PARASITES FOUND IN THE TAWNY FROGMOUTH (*PODARGUS STRIGOIDES*) IN SOUTH-EAST QUEENSLAND

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INTRODUCTION

The following is a case report describing the parasites found in wild tawny frogmouths (*Podargus strigoides*) from south-east Queensland as presented to the Australian Wildlife Hospital in Beerwah, Queensland.

Tawny frogmouths belong to the Order Caprimulgiformes, Family Podargidae. They are often mistakenly called owls but are actually more closely related to nightjars. They are found throughout mainland Australia and Tasmania, often close to major cities where suitable woodland habitat persists. They are nocturnal, using their wide mouths, large eyes and silent flight to capture insects and small vertebrates. By day, their cryptic colouration and static perching posture allow them to melt into their background.

This case report describes an epizootic outbreak of neurologic disease in tawny frogmouths originating from southeast Queensland during May 2009.

CASE REPORT

During a period of 28 days 10 birds were presented with a clinical history of being found on the ground, unable to fly. Clinical examination revealed the birds to be paretic and unable to perch. Deep pain responses were present but withdrawal reflexes were variable, from very slow to nearly normal. Most birds were able to extend their wings but attempts at coordinated wing flapping or flight were unsuccessful. Pupillary light reflexes were variable but this is not unusual with this

species in our experience. No strabismus or nystagmus was noted in any bird and ocular examination was unremarkable. Most birds were presented in poor body condition. All birds were anaesthetised, radiographed and blood samples taken for determination of PCV, TP and for blood smear examination.

Radiographs taken in both ventro-dorsal and lateral views were unremarkable. PCVs varied between 20-55 L/L, but most birds ranged between 20-30 L/L. Total Protein as determined by refractometer varied between 40-60 g/L with most birds at the lower end of the range.

Early treatments included meloxicam 0.4mg/kg IM BID for 5 days; vitamin B complex 0.1ml IM SID for 5 days and parenteral fluid therapy (Hartmann's Solution or 0.9% Sodium Chloride) SQ, IM or IV.

None of the initial birds responded and in some the neurologic signs progressed to total paresis. These birds were euthanased on welfare grounds, in some instances only 48 hours after admission. Both sexes were affected and most birds were identified as juveniles. Following the results of histopathological examination, it was decided to attempt anthelmintic treatment in combination with anti-inflammatory treatment for subsequent cases.

One bird was treated with Wormout Gel (Oxfendazole 20mg/ml; praziquantel 20mg/ml) at 10mg/kg oxfendazole , twice, 7 days apart; enrofloxacin 15 mg/kg diluted with 0.9% sodium chloride IM BID for 5 days; Meloxicam 0.5mg/kg IM BID for 5 days and subcutaneous fluids for 3 days. The bird showed a slight level of improvement after the first week, by starting to stand and perch, but after 4 weeks had regressed to the point that it was euthanased.

A further bird was treated as above except that injectable Ivermectin was used as the anthelmintic at 200 ug/kg IM daily for 5 days. This bird also improved to the point that it was able to stand up and attempt wing-flapping, but would fall over if it tried to fly. After a further four weeks in care, there was no further improvement and the bird was euthanased.

The final bird presented was initially treated with Solu-Delta-Cortef (prednisolone sodium succinate) at 15 mg/kg IV, followed 12 hours later by Meloxicam 0.4 mg/kg IM BID/5days; Enrofloxacin 20 mg/kg in subcutaneous fluids SID for 5 days and Moxidectin 200ug/kg IM , then orally at the same dose SID for 4 days. This bird showed steady improvement so that by six weeks post presentation its ability to perch, grasp, fly and judge distances was considered normal. This bird was subsequently released.

HISTOPATHOLOGICAL FINDINGS

The one finding common to all birds examined histopathologically was the presence of larval nematodes in the CNS. They were found either in the brain parenchyma, meninges, ventricles, cerebellum or spinal cord. These larval nematodes were identified as *Angiostrongylus cantonensis*,

the rat lungworm.

Infections were characterised by mild-moderate mixed inflammatory cellular infiltration, mild congestion and vascular proliferation. Some exhibited moderate diffuse infiltration of macrophages; others showed multifocal areas of granulomatous inflammation. Occasionally, *A. cantonensis* larvae were found in other organs such as the liver and adrenal gland.

Leucocytozoonosis was also diagnosed in all affected birds. Gametocytes were present throughout the blood vessels especially in organs such as the liver. Large endothelial schizonts were seen in the blood vessels of the kidney in one bird. There was no associated pathology found.

Large numbers of trematodes identified as *Brachylecithum podargi* were present in the liver, bile ducts and in one case the pancreatic ducts. These were not associated with any inflammatory reaction.

Unidentified intestinal cestodes were found in the gastrointestinal tract of two birds. Gastrointestinal nematodes identified as *Allodapa suctoria* and an unidentified species of *Trichostrongylus* were also found but were also not associated with any pathology.

Microfilaria were found in blood vessels around the body of several birds. However, no adult filaroids were identified. Unidentified subcutaneous mites were identified in loose connective tissue. These were not associated with any inflammation. Hippoboscid flies were also found on several birds. Their presence was not associated with anaemia or other pathology.

DISCUSSION

Angiostrongylosis has previously been described as a cause of neurologic disease in tawny frogmouths from Sydney, NSW (Monks et al., 2005; Montali et al., 2004). However, this is the first report of such an infection occurring in this species in south-eastern Queensland.

***Angiostrongylus cantonensis*, the rat lungworm, lives as an adult in the pulmonary arteries of the introduced brown rat (*Rattus norvegicus*) and black (ship) rat (*Rattus rattus*). The eggs are deposited in the lungs and the first stage larvae are expelled through the rat's faeces. These larvae infect a molluscan intermediate host, usually a snail or slug. The rat becomes infected by eating infected molluscs or infected paratenic hosts such as frogs and toads, freshwater prawns, land crabs and planarians. The third stage larvae then pass through the brain to reach the pulmonary arteries. If the infected intermediate hosts are ingested by accidental hosts such as tawny frogmouths or human beings, then the life cycle is not completed. However, the obligatory migratory phase through the host's central nervous system may still occur, causing severe clinical signs and pathology.**

Human beings may also become accidental hosts with serious consequences. Infection in humans is characterised by an eosinophilic meningoencephalitis which may prove fatal. Human infection usually occurs by either ingesting unwashed salads which may have been contaminated by infected

molluscs, or by the ingestion of uncooked snails or slugs.

Other vertebrates reported to be infected and showing neurological signs include the captive yellow-tailed black cockatoo (*Calyptorhynchus funereus*), dog, horse, tamarin (*Saguinus* spp), Bennett's wallaby (*Macropus rufogriseus*), rufous bettong (*Aepyprymnus rufescens*) and grey-headed flying fox (*Pteropus poliocephalus*). Paratenic hosts include frogs and toads, freshwater prawns, land crabs and planarians. It is suspected that tawny frogmouths are infected by ingesting snails and slugs which have themselves accidentally ingested infected rat faeces.

It is interesting to note that there is another species, *Angiostrongylus mackerrasae*, which is endemic to Australia and completes its lifestyle within two native rat species, the bush rat (*Rattus fuscipes)* and the swamp rat (*R. lutreolus).* In contrast to *A. cantonensis*, it has not been found in abnormal vertebrate hosts, even though the two species have been found to coexist in the introduced *R. norvegicus* around Brisbane (Spratt, 1999).

Treatment options investigated during this study were not particularly successful. It has been suggested that killing the parasite may actually increase the inflammatory reaction and hence the severity of the clinical signs. However, the combined use of extended concurrent use of anthelmintics and anti-inflammatories warrant further investigation in future outbreaks, given the response noted in the later cases.

Another nematode identified is *Allodapa suctoria,* a subulurid nematode in the order Ascaridida. The adult commonly infects the caecum of poultry where it is considered to be mildly pathogenic. It has an indirect life-cycle with the infective juveniles (J2) being encapsulated within the haemocoel of the intermediate host (cockroaches and beetles). J2s are released from inside the beetle by the action of bile acids (Parker et al., 1982). Within the tawny frogmouths examined, this parasite did not appear to cause any pathology.

Various trichostrongylid species have been described from the gastrointestinal tract of birds with varying pathogenicity. For example, *Trichostrongylus tenuis*, which inhabits the small intestines and caeca of galliform birds, ducks and geese, has been implicated in outbreaks of severe enteritis in game birds. However, it is not considered to be pathogenic in North American *Galliformes*, being commonly found in northern bobwhite (C*olinus virginianus)* without causing any lesions or inflammation (Freehling and Moore., 1993). This would also appear to be the case in the tawny frogmouth. Although the exact species of trichostrongylus was not determined, the life-cycle of these parasites tends to be direct (Urquhart et al., 1987).

The liver fluke *Brachylecithum podargi* was previously found to infect tawny frogmouths from the Gatton area of Queensland (Mawson et al., 1986). Its presence was not associated with any damage or inflammation. Although its exact life cycle has not been determined it would be expected to follow that of other liver flukes ie indirect life cycles involving snails and slugs as intermediate hosts. Thus snails, which are known to form part of the tawny frogmouth's diet, may serve as an intermediate host for at least two tawny frogmouth parasite species: *A. cantonensis* and *B. podargi.*

Many unidentified cestodes were found in the gastrointestinal tracts of some birds, but were not associated with any lesions. Cestodes typically have indirect life cycles involving insect or arthropod intermediate hosts.

Leucocytozoon sp **are haemoparasites, with the gametocyte stage present in the peripheral blood of birds where the parasites grossly distort the cytoplasm of either the erythrocytes (or leucocytes) which they infect. They are transmitted by biting fly (*Simulium* spp) vectors, in which sporozoites are produced within the salivary glands. Many native bird species can be infected, including honeyeaters, tawny frogmouths, psittacine birds, pigeons, orioles, magpies, currawongs, and kingfishers, with most infections appearing to be asymptomatic (Peirce et al., 2004). This also as appears to be the case with all the tawny frogmouths examined in this study. However, anaemia, paralysis and sudden death has been attributed to this parasite in several species (Urquhart et al. 1987; Macwhirter 1994). Some species have been found to be highly pathogenic for some species such as young waterfowl and turkeys (Campbell and Ellis, 2007). Diagnosis depends on the recognition and differentiation of the parasites in the erythrocytes (or leucocytes) of stained blood films.**

The microfilariae seen were non pathogenic and no adult parasites were found. They have also been seen by the authors in magpies and currawongs from southeast Queensland. Microfilaria have been found in at least 60 species of Australian native birds where adult filarial worms were not identified (Reppas et al., 1995; Ladds, 2009). These studies also noted little or no inflammatory response to the microfilarial infections.

Two species of arthropod parasites were identified in the tawny frogmouths examined. The first, the subcutaneous mite, was not formally identified but given the lack of pathology associated with it is likely to be a harmless commensal. The second is the hippoboscid fly, a dorsoventrally flattened fly with a soft leathery abdomen which lives under the bird's feathers and feeds on blood and other body fluids. It is found on many Australian avian species and is commonly seen in species such as kookaburras and magpies and some psittacine birds. This may make it an important vector for haemoparasites, in particular *Leucocytozoon* spp. Heavy burdens in debilitated birds may also result in severe anaemia.

CONCLUSIONS

This study documents the presence of neurological disease in tawny frogmouths in southeast Queensland caused by *Angiostrongylus cantonensis.* It highlights the importance of monitoring disease outbreaks in native wildlife, not only for the protection of the wildlife itself, but also for understanding the pathobiology of potential zoonotic diseases.

The presence of an array of apparently non pathogenic parasites of tawny frogmouths is also recorded including a number of nematodes, cestodes, flukes, haemoparasites and arthropods.

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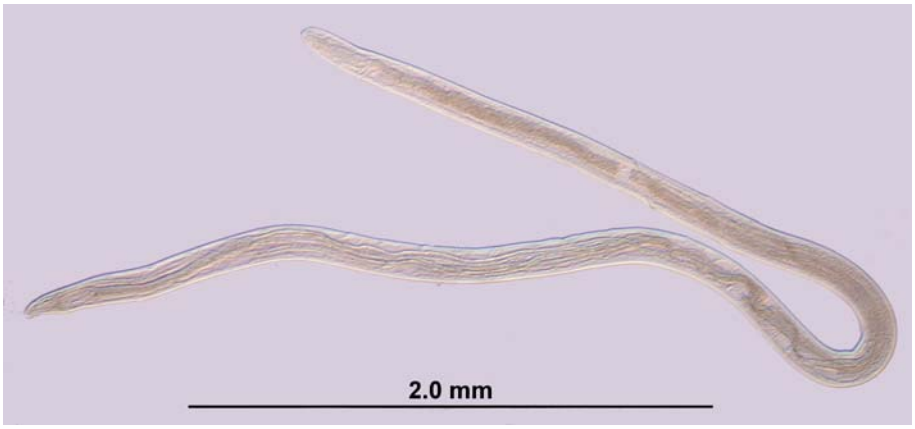

Figure 1. Larval Angiostrongylus cantonensis recovered from the formalin-fixed brain of a Tawny frogmouth.

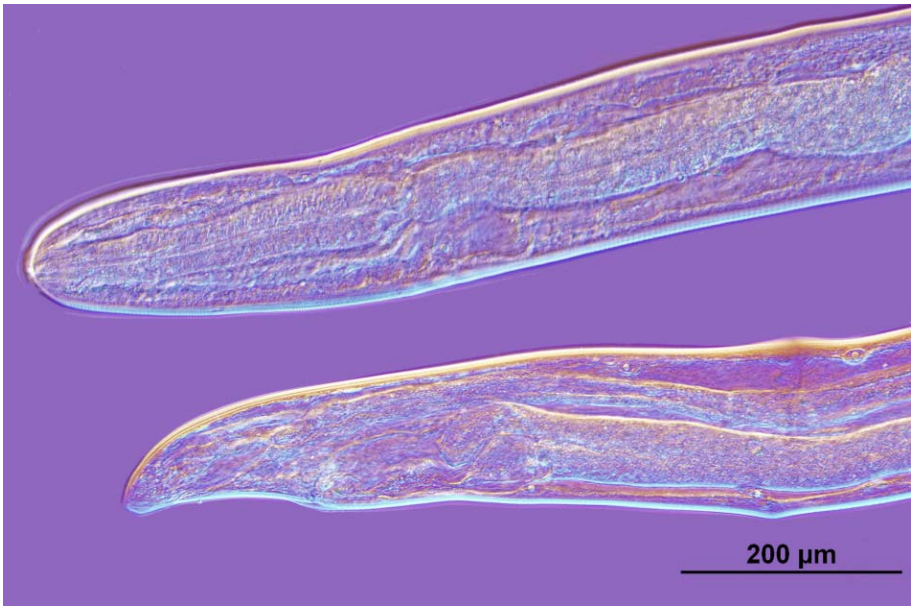

Figure 2. Higher power view of head and tail regions of larval A cantonensis shown in Fig 1.

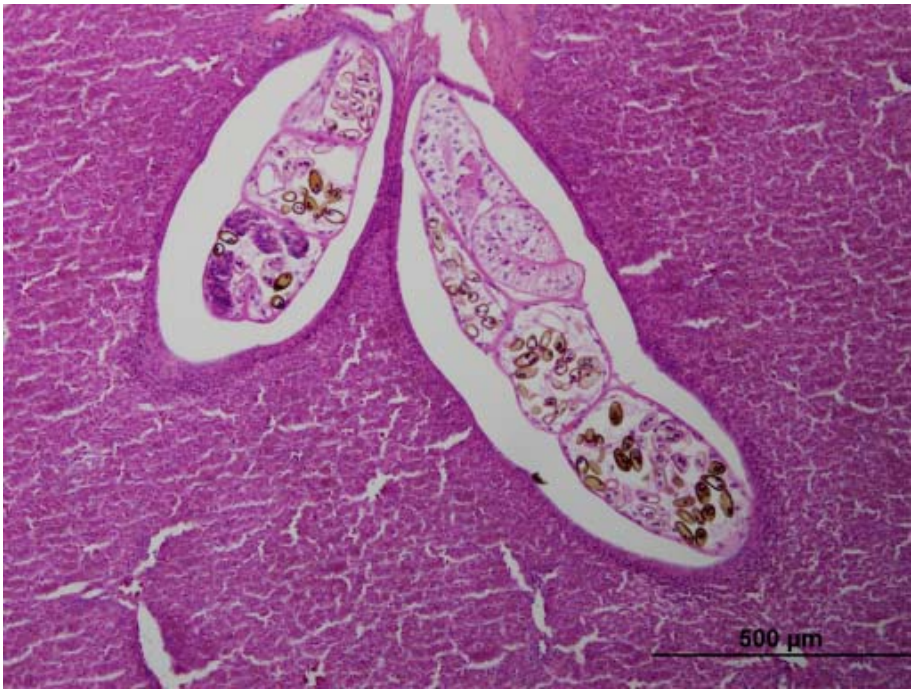

Figure 3. **Histological section of liver demonstrating two adult *Brachylecithum podargi* within bile ducts.**

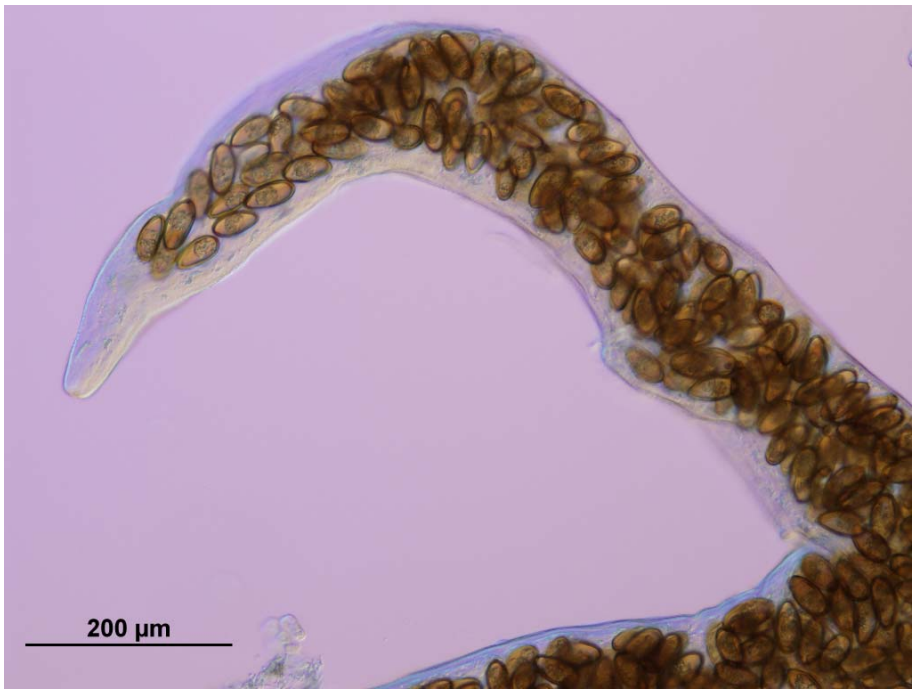

Figure 4. **Higher power view of an adult Brachylecithum podargi recovered from formalin fixed liver showing abundant eggs within the trematode.**