Spiked Awl Snail
***Allopeas clavulinum* (Potiez & Michaud, 1838)**
(Gastropoda: Subulinidae)

Photo: Nick Lloyd

Species description
The spiked awl snail is a very small (7-11mm long) terrestrial pulmonate gastropod mollusk (air-breathing land snail) in the family Subulinidae.
The body is bright yellow and the shell is almost colourless and translucent.
Dead snails have a pearly shell.

Distribution
Probably native to Asia, where it locally has a pre-human fossil record, but with a wide tropical synanthropic distribution.
In tropical climates, the snail lives in leaf litter and under rocks, logs and debris in gardens, parks, nurseries and wastelands. In cooler climates it survives in heated greenhouses under pots in a tropical greenhouse.
It is known from Mascarene Islands, Australia, Japan and many Pacific islands, In the Cook Islands, Rarotonga, Aitutak and Mangaia.
This species was widely distributed in the Pacific by the early 1900s. The earliest published records are from Hawai‘i and Makatea, but collections indicate that it was also present on Rapa, Austral Islands, by 1921 Upolu, Samoa, by 1923; Viti Levu, Fiji, by 1924; Tahiti, Moorea and Borabora, Society Islands, by 1925–26; Rarotonga, Cook Islands, by 1925 and Tongatapu, Tonga, by 1928.
In 2005– 07, *Allopeas clavulinum* was present in forest on Motutapu, and had a scattered distribution in forest remnants and highly modified, open, anthropogenic habitats on the coastal plain of Rarotonga. It was also widely distributed in disturbed forest, slope forest and cloud forest up to 650m elevation in the interior of the island
They also occur in Great Britain, Czeck Republic as greenhouse aliens

Interception NZ 2014
May 2014
Late in 2014 the exotic pest (Spiked awl snail) in a heated greenhouse in Auckland has been detected. A population assessment survey was carried out and no live snails were found. A number of shells were located but also confirmed as being dead. Molluscicides have been used in the vicinity of the original detection.
December 2014
In December a Spiked Awl Snail *(Allopeas clavulinum*) in a heated glasshouse in Auckland has been detected. All sites have come up negative but MPI had still to look at the one known population in Auckland.
See: http://nzppi.co.nz/news/4-385/spiked-awl-snail

Health Risk
The Spiked Awl Snail is a vector for parasitic helminthes such as the rat lung worm *Angiostrongylus cantonensis*.

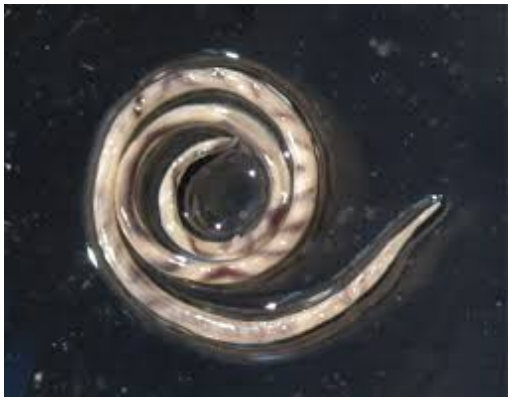
Snails are the primary intermediate hosts, where larvae develop until they are infective. The nematode commonly resides in the pulmonary arteries of rats, giving it the nickname the rat lungworm

Humans are incidental hosts of this roundworm, and may become infected through ingestion of larvae in raw or undercooked snails from contaminated water and vegetables. The parasite has a high infection rate spreading through an association with poorly washed home-grown crops, such as lettuce.

The larvae are then transported via the blood to the central nervous system (CNS), where they are the most common cause of eosinophilic meningitis, a serious condition that can lead to death or permanent brain and nerve damage.

Eosinophilic meningitis is an infection of increasing public health importance as globalization contributes to the geographic spread of the disease. 1961infected brains confirmed *A. cantonensis* infection in humans as the cause of the majority of eosinophilic meningitis cases in Southeast Asia and the Pacific Islands.Since then, cases of *A. cantonensis* infestations have appeared in American Samoa, Australia, Hong Kong, Bombay, Fiji, Hawaii, Honshu, India, Kyushu, New Britain, Okinawa, Ryukyu Islands, Western Samoa and most recently mainland China. Other sporadic occurrences of the parasite in its rat hosts have been reported in Cuba, Egypt, Louisiana, Madagascar, Nigeria, New Orleans and Puerto Rico. *Angiostrongylus cantonensis* and eosinophilic meningitis are already established in Fiji.

Angiostrongylus cantonensis



http://www.ym.edu.tw/par/html/ParPic/Helminthes/Nematode/Angiostrongylus_cantonensis/Ang-can-AduFem2.htm

Distribution

Angiostrongylus cantonensis was described from southern China in 1935. It was reported from Taiwan in 1937 and subsequently from other parts of Southeast Asia (Thailand, Malaysia). It probably originated somewhere in this region. Also by the 1960s, it had been reported from numerous Pacific islands including New Caledonia, Vanuatu, Fiji, Guam, Saipan, Chuuk, Pohnpei, Marshall Islands, Tahiti, Cook Islands, and Hawai‘i.

Reports of cases of eosinophilic meningitis in many of these islands probably reflected the spread of *A. cantonensis.* It has been recorded widely, including in Okinawa and mainland Japan, Papua New Guinea,American Samoa, Indonesia, the Philippines, Australia, Sri Lanka, India, Réunion, Mauritiuis, Ivory Coast, Egypt, South Africa, Madagascar, Cuba, Jamaica, Puerto Rico, Haiti, Dominican Republic, Ecuador, Brazil, the Canary Islands, and the southeastern USA;

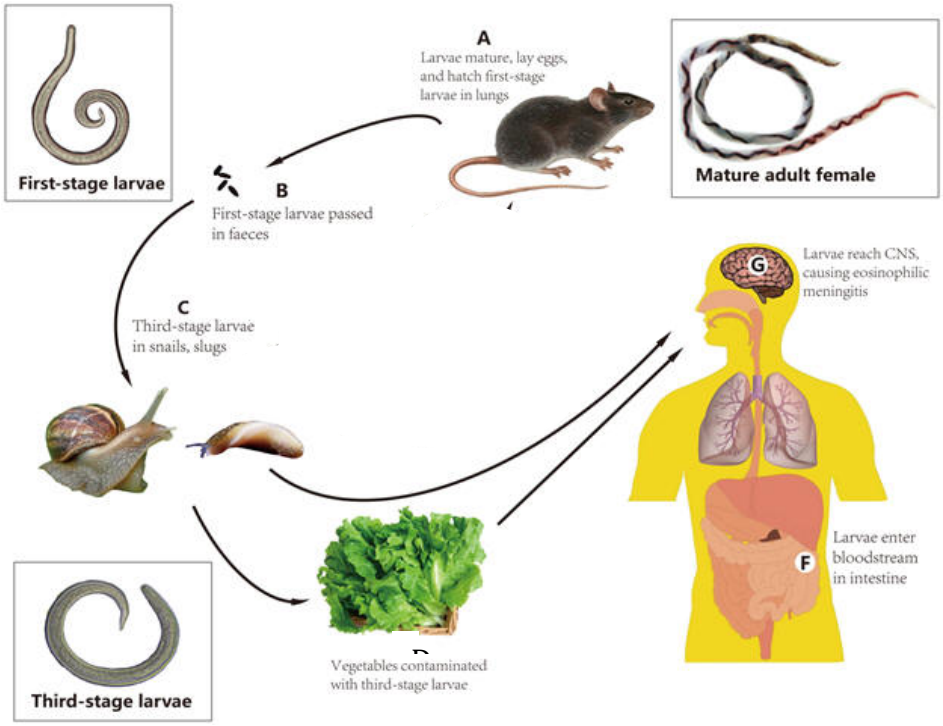
It has the potential to expand its range beyond the tropics, facilitated by climate change, as shown in China.

There are increasing numbers of cases recorded in locations where *A. cantonensis* is not present (eg, various European countries and northern USA), mostly in people returning from regions where it is present.

The giant African land snail, *Achatina* fulica is also a good host of *A. cantonensis.*

Life Cycle

First stage larval worms are expelled in the feces of rats (the definitive host). Various species of rats can act as hosts. These infected feces are ingested by snails or slugs (intermediate hosts), but it may also be possible that the larvae enter the snail by penetrating the body wall or via the respiratory pore. Many species of snails and slugs can act as intermediate hosts. The larvae develop to the third larval stage in the snails, remaining at that stage until either the snail is eaten or dies. Once snails carrying third stage larvae are eaten by a rat, they move through the rat gut to the small intestine. They then penetrate the walls of the intestine and enter the blood stream. They then travel passively in the blood stream, a proportion of them eventually entering the central nervous system and reaching the brain. Once in the brain the larvae develop to the sub-adult stage. Light infections appear to cause little damage to the brain and no obvious behavioral or other reaction, but heavy infections may cause more serious damage as well as behavioral symptoms.



Adapted from http://www.antimicrobe.org/b028.asp

Having reached the sub-adult stage the worms leave the brain, passing into the venous circulatory system, and thence to the right ventricle of the heart and to the pulmonary arteries. Here the worms grow and mature, mate, and the females lay eggs. An adult female nematode in the pulmonary arteries of infected rats can lay around 15,000 eggs daily. The eggs travel in the blood stream to the lungs (hence the name rat lungworm disease). The eggs hatch into first stage larvae in the tissue of the lungs. Depending on the level of infection, the rat may suffer significant damage to the arteries, caused by the bulk of the adult worms, and to the lungs, caused by inflammatory reactions to the larvae. These first stage larvae then break through the walls of the bronchioles and alveoli, move up the trachea in respiratory secretions, and are swallowed, to be released in the feces. The cycle then repeats when snails ingest these infected feces. It takes approximately 6-8 weeks for an infected rat to start to excrete first-stage larvae after ingesting an infected mollusc.

Eosinophilic meningitis

Symptoms

Initial invasion through the lining of the brain, the meninges, may cause a typical inflammation of the meninges and a classic meningitis picture of headache, stiff neck and often fever. The parasites subsequently invade deeper into the brain tissue, causing specific localizing neurologic symptoms depending on where in the brain parenchyma they migrate. Neurologic findings and symptoms wax and wane as initial damage is done by the physical in-migration of the worms and secondary damage is done by the inflammatory response to the presence of dead and dying worms. This inflammation can lead in the short term to paralysis, bladder dysfunction, visual disturbance and coma and in the long term to permanent nerve damage, mental retardation, nerve damage, permanent brain damage or death.

Treatment

The severity and clinical course of Angiostrongylus disease depends significantly on the ingested load of third-stage larvae,[17] creating great variability from case to case making it difficult to design clinical trials and to judge the effectiveness of treatments. Typical conservative medical management including analgesics and sedatives provide minimal relief for the headaches and hyperesthesias. Removing cerebrospinal fluid at regular three- to seven-day intervals is the only proven method of significantly reducing intracranial pressure and can be used for symptomatic treatment of headaches. This process may be repeated until improvement is shown. Recent studies have shown that treatment with an antihelminthic such as mebendazole or albendazole combined with prednisone or prednisolone can reduce the severity and duration of headaches but have not been shown to improve long-term neurologic outcomes.

Diagnosis

The diagnosis of disease caused by *Angiostrongylus cantonensis* infestation is often difficult and relies heavily on the history of a likely ingestion of a commonly infested host and the presence of typical features of the disease. The presumptive diagnosis is particularly strong when eospinophilic meningoencephalitis can be confirmed. The diagnosis of eosinophilic meningitis can be arrived at through detection of elevated cranial pressure and increased numbers of eosinophils. The diagnosis of the cause of eosinophilic meningitis and the presence of *A. cantonensis* is remarkably more difficult. A spinal tap, or a sample of CSF, must be taken to search for *A. cantonensis* worms or larvae. *A. cantonensis* is undetectable in the CSF of more than half of the infected individuals. Current methods of detecting specific antigens associated with *A. cantonensis* are also unreliable. Consequently, alternative approaches to detect antigen-antibody reactions are being explored, such as Immuno-PCR.

Incubation Period

The incubation period in humans is usually from 1 week to 1 month after infection, and can be as long as 47 days. This interval varies, since humans are intermediate hosts and, the life cycle does not continue predictably as it would in a rat.

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