# Neuroangiostrongyliasis: disease in wildlife and humans

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Angiostrongyliasis is a neurological disease caused by the rat lungworm, *Angiostrongylus cantonensis,* one of the most catholic nematode parasites of vertebrates. Infection has occurred accidentally in humans, a broad spectrum of eutherian and marsupial mammals, and recently in birds[1,2].

In February 1998, *A. cantonensis* infection was diagnosed in a captive-bred yellow-tailed black cockatoo in Brisbane[2]. Between December 2000 and May 2002, *A.. cantonensis* infection was diagnosed in two free-living tawny frogmouths in the Sydney area[2]. Subsequently, in the period March to June 2004, a disease affecting the central nervous system of frogmouths became apparent in the northern suburbs of Sydney. Birds were weak, unable to perch or fly and often unable to right themselves (Figure 1). *Angiostrongylus cantonensis* was recovered from or observed in histological sections of the brains and spinal cords of 13 of 22 tawny frogmouths necropsied, the birds serving as virtual biosentinels for the occurrence of this parasite in the suburban environment.

Infection has occurred in captive kangaroo, wallaby, rock wallaby and bettong species, and in brushtail and ringtail possums[1]. Captive and wild flying foxes suspected of lyssavirus infection, some exhibiting neurological signs, also have been shown to be infected with *A. cantonensis*[1,3].

This parasite is now well recognised as the primary cause of eosinophilic meningoencephalitis in humans in an expanding area of the world[1]. It has a wide geographic distribution throughout much of Southeast Asia, Melanesia and Polynesia, has established a foothold in Africa, India, the Caribbean and most recently the southeastern USA, and is extending its range in eastern Australia[1] *(Stokes and Spratt)*. The life cycle of what was thought to be *A. cantonensis* was studied in Brisbane at a time when it was not generally known to be a wildlife or human pathogen[4]. Many years later it was demonstrated that two species of *Angiostrongylus* occurred in rats in Brisbane and that the original life cycle studies were not of *A. cantonensis* but rather of a new but closely related species, *A. mackerrasae*[5,6].

*Angiostrongylus mackerrasae* occurs in the pulmonary arteries and more rarely the right side of the heart of Australian native bush rats in Queensland and swamp rats in Tasmania. *Angiostrongylus cantonensis* occurs in introduced black rats and Norway rats and co-occurs with *A. mackerrasae* in Norway rats in Brisbane[5,6]. *Angiostrongylus cantonensis* was not found in individual black rats at Glenreagh (near Grafton) in 1994, Hoskinstown in 1977, Milton in 1977, Buckenboura State Forest (near Mogo) in 1986 and Broulee in 1980. Neither species of *Angiostrongylus* were seen in bush rats, swamp rats nor black rats during parasitological studies in temperate forests 30-45 km south of Eden during 1977 to 1991 *(Spratt)*. However, *A. cantonensis* has been detected increasingly in Sydney[2,3] and in 2004 was found in black rats as far south as Jervis Bay *(Stokes and Spratt)*.

*Angiostrongylus cantonensis* undergoes an obligatory migration through parts of the central nervous system and brain en route to its definitive site in the pulmonary arteries and right heart of many species of rats[3,5]. Eggs are deposited into the blood and lodge as emboli in smaller vessels of the lungs, where they embryonate. Hatched larvae are passed up the bronchial escalator, swallowed and passed out in the faeces. Terrestrial, aquatic and amphibious gastropods (snails, slugs and semi-slugs) foraging on rat faeces containing first-stage larvae serve as intermediate hosts, in which there is obligatory development from late first to third stage larva[3,5]. A range of other animals serve as paratenic or 'transport' hosts (land planaria, crabs, frogs, toads, fresh-water shrimp and fish, marine fish and sea snakes) in which no further development of larvae occurs[7-10]. Animals and humans become infected through ingestion of intermediate or paratenic hosts. In Australia, humans are likely to acquire infection by ingesting tissues of raw[11] or undercooked intermediate or paratenic hosts. This is likely to occur most frequently by the ingestion of salad materials, especially lettuce, contaminated with small gastropods or their slime containing infective, third-stage larvae which have

Figure 1. Tawny frogmouth infected with *Angiostrongylus cantonensis* and unable to right itself.

escaped in this secretion [12]. The Australian situation contrasts with that seen in some islands of the Pacific, in Taiwan and in Thailand where the usual causes of human infection have been traced to the consumption of uncooked molluscs, shrimp and crabs, or sauces prepared from them [1].

The first observation of young adult nematodes in the cerebrospinal fluid of a patient with symptoms of meningitis remained mostly unknown until 1964, having been published in a medical journal of limited circulation in Japanese-occupied Taiwan during WWII [13]. Eosinophilic meningitis was reported in an 11 month old girl in Brisbane with clinical signs of serious brain damage, persistent blindness, mental retardation, spasticity and epilepsy who died in an institution for severely handicapped children more than three years later [14].

The family lived on a four ha property with a stream at the front gate, where bottled milk was delivered at night. Each morning this was brought into the house by one of the older children and the bottles left standing for variable periods on the kitchen floor where the youngster often played. Slugs were sometimes seen on the bottles and when animals were examined from the property. *A. cantonensis* was found in 1 of 3 rats, 0 of 9 slugs and in a land planarian that was found at the base of the fence post where the milk bottles were placed.

In Australia, nematodes have been available for specific identification only in the two most recent human cases; both involved young children and in both instances large numbers of *A. cantonensis* were recovered from the pulmonary arteries [15, 16].

Neuroangiostrongyliasis remains a life-threatening disease of wildlife and youngsters wherever introduced black and Norway rats proliferate in cities, around fauna parks and zoos and, increasingly for the black rat, in temperate coastal forest areas and campgrounds where a spectrum of intermediate and paratenic hosts exist.

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