cases involving Grey-headed compared to Black flying foxes (4 and 13, respectively) reflects the relative numbers of these species in the study (24 and 51, respectively). Whereas the parasite was not identified in the Little red (*P scapulatus*) or Spectacled flying fox (*P conspicillatus*), this may

Table 2. Numbers and lengths of *Angiostrongylus cantonensis* recovered from 16 free-living flying foxes and estimated duration of infection.

Bat	Male worms		Female worms		Total worms	Time in care	Estimated duration
	No.	Length (mm)	No.	Length (mm)	per half brain[a]	(days)	of infection[b] (days)
1	0	–	0	-	0	7	–
2	1	3.5	0	-	1	3	16
3	?	–	?	-	11[c]	1	2 – 3
4	?	–	?	-	7[c]	1	2 – 3
5	2	4.2 and 6.0	14	4.5 – 15.4	16	7	34
6	9	4.4 – 7.2	11	3.9 – 9.7	20	22	24
7	0	–	0	–	0	1	–
8	?	–	?	–	1 (piece)	1	–
9	7	3.9 – 8.9	7	8.6 – 10.1	14	16	25
10	3	3.9 – 4.3	5	4.4[d]	8	8	18
11	8	2.9 – 5.0	12	3.6 – 4	20	1	19
12	12	2.6 – 5.1	19	2.6 – 4.7	31	3	19
13	26	4.4 – 9.2	14	6.8 – 11.0	43	7	27
14	0	–	0	–	0	1	–
15	–	–	1[e]	8.8	1	9	23
16	2	2.3 and 2.9	1	2.2	3	3	14

[a]Total number derived from intact specimens plus pieces recovered.
[b]Estimated duration of infection at death calculated from recovered worm lengths and published growth rates.
[c]Immature third stage larvae 420 to 565 µm long, sex differentiation not possible.
[d]Only one could be measured.
[e]Single female worm recovered from brain surface, worms not extracted from fixed brain.

simply reflect their small numbers in the study (10 and 2, respectively). Histological evidence of *Angiostrongylus* sp has been reported in *P scapulatus* from Queensland.[3] The presumptive finding of this parasite in histological sections from 1992 (bat 17) suggests that it has been affecting flying foxes for some time. Surveillance of flying foxes has intensified since they were linked with Hendra virus (previously equine morbillivirus), and the discovery of Australian bat lyssavirus. Given the scanty material available from flying foxes before 1996, it is not possible to conclude that the apparently high number of cases described here reflects a changing prevalence of angiostrongylosis.

Presumably, flying foxes acquire infection by ingesting intermediate or paratenic hosts, such as slugs, snails and planarians, or their slime (although slime has still not been confirmed to be a vehicle of infection).[2] These hosts could readily be concealed among fruit. While it might seem unlikely that flying foxes would eat these invertebrates, faecal analysis has shown that flying foxes regularly ingest insects, spiders and mites.[6] Slugs and snails do climb high into trees, especially in wet weather (P Prociv unpublished observation), and might either prove attractive to a hungry flying fox, or be ingested unintentionally.

Continuing habitat destruction in south-east Queensland has been linked to a reduction in flying fox numbers and fragmentation of their colonies, with the establishment of smaller, more numerous colonies in urban areas.[7] Habitat destruction, with overgrowth of introduced plant species, could force hungry flying foxes to eat unusual foods, such as molluscs. When food is scarce, flying foxes will retrieve fallen fruit from the ground, thus increasing their risk of ingesting molluscs. However, the good nutritional condition of 11 of the 17 cases suggests these bats had not had difficulty finding food. The circumstances and climatic conditions under which the flying foxes acquired the parasite are unknown, because infection might have occurred considerable distances from where they were found. Black flying foxes in urban south-east Queensland typically fly up to 7 km per night to feed (N Markus personal communication) and also migrate considerable distances (>100 km during 10 weeks) to find native fruit and flowering trees.[8] Grey-headed flying foxes from northern NSW have been shown to fly even greater distances each night (up to 50 km) and seasonally (in excess of 600 km).[9] During the 3 years of the study, cases were clustered in the months of April-June or October-December. It is possible that during these months, particular trees that are in flower or fruit attract both flying foxes and molluscs, bringing these species together. Four captive Grey-headed flying foxes with angiostrongylosis from Sydney also presented during May and June,[3] but this coincidence should be interpreted cautiously as the factors leading to infection of captive flying foxes in temperate Sydney might differ from those for free-living flying foxes in subtropical Queensland.

In each bat, worms were likely to have died before necropsy and fixation of the brain, and the worm lengths as measured are almost certainly accurate. Worm growth rates in flying foxes might differ from those in their normal definitive hosts. Nevertheless, assuming growth rates less than or equal to the rate in rats, the estimated duration of infection in some of the cases allows for interesting conclusions. Three flying foxes (bats 3, 4 and 14) had only very small nematodes in histological sections of brain and virtually no encephalitis, suggesting early infection. Immature L3 only were recovered from bats 3 and 4, and all three flying foxes were killed within 24 hours of being found. It appears these three flying foxes presented with acute, severe disease just 1 to 3 days after infection. Bat 6 also appears

to have been found only 1 to 2 days after infection, but survived in care for as long as 22 days. The remainder (n = 9) appear to have been found, unable to fly, 9 to 27 days after infection, and died or were killed 14 to 34 days after infection. Bat 11, the captive-born juvenile, appears to have been infected shortly after release. Bat 13 had a particularly intense exposure, with 43 worms recovered from half the brain, yet this bat appears to have been infected for 20 days before being found, and survived a further 7 days in care before being killed. There seems to be no correlation between the number of worms recovered (per half-brain) and either the estimated interval after infection when found, or the duration of infection before death. These estimates suggest that most bats become affected and cannot fly 1 to 3 weeks after infection. This parallels human infections: in one well-documented outbreak in a group of men, several developed neurological symptoms as early as 1 to 2 days after exposure, whereas most were hospitalised within 1 to 2 weeks of the onset of symptoms.[10]

Ideally, finding worms in the right ventricular outflow would require fresh tissue for careful dissection of the heart and pulmonary arterial tree, but all heart and lung samples had been fixed. Branches of the major pulmonary arteries were considered most likely to harbor adult worms, yet none was found in transverse sections of the pulmonary hilar region, suggesting that *A cantonensis* had not migrated beyond the CNS. The lungs of infected aberrant hosts are rarely examined specifically for the presence of adult worms, but when this parasite has been found in human lungs, it has been in cases with heavy and prolonged infections.[11,12] The ability of *Angiostrongylus* to reach the lungs and develop further probably varies with the host species.

Of the 16 recent cases, eight were found on the ground, six were found hanging inappropriately low in a tree and unable to fly away, and two were rescued from fences. The clinical picture was dominated by paresis, particularly of the hind limbs, and depression. Bat 3, which had severe hindlimb paralysis and incontinence, was shown radiologically to have a fractured lumbar vertebra. Presumably, this flying fox had been predisposed to an accident by infection with *Angiostrongylus* L3, and the clinical presentation reflected both the spinal fracture and neuro-angiostrongylosis. Whereas six flying foxes died or were killed the day they were found, 11 of the 17 cases survived in the care of voluntary wildlife carers for variable periods: 3 days (n = 3), 7 to 9 days (n = 5) and > 15 days (n = 3). Their histories and initial clinical signs closely resemble cases of Australian bat lyssavirus infection[13,14] (J Barrett, unpublished observations). Given the difficulty of distinguishing angiostrongylosis from Australian bat lyssavirus infection on clinical signs, the risks associated with caring for bats infected with Australian bat lyssavirus,[15] and the poor prognosis of both diseases, wildlife carers should be urged not to care for flying foxes with neurological disease, but to submit them for lyssavirus testing.

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