Wild Rodents as Experimental Intermediate Hosts of 
*Lagochilascaris minor* Leiper, 1909

Julieta Machado Paçô, Dulcinéa Maria Barbosa Campos*,
Jayrson Araújo de Oliveira

Instituto de Patologia Tropical e Saúde Pública, Universidade Federal de Goiás, Setor Universitário,
Caixa Postal 131, 74605-050 Goiânia, GO, Brasil

A total of 25 specimens of *Cavia porcellus* (guinea pig), 5 *Dasyprocta agouti* (agouti), and 22 *Calomys callosus* (vesper mice) were inoculated with infective eggs of *Lagochilascaris minor*. The inoculum was prepared with embryonated eggs and orally administered to each individual animal through an esophagus probe. In parallel, 100 specimens of *Felis catus domesticus* were individually fed with 55-70 nodules containing 3rd-stage larvae encysted in tissues of infected rodents. Animals were examined and necropsied at different time intervals. The migration and encystment of L3 larva was observed in viscera, skeletal muscle, adipose and subcutaneous tissues from all rodents. Adult worms localized at abscesses in the cervical region, rhino, and oropharynx were recovered from domestic cats inoculated with infected rodent tissues. Through this study we can conclude that: (1) wild rodents act as intermediate hosts, characterizing this ascariid heteroxenic cycle; (2) in natural conditions rodents could possibly act as either intermediate hosts or paratenic hosts of *Lagochilascaris minor*; (3) despite the occurrence of an auto-infecting cycle, in prime-infection of felines (definite hosts) the cycle is only completed when intermediate hosts are provided; and (4) in the wild, rodents could serve as a source of infection for humans as they are frequently used as food in regions with the highest incidence of human lagochilascariasis.

**Key words:** *Lagochilascaris minor* - heteroxenic cycle - intermediate host - paratenic host

The genus *Lagochilascaris* was described after specimens obtained from subcutaneous lesions of two patients in Trinidad. This nematode has both peculiar morphology and habits. To date, five species have been recognized in this genus: *L. minor* Leiper, 1909; *L. major* Leiper, 1910; *L. turgida* (Stossich, 1902) Travassos, 1924; *L. sprenti* (Bowman et al. 1983) and *L. buckleyi* Sprent, 1971.

Human lagochilascariasis is caused by *L. minor* and it is considered an emerging helminthiasis limited to the American continent. The parasitosis is not yet a public health problem. Nevertheless, it is prevalent in individuals of the lowest socioeconomic class, notably from rural areas. *Lagochilascaris* pathogenicity varies from mild manifestations to more severe forms, involving the central nervous system, which in some cases may lead to death (Rosemberg et al. 1986, Orihuela et al. 1987). The parasite is frequently found in tumoral lesions at the cervical region and neighboring tissues. Human *L. minor* lesions have been reported in the mastoid, tonsils, eyeball, neck, nasal sinuses, middle ear, central nervous system, lungs, rhino pharynx, dental alveolus, cervical region, and sacral region (Fraiha et al. 1989, Bento et al. 1993). *Lagochilascaris* lesions contain different stages of the parasite which indicates potential auto-infection and favours the development of chronic disease (Campos et al. 1983, Moraes et al. 1985).

Reported human infections have neotropical distribution in countries such as Mexico, Costa Rica, Trinidad, Tobago, Colombia, Venezuela, Suriname, Bolivia and Brazil (Olle-Goig et al. 1996, Vargas-Ocampo & Alvarado-Aleman 1997, Paçô & Campos 1998). Moreover, Brazilian Amazon currently represents an important focus of the helminthiasis (Fraiha et al. 1989). It is suggested that the Brazilian Amazon, especially the region between Tocantins and Araguaia rivers, presents the best ecological resources for *Lagochilascaris* development.

*L. minor* infection through ingestion of either uncooked or lightly cooked meat containing encysted larvae was hypothesised by Smith et al. (1983) and later confirmed by Campos et al. (1992). As yet, natural hosts for *L. minor* are unknown. However, it seems reasonable to believe that such a host exists because most patients infected with

---

*Corresponding author. Fax: +55-62-202.3066. E-mail: dmcampos@ufg.br
Received 10 July 1998
Accepted 18 March 1999*
L. minor come from woody areas. In general, infected individuals in small newly deforested areas work upon the land for subsistence and feed almost exclusively upon hunted meat (Campos et al. 1991, Paçô 1994).

Taking into consideration the feeding habits of patients with lagochilascariasis and experimental data that demonstrate the heteroxenic cycle for the helminth, we decided to investigate the potential susceptibility of some wild rodent species, frequently used as food source in regions of high prevalence of L. minor infection. In our study wild rodents were inoculated with infecting eggs of L. minor while domestic cats were fed carcasses of infected rodents.

MATERIALS AND METHODS

Parasite isolate - We used the HGS isolate of L. minor kept at the Instituto de Patologia Tropical e Saúde Pública, Universidade Federal de Goiás (IPTSP, UFG) throughout the experimental system; mouse (Mus musculus) as the intermediate host and domestic cat (Felis catus domesticus) as the definitive host.

Animals - We used a total of 25 specimens of Cavia porcellus (guinea pig), 5 specimens of Dasyprocta agouti (agouti), and 22 specimens of Calomys callosus (vesper mice). All animals were adults and of both sexes. One male specimen of D. agouti was captured at the Paraupebas municipality and the others were donated by the local Zoo. Animals were kept in captivity for three months undergoing treatment with albendazole to eradicate any intestinal and tissue parasites. C. porcellus and C. callosus specimens were obtained locally at the IPTSP, UFG. The study proposal using wild rodents was authorized by Instituto Brasileiro do Meio Ambiente (Ibama).

One hundred and thirty specimens of stray domestic cats (F. c. domesticus) were obtained from several districts in Goiânia with ages ranging from 3 to 18 months. Felids were divided in two groups, namely G1 and G2 comprised of 100 and 30 animals, respectively.

Control group - For this study we used 125 inbred mice C57BL/6 from both sexes at approximately three months old as well as 30 specimens of F. c. domesticus (G2 group above).

Inoculum - The inoculum consisted of uterine eggs obtained by dissection of L. minor adult female recovered from experimentally infected F. c. domesticus. Adults females were recovered from experimentally infected F. c. domesticus. Eggs were kept in 1% formaldehyde solution at room temperature for 30-40 days. During this time, 3rd-stage larva developed, resulting in infecting eggs. The embryonated egg suspension was centrifuged at 3,500 rpm for 2 min. Three equal amounts of 5 ml were withdrawn and the eggs were counted under the microscope to determine the concentration of eggs per ml.

Experimental inoculation - Each individual specimen of D. agouti and C. porcellus was infected orally with 5,000 infecting eggs of L. minor through an esophagus probe. A similar procedure was used to infect C. callosus and C57BL/6 mice. However, for the small rodents the inoculum contained only 1,500 infecting eggs. Felids from the G1 group were not fed for 24 hr prior to inoculation. Each animal was individually fed 55-70 3rd-stage larva encysted in carcasses of C. porcellus, D. agouti, and C. callosus. Felids from the G2 group acting as a control group were similarly infected using mouse carcasses instead.

Necropsies - All animals were killed by vapour inhalation of ether/chloroform. Animal tissues from the digestive and respiratory tracts, circulatory, urinary, and genital systems, brain, and skeletal muscles were scrutinized macroscopically and microscopically in order to determine the presence of parasites. In order to identify both adult and larval stages, individual tissue was submitted to different methodologies such as: tissue compression between glass microscopic slides; histological preparations stained with hematoxin-eosin; and finally Baermann-Moraes method.

In order to observe larval migration and encystment, inoculated animals were necropsied at different time intervals postinfection. Specimens of C. porcellus, C. callosus, and C57BL/6 mice were necropsied daily and examined at 6, 12, and 24 hr from the 2nd to 7th day at different intervals until the 241st day after inoculation (DAI). Adult D. agouti were necropsied and examined at 60, 95, 96, 152, and 276 DAI. Cats from both groups G1 and G2 were necropsied daily and examined at 6, 12, and 24 hr from the 2nd to the 9th day at different intervals until the 235th DAI.

Coproscopic analysis - In order to follow the progress of the inoculum through the digestive tract of the animal, coproscopic analysis were carried out 96 hr post-inoculation using the methodology of Baermann-Moraes and Lutz.

RESULTS

All the wild rodents and experimentally inoculated felids exhibited infection by L. minor. Among specimens of C. porcellus and C. callosus we observed hatching of 3rd-stage larva (L3) and the presence of eggs of L. minor at 6-12 hr post-inoculation in the small intestine. L3 migration to liver and lung was observed at 24 hr post-inoculation while migration to skeletal muscles and subcutaneous tissues was detected 13 DAI. Granulo-
matous nodules containing encysted 3rd-stage larva were found 38 DAI at the subcutaneous and adipose tissues, skeletal muscles, lungs, liver, tonsils, eyeballs, and mesentery in both experimentally inoculated *C. porcellus* and *C. callosus*. In addition, in these animals some larger nodules containing only one adult worm were observed. The adult worms were immersed in a viscous and purulent secretion which contained the products of an intense granulomatous reaction (Fig. 1). Egg shells and whole embryonated eggs of *L. minor* were detected in faeces of *C. porcellus* and *C. callosus* at 72 hr post-inoculation.

All five specimens of *D. agouti* necropsied between 60-276 DAI exhibited several small nodules which contained encysted L3 larva surrounded by a granulomatous reaction. In *D. agouti* there was visible nodule dissemination through the host tissues, mainly subcutaneous, muscular, adipose tissues, as well as throughout the lungs and mesenteries. Neither larger nodules nor adult worms were observed in these five animals (Figs 2, 3). Only egg shells were found in faeces of all five agoutis which were examined up to 96 hr after inoculation.

Among C57Bl/6 mice that comprised the control group we observed hatching of 3rd-stage larva in their small intestine at 4-6 hr post-inoculation. Larval migration to the liver and lungs were observed 6-24 hr post-inoculation. Larval concentration at subcutaneous tissue and skeletal muscle was noticed eight DAI, although encystment was evident only around the 30th DAI.

Migration patterns and 3rd-stage larvae encystment were similar in all the wild rodents from our study and appeared similar to the infection observed in mice. All 100 cats in the G1 group, which were inoculated with encysted larva in carcasses of *C. porcellus*, *D. agouti*, and *C. callosus*, exhibited 3rd-stage (L3) larvae in their stomach at 6 hr post-inoculation. Larva migrated directly from the stomach to the upper portions of the digestive tract, where they developed into 4th-stage (L4) larvae, finally reaching maturity around the 12th DAI. L3 and L4 larva, and adult worms were retrieved from tissues of the pharynx, trachea, mastoid, ears, nasal sinuses, oropharynx, soft palate, tonsils, tongue, lung, eyeball, central nervous system, and cervical and sub-mandibular regions.

In recent infections, 4th-stage larva (L4) and adult parasites were found free in the sites mentioned in the above paragraph. In late infections, from the 18th DAI, L4 bored through the tissues and developed in tumoral lesions, generally with fistula and a large quantity of purulent secretion, which most of the time contained abundant eggs (Figs 4, 5, 6).

Hemorrhagic discharge was observed in the wall of both esophagus and trachea during larval

---

**Fig. 1**: cross section of a lung of *Calomys callosus* showing 3rd-stage larvae in the center of a granulomatous lesion.
Fig. 2: carcass of *Dasyprocta agouti* examined at 152 days after inoculation showing intense nodule dissemination through the animal tissues.

Fig. 3: each individual nodule contains a 3rd-stage larva of *Lagochilascaris minor* which were encysted in tissues of *Dasyprocta agouti*. 
Fig. 4: cervical lesion from a cat inoculated with 55 larva of *Lagochilascaris minor* which were encysted in tissues of *Calomys callosus*, examined 35 days after inoculation.

Fig. 5: tonsil lesion from a cat inoculated with 70 larva of *Lagochilascaris minor* which were encysted in tissues of *Calomys callosus*, examined 57 days after inoculation.
migration from the stomach to the upper portions of the digestive tract. Although some of the infected animals seemed healthy, the majority of them exhibited a depauperated physical aspect.

Histopathology analysis revealed sections of tissues with worms held at the center of abscesses, sometimes at different stages of maturity, including intense inflammatory infiltrate, neutrophils, and vascular neo-formation. Additionally, granulomatous abscesses, containing peripheral histiocytes surrounding a necrotic center, were found at both lymph nodes and skin lesions. The frequency distribution of lesion in cats inoculated with larva encysted in tissues of rodents can be found in Table I.

Despite the diversity observed in the frequency distribution of L. minor lesions in infected animals, they were most frequently found in the oropharynx (pharynx, soft palate, and tonsils). In addition, lesions were also observed in the cervical region, ear, mastoid, tongue, nasal sinuses, central nervous system, esophagus, eyeballs, and lungs (Table II).

**TABLE I**

<table>
<thead>
<tr>
<th>Total of lesions</th>
<th>Total of animals</th>
<th>Frequency distribution (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>7</td>
<td>5.4</td>
</tr>
<tr>
<td>1</td>
<td>65</td>
<td>50</td>
</tr>
<tr>
<td>2</td>
<td>43</td>
<td>33</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
<td>11.5</td>
</tr>
<tr>
<td>Total</td>
<td>130</td>
<td>100</td>
</tr>
</tbody>
</table>

**TABLE II**

<table>
<thead>
<tr>
<th>Local</th>
<th>Total of lesions</th>
<th>Frequency distribution (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oropharynx</td>
<td>104</td>
<td>53.1</td>
</tr>
<tr>
<td>Neck</td>
<td>27</td>
<td>13.8</td>
</tr>
<tr>
<td>Inner ear</td>
<td>18</td>
<td>9.2</td>
</tr>
<tr>
<td>Mastoid</td>
<td>13</td>
<td>6.6</td>
</tr>
<tr>
<td>Tongue</td>
<td>12</td>
<td>6.1</td>
</tr>
<tr>
<td>Nasal sinuses</td>
<td>9</td>
<td>4.6</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>5</td>
<td>2.6</td>
</tr>
<tr>
<td>Esophagus</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Eyeballs</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>Lung</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>196</td>
<td>100</td>
</tr>
</tbody>
</table>
Lesions containing eggs in different stages and adult worms were observed in seven cats examined at 43, 55, 61, 69, 100, 176, and 225 days after inoculation, characterizing auto-infection. Larval migration and adult worm development in lesions at the cervical region as well as rhino and oropharynx were similar in all 30 cats from the control group, which received inocula via C57BL/6 mice, and in animals from the G1 group, which were infected with larva encysted in wild rodent tissues.

**DISCUSSION**

With respect to experimental life cycle of *L. minor* data was provided by Campos et al. (1989) who first described the development of 3rd-stage larvae inside eggs. Similar results were registered earlier by different authors while studying the life cycle of other ascarid, including *Ascaris suum*, *Travassacaris*, *Hexametra*, *Ophydascaris*, *Toxo- cara*, and *Ascaris lumbricoïdes* (Araújo 1972). Campos and Freire-Filha (1989) investigating the susceptibility of different mouse strains and hamsters to *L. minor* infection, demonstrated that embryonated eggs containing 3rd-stage larva were indeed the infecting form for this parasite. Subsequent work regarding the experimental life cycle of the ascarid (Campos et al. 1990) including mice as the intermediate hosts and cats as the definitive hosts, demonstrated the heteroxenic nature of the *L. minor* life cycle. Further detailed work by Campos et al. (1992) demonstrated all the life cycle stages of this helminth and registered its auto-infecting cycle. This group also concluded that in a primary infection an intermediate host is required. Volcán and Medrano (1990) and Volcán et al. (1992) reported on an experimental infection of *Dasyprocta leporina* with infecting eggs of *L. minor*, leading to the formation of nodules in skeletal muscles containing encysted 3rd-stage larva. However, adult froms of the parasite were not recovered from the animals.

In this study, all the rodents inoculated with infecting eggs of *L. minor* exhibited 3rd-stage larva encysted in their tissues. Therefore, the rodents act as the intermediate hosts similarly to the infected mice. Among the wild rodents, *D. agouti* was found to be the most susceptible to *Lagochilascaris* infection. We observed that in *D. agouti* the inoculum in the digestive tract was significantly productive because only egg shells were found in their faeces at 96 hr post-inoculation. Furthermore, neither intact embryonated eggs nor free larva were found in the digestive tract. The lack of larger nodules containing adult worms in tissues of *D. agouti* also supports the observation of higher susceptibility of these animals to *L. minor* infection. In all other rodents, including mice faeces contained both embryonated eggs and egg shells up to 72 hr post-inoculation.

We also report on finding nodules containing adult worms in intermediate hosts, such as the ones observed in *C. porcellus, C. callosus* and mice. This finding has been previously reported when using both *L. minor* (Campos et al. 1989, 1990, 1992) and *L. sprenti* (Sprent 1971) in experimental infections.

All 130 domestic cats fed rodent carcasses containing encysted 3rd-stage larva developed *Lagochilascaris* infection. All forms of the helminth were observed. Therefore, the infection in cats confirms the infective nature of encysted L3 larva as well as the existence of a heteroxenic life cycle for this ascarid.

We frequently observed loss of weight in the infected animals, associated with raised hair and loss of appetite. Additionally, infected animals manifested locomotory difficulties, loss of body balance, midriasis, and head hanging to the sides as the most noticeable signs of the infection involving the central nervous system.

No significant differences were found, either clinically or parasitically, in the pattern of infection development in cats from the control group inoculated with infected mouse carcasses and cats inoculated with larva encysted in wild rodent tissues. With respect to felid infection we can conclude that cats acted as definitive hosts because the helminth life cycle was completed very quickly when felids were fed intermediate host tissues containing infective larva of the parasite. None of the felids, our experimental definitive host, exhibited a cardiac-pulmonary cycle. Therefore, it is very likely that the human disease originates from the migration of larva from the stomach to tissues of the pharynx in a way similar to the disease observed in cats.

*L. minor* infection in rodents, as intermediate hosts, was characterized by the encystment of 3rd-stage larva in their tissues. However, in definitive hosts, the infection was characterized by the presence of adult worms inside abscesses with or without fistulas. The occasional finding of adult worms in intermediate hosts and encysted larva in definitive hosts supports the complex and obscure nature of this parasite’s life cycle. In this study we also report the occurrence of an auto-infecting cycle for *L. minor*. In addition we have identified its heteroxenic nature as reported earlier by Campos et al. (1992). It is likely that a component of the digestive tract of carnivores may have a fundamental role on larval destiny at the time of primary infection.

Fraia et al. (1989) reported cases of osteolysis in patients infected with *L. minor*. As in human
disease, in experimental lagochilascariasis the bone tissue offers no barrier to the parasite. There are registers of bone tissue destruction by the helminth even in recent infections. Our results demonstrated that experimental infection in felids with *L. minor* developed in a way similar to that observed in human infections, predominantly with respect to location, capacity of invasion, lesion aspects, auto-infection, and even relapse.

Several author have claimed that the parasite probably has a wild origin (Leiper 1909) based on the fact that most of the patients with lagochilascariasis come from the lowest social-economic class, notably from rural areas (Veloso et al. 1992). Patients very often live very close to woody areas and report frequent ingestion of hunted meat such as guinea pigs and agoutis (Fig. 7).

Humans have been considered as accidental hosts due to the rarity of cases and the exceptional location of lesions. However, to date, the natural host and other important aspects of the biology of this ascarid remain to be determined (Paçô & Campos 1998).

In the present study we noted the complete development of the life cycle of *L. minor*, having wild rodents acting as intermediate hosts and felids as definitive host for the worm. It is possible that animals of agrarian origin may act as potential paratenic or intermediate hosts in the natural life cycle of *L. minor*. Moreover, it is also possible that wild animals may act as a source of infection to humans because these animals are not only susceptible to this parasitic infection, but also, they are frequently used as food in regions with the highest incidence of the human disease.

**ACKNOWLEDGEMENTS**

To the local Zoo, the Jardim Zoológico de Goiânia, for donating specimens of *Dasyprocta agouti*.

**REFERENCES**


---

**Fig. 7:** residence of patient with lagochilascariasis near a woody area in Araguaína, Tocantins, Brazil.


