



THE GIANT AFRICAN LAND SNAIL: what you need to know about the rat lungworm, molluscs and human disease

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The rat lungworm *Angiostrongylus (=Parastrongylus) cantonensis* belongs to a group of parasitic worms commonly referred to as round worms (see text box at right) and has an indirect life cycle (see figure below) with rodents and molluscs respectively acting as definitive and intermediate hosts. Human infection can also occur. The exotic giant African land snail has been found at several sites in Miami-Dade. Not only is it a serious agricultural pest, but of public health concern as a highly effective intermediate host of rat lungworm.

Definitive host: adult worms are slender up to 1” in length and found in **rodents**, most often *Rattus* spp. especially the **Norway rat** (*R. norvegicus*) and the **roof rat** (*R. rattus*) where they localize in the pulmonary artery. Female worms release eggs into the bloodstream which are then carried to the lungs, become lodged in blood capillaries and hatch to produce first stage larvae. The larvae break through into fine air passages, progressing to the trachea where they are swept up into the pharynx and swallowed. They then pass through the gastro-intestinal tract and are expelled when the rat defecates.

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**).

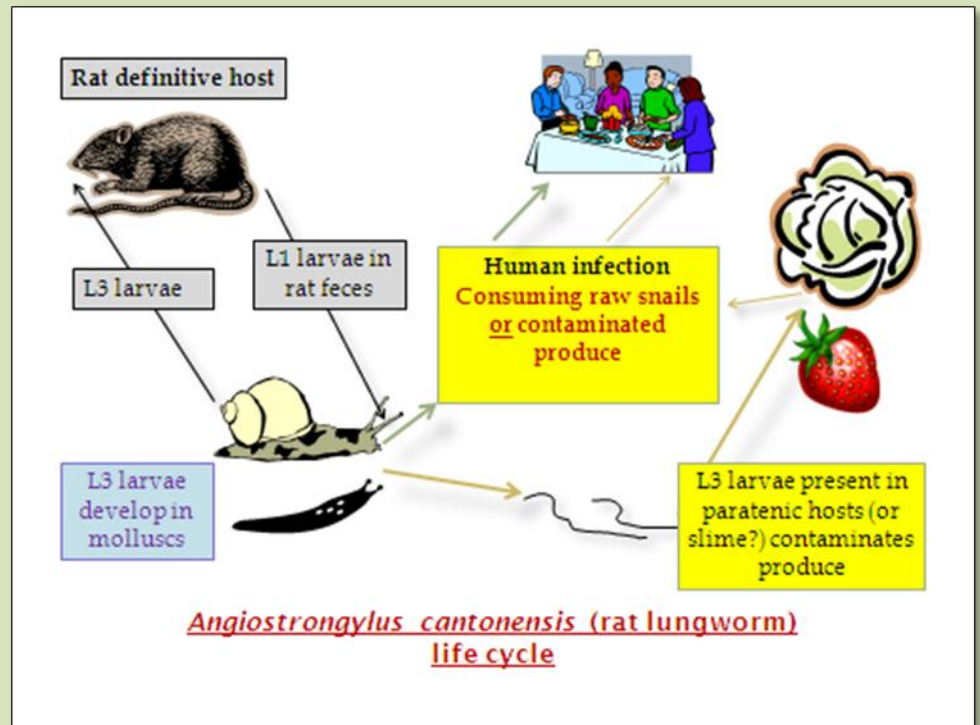
Nematode parasites with a direct life cycle include those commonly found in the gastro-intestinal tract. Blood and tissue nematode parasites have an indirect life cycle that requires an invertebrate intermediate host. The rat lungworm, which falls into this latter group, also involves several paratenic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

Diagnosing angiostrongylus as the cause of eosinophilic meningitis is not easy. Worms are rarely recovered and it can be difficult to detect an increase in eosinophils during early phases of the infection. Immunological methods can be useful, but the antigen used is not widely available.

A positive diagnosis is usually based on patient symptomatology (severe headache, paresthesia etc), eosinophilia, and a previous history that includes consumption of molluscs either deliberately or accidentally (e.g., garden produce that could have been contaminated with small snails or slugs).

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 contributory 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

In Brazil human disease due to *A. cantonensis* is of recent occurrence the first case having only been diagnosed in 2007. Prior to this, laboratory studies had suggested that in Brazil *A. fulica* is not especially susceptible to *A. cantonensis* and the snail would not pose a future problem in spreading rat lungworm. However human infection with rat lungworm is becoming more of a concern in Brazil, coinciding with a rapidly mounting problem with *A. fulica* as an invasive pest (and which along with other snails has been found to act as an intermediate host).

In Jamaica giant African snails have not been identified but rat lungworm is present and has featured in several outbreaks of human disease. Jamaica has a rich diversity of molluscs several of which are effective intermediate hosts. In Cuba, where the first case of human infection with rat lungworm was reported 30 years ago, *A. fulica* is not known but several other molluscs especially the slug *Veronicella cubensis* are effective intermediate hosts.

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

◆ The most obvious precaution is not to consume raw or undercooked snails. If you need to handle snails wear gloves, especially when removing dying snails (e.g., after using snail bait) which are more likely to be shedding infective third stage larvae.

◆ Do not allow children to play with either snails or paratenic hosts such as frogs, and closely supervise them when helping in the garden especially if the yard has been infested with snails.

◆ A review of recent *A. cantonensis* cases in Hawaii found that most involved consumption of uncooked produce from home gardens or other non-commercial sources. There are no specific methods for rendering garden produce free of slugs or snails; be cognizant of the small size of some immature slugs and snails (as small as 2mm) especially when washing leafy salad greens and strawberries. You should already be washing home produce to reduce potential contamination from pathogenic bacteria – e.g. *Salmonella* from animal droppings, especially birds and reptiles.

◆ If you encounter giant African snails on your property contact the FDACS hotline at 1-888-397-11517 and [use appropriate snail bait as a control](#).

***Angiostrongylus costaricensis* is another rodent parasite that can infect humans** and it has been found in zoo borne primates and wild raccoons and opossums in Miami-Dade. Unlike *A. cantonensis* the intermediate hosts are usually slugs and the adult worms are found in the blood vessels of the small intestine of the rodent definitive host (see text box above right for more information).

There are other causes of EM apart from *A. cantonensis*: *Gnathostoma* spp. are roundworm parasites found in the intestinal wall of mammals such as cats, dogs and pigs; they utilize two intermediate hosts (a copepod then a frog or fish) and several paratenic hosts including birds. Humans usually acquire the parasite by consuming raw or insufficiently cooked freshwater fish and to a lesser extent game birds. The parasite is endemic in SE Asia where most cases of human infection occur; it is found in scattered locations from Mexico into S. America. It is extremely rare in the US (no known autochthonous cases).

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

Also present in Miami-Dade.....

Angiostrongylus costaricensis 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. costaricensis* 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 deposited in the mucus 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.

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.

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