The majority of parasitic worms belong to either the Platyhelminthes (flatworms/flukes and tape-worms) or Nematoda (roundworms/nematodes). Unlike earthworms, nematodes are un-segmented, with a flexible but resilient outer cuticle and more primitive internal organ systems. Most species are free-living but some have evolved as parasites of plants or animals.

The life cycle of nematode parasites of animals is either direct involving a definitive host or indirect with both a definitive host and intermediate host. Infection of the definitive host frequently involves a complex migratory phase. The intermediate host is where early development of the parasite occurs— the life cycle can only be completed when the definitive host consumes the intermediate host (in part or whole). The definitive host is where the parasite becomes capable of reproducing (eggs produced after male worm inseminates female).

In some instances the life cycle can involve paratenic hosts— these consume the infected intermediate host but no further development of the parasite occurs. Development can resume when the paratenic host is consumed by the definitive host. In an abnormal definitive host development is usually incomplete resulting in a failure of the parasite to reproduce and complete the life cycle (a dead-end host).

Nematode parasites with a direct life cycle include those commonly found in the gastro-intestinal tract. Blood and tissue nematode parasites have an indirect life cycle that requires an invertebrate intermediate host. The rat lungworm, which falls into this latter group, also involves several paratenic and dead-end hosts.

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Intermediate hosts – a wide variety of **molluscs** including species of land and freshwater snails as well as slugs. First stage larvae can survive in rat feces for several weeks under moist conditions before being consumed by their mollusk host (or they may actively penetrate the mantle). Within the intermediate host they molt twice to produce third stage larvae - this is the stage that infects the definitive host.

Paratenic hosts – several invertebrates can act as paratenic hosts (see text box above) including frogs, land crabs, freshwater shrimps as well as slugs, other snails (especially predatory snails) and predatory flatworms (Turbellaria).

Migration within the definitive host: Definitive hosts (rodents) acquire third stage larva on consuming infected snails or paratenic hosts (in whole or in part). Food items contaminated with slime, which has been reported to contain third stage larvae, could present another potential means of transmission. Once ingested, third stage larvae penetrate the small intestine and migrate via the circulatory system to the central nervous system (CNS). Development occurs to fourth stage larvae then immature adults in blood capillaries within the meninges (the three layers that cover the brain and spinal cord). Immature adults then migrate via the venous system to the heart and finally to the pulmonary artery where mature adult worms mate and female worms release eggs into the bloodstream.

Human Infection: *A. cantonensis* is one of two *Angiostrongylus* spp. causing disease in humans (referred to as **angiostrongyliasis**) and is the leading cause of **eosinophilic meningitis** (EM -described below). Worldwide most instances of human infection with *A. cantonensis* are the result of consuming raw or undercooked infected snails (occasional instances of paratenic hosts) or food items contaminated with third stage larvae. In the latter instance this is usually due to the accidental ingestion of a paratenic host present on the item consumed. Contamination of food items with third stage larvae present in slime trails secreted by snails and slugs is often cited as a potential source of infection. However several investigators have cast doubt on the importance of slime as a source of third stage larvae in cases of human infection. In instances
where disease outbreaks have been linked to consumption of contaminated fresh produce (as in salads) it is believed more likely that the cause was failure to remove infected small snails or paratenic hosts (e.g., immature slugs) rather than the presence of slime containing infective larvae.

Other less important modes of transmission have included fresh water, vegetable juice and, in the case of children, playing with infected molluscs or contaminated soil.

Once ingested, third stage larvae penetrate the small intestine and within 2-3 days have migrated to the CNS where they localize within blood vessels of the meninges. There is no final migration to the pulmonary artery as occurs in the rat; instead worms remain in the meninges as larvae/immature adults where they eventually die.

The dying worms elicit a severe inflammatory response involving a type of white blood cell (eosinophil) that is commonly (though not exclusively) associated with infections due to helminth parasites. Eosinophils possess cytotoxic granules that are released (degranulation) in an effort to kill the invading parasite. However where a response stimulates the production of large numbers of eosinophils, the released cytotoxins can also prove injurious to surrounding host tissue. This is believed to account for most of the CNS damage that occurs; physical activity of the worms while migrating within the meninges, and toxicity of products released by dying worms are also probable contributary factors.

**Symptoms** – The pre-patent period (before symptoms first appear) can be from 2 to 45 days after ingestion of third stage larvae. The most consistent feature is a persistent, severe, frontal or occipital headache believed due to increased intra-cranial pressure resulting from the pronounced inflammatory response to the worms (see above). One or more other, less common symptoms have also been described including: paresthesias (tingling, prickling or burning sensations), hyperesthesias (decreased tactile sensitivity), blurred vision, stiff neck, vomiting, mild fever and paralysis of facial muscles.

In most instances symptoms spontaneously resolve within 35 days with no long term problems, infection rarely being fatal. Sometimes more serious complications occur with fever and encephalitis (more so in children) while in 2009 one case in Hawaii involved prolonged coma and several others have experienced residual cognitive and visual impairment. Extent of the severity of symptoms is dictated by the magnitude of the inflammatory response which in turn depends on the number of larvae ingested and/or the rate at which they die and disintegrate. Treatment is supportive; anthelmintics (drugs active against helminth parasites) can worsen symptoms by increasing the rate at which worms die and disintegrate. Easing of symptoms has been reported after corticosteroids were administered to reduce the inflammatory response.
Angiostrongylus cantonensis in the USA  All but one of the cases of *A. cantonensis* infection diagnosed within the continental USA were non-autochthonous (i.e., contracted at a location outside the continental states).

In Louisiana one autochthonous case involved a boy who swallowed an infected snail on a dare. A subsequent Louisiana case of EM involved a young adult who had previously swallowed frog eggs (possible paratenic host). While non-parasitic causes were ruled out, and *A. cantonensis* seems the probable cause, no firm conclusions are presented in this second case.

*Angiostrongylus cantonensis* is widespread in both Louisiana rodents and molluscs and is believed to have been introduced via infected rats from ships docking in New Orleans rather than infected molluscs.

In Hawaii the first autochthonous cases of infection with *A. cantonensis* were reported in the 1960’s. Since then EM due to the parasite has been rare though several cases since 2004 have increased awareness of infection as an important public health issue. Spread of the parasite has undoubtedly been facilitated by the presence of both the apple snail *Pomacea canaliculata* and the giant African land snail *Achatina fulica* (both highly effective in disseminating third stage larvae). In addition several other species of land/freshwater snails and slugs are present that can act as intermediate/paratenic hosts. In some parts of Hawaii * Parmarion martensi* (yellow-shelled semi-slug) an accidental introduction from SE Asia in 2004 is seen as more important than other molluscs in transmitting *A. cantonensis*, 74% of all specimens examined being positive for lungworm larvae.

Angiostrongyliasis - is there need for concern locally?  A case of eosinophilic meningitis was reported involving a primate (gibbon) at Miami Metro Zoo, with worms recovered at autopsy identified as *A. cantonensis*. It is not known for certain whether the source of the third stage larvae was from contaminated feed brought in from another country or locally infected molluscs. The source of the feed was from areas where *A. cantonensis* was believed to be absent. If already present the parasite could have been introduced to Miami-Dade during a previous invasion (eradicated in 1975) of *A. fulica*, or rats via local shipping. Rat lungworm is found widely in Caribbean locales – Puerto Rico, Cuba, Haiti, Dominican Republic, Jamaica and Bahamas. If previously introduced to Miami-Dade, there are both rodent (rats) and mollusc hosts present that could have maintained the life cycle. For instance the Asian tramp snail *Bradybeana similaris* is widespread in Florida and a known intermediate host for *A. cantonensis* (Pacific basin as well as Cuba and more recently Brazil).

In Miami-Dade rat lungworm larvae have recently (fall 2012) been found in a few of the locally sampled giant African land snails. Previously samples of the apple snail *Marisa cornuarietis* taken from around Miami Metro Zoo were negative for *A. cantonensis*. Apple snails especially *P. canaliculata* (which is not as yet found in Florida) along with *A. fulica* are important intermediate hosts of rat lungworm in regions of the world where the parasite has become a public health concern. To date infected rats have not been reported; bearing in mind
that snails become infected by first stage larva eliminated in rat feces it is logical to assume lungworm is present in the local rat population.

In assessing where conditions within the continental US most favored establishment of giant African land snails, extreme S.E Florida was the only area that that came close to satisfying year round climate conditions. Even here, a dry season of several months and temperatures that on occasion can approach freezing during winter would be expected to induce a period of aestivation which would limit the snail’s potential to disseminate infective rat lung worm larvae. Optimum conditions for the giant African land snail include a year round humid, warm to hot climate (as in areas of Hawaii). Although local conditions may not be optimum for year round proliferation of A. fulica, the concern is that their numbers will escalate sufficiently to increase the incidence of A. cantonensis in the local rat population. This in turn could increase the incidence of rat lungworm in other susceptible intermediate and paratenic hosts, ultimately heightening the risk of human infection. This is one reason why the intensive ongoing program to eradicate A. fulica from Miami-Dade is so important.

It is clear that while A. fulica can and has played an important role in the spread of rat lungworm it is not essential as other intermediate hosts are proving to be as effective (apple snails and some slugs). Compare the role of A. fulica in the spread of rat lungworm in Brazil and Jamaica (see text box at left) where in the first case it has been a factor but of no importance in the latter.

The presence of a large infected urban rodent population is also crucial (as in Brazil) and effective programs of rat as well as snail/slug control are equally important in limiting human infection with rat lungworm.

A third factor is local dietary habits. In those parts of the world where human infection with rat lungworm is most prevalent (S.E. Asia and Pacific basin) snails (both raw and undercooked) are not uncommon as food items. The fact that snails are rarely consumed in most of the Caribbean is thought to be one reason the region has not experienced more cases of human infection. Those instances where human infection has been found, as in Jamaica, were due to accidental consumption of intermediate/paratenic hosts (e.g., small snails or slugs) present on food items such as garden produce. This would be expected to be most likely means of transmission in Miami-Dade.

What to do to avoid being infected with rat lungworm?
Human infection with rat lungworm is rare and recovery is usually complete, but there can on occasion be serious long term neurological problems
Angiostrongylus costariciensis is a small roundworm parasite of rodents, notably the cotton rat, adult worms being located in the mesenteric arteries of the lower portions of the small intestine. It is here that eggs are laid and hatch, with the released first stage larval worms then penetrating the wall of the intestine to be subsequently eliminated with the stools. Further development requires the larval worms to be eaten by an intermediate host, usually a veronicellid slug but some snails too can be infected.

Human Infection

Unlike A. cantonensis, A. costariciensis develop to mature egg laying adults in humans. Eggs and first stage larvae induce severe local inflammatory reactions, leading to the formation of large granulomatous masses in the intestinal wall, and symptoms that mimic appendicitis (abdominal angiostrongyliasis). In some instances intestinal blockage, perforation or bleeding occur. Because of this pronounced host response, first stage larvae are unable to break out into the gut and the thus humans play no role in spreading the disease, being a dead end host. Disease outbreaks in humans are usually associated with eating fresh vegetables contaminated with infective third stage larvae secreted by feeding slugs/snails. Most human cases are found from Central America into South America with a few in the Caribbean. No autochthonous cases reported in the US.

Baylisascaris procyonis is an intestinal roundworm parasite of raccoons that can cause a rare but often fatal meningo-encephalitis, especially in infants. Human cases in the US have
been restricted to the upper Midwest, Pacific North West and some states in the north east. There, the parasite is far more prevalent though it has recently been recorded in Florida raccoons.

Source Material and Further Reading
(All hyperlinks last accessed October 2012)


associated with rat lungworm infection and human eosinophilic meningitis (University of Hawaii CTAHR Publication FST-39).


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