

## Intestinal Nematodes: A Review.

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### ABSTRACT

A review of intestinal nematodes has been undertaken. The study provides a detailed review of nematodes, taxonomy and systematics, location, adaptation, reproduction, various species, and the general features of nematodes. A study of intestinal nematodes such as *Ascaris lumbricoides*, *Enterobius vermicularis*, *Ancylostoma duodenale*, *Necator americanus*, *Strongyloides stercoralis*, and *Trichuris trichiura* was also carried out. Finally, the life cycle of these intestinal nematodes was examined including their symptoms, diagnosis, prevention and treatments.

(Keywords: *Ascaris lumbricoides*, *Enterobius vermicularis*, *Ancylostoma duodenale*, *Necator americanus*, *Strongyloides stercoralis*, *Trichuris trichiura*, intestinal roundworm, pinworm hookworm, threadworm, whipworm, nematodes, life cycle)

### INTRODUCTION

Nematodes belong to the phylum *nematoda* [1, 2, 3]. The name Nematoda means “the thread-like ones”, from Ancient Greek *nema* and *-ode* which means “thread” and “like” respectively. They are the most diverse phylum of pseudocoelomates, and one of the most diverse of all animals. Nematode species are very difficult to distinguish; over 80,000 have been described, of which over 15,000 are parasitic. It has been estimated that the total number of described and undescribed roundworms might be more than 500,000. Unlike cnidarians or flatworms, roundworms have a digestive system that is like a tube at both ends [2, 4].

The group was originally established by Karl Rudolphi in 1808 under the name Nematodea, but reclassified as family Nematodes by

Burmeister in 1837. They were eventually renamed Nematoda by K.M. Diesing in 1861. Nathan Cobb in 1919 argued that they should be called Nemata or Nematodes, and in English *nemas* rather than *nematodes*, but Diesing’s revision has stuck [2].

Due to a lack of knowledge regarding many nematodes, their systematics is contentious. Traditionally, they are divided into two classes, the Adenophorea and the Secernentea, and initial DNA sequence studies suggested the existence of five clades:

- Dorylaimia
- Enoplia
- Spirurina
- Tylenchina
- Rhabditina

The Secernentea are indeed a natural group of closest relatives but the Adenophorea appear to be a paraphyletic assemblage of roundworms simply retaining a good number of ancestral traits. The Enoplia do not seem to be monophyletic either but to contain two distinct lineages. The old group Chromadoria seems to be another paraphyletic assemblage, with the Monhysterida representing a very ancient minor group of nematodes. Among the Secernentea, the Diplogasteria may need to be united with the Rhabditia while the Tylenchia might be paraphyletic with the Rhabditia [2, 5]

### Adaptation of Nematodes

Nematodes have successfully adapted to nearly every ecological niche from marine to fresh water, from the polar regions to the tropics, as well as the highest to the lowest of elevations. They are ubiquitous in freshwater, marine, and terrestrial

environments, where they often outnumber other animals in both individual and species counts, and are found in the locations as diverse as Antarctica and oceanic trenches. They represent, for example, 90% of all life on the seafloor of the Earth. The many parasitic forms include pathogens in most plants and animals (including humans). Some nematodes can undergo cryptobiosis [2].

### **Reproduction of Nematodes**

Reproduction is usually sexual. Males are usually smaller than females (often much smaller) and often have a characteristically bent tail for holding the female for copulation. During copulation, one or more chitinized spicules move out of the cloaca and are inserted into genital pore of the female. Amoeboid sperm crawl along the spicule into the female worm. Nematode sperm is thought to be the only eukaryotic cell without the globular protein G-actin [2].

Eggs may be embryonated or unembryonated when passed by the female, meaning that their fertilized eggs may not yet be developed. In free-living roundworms, the eggs hatch into larva, which eventually grow into adults; in parasitic roundworms, the life cycle is often much more complicated.

Nematodes, as a whole, possess a wide range of modes of reproduction. Some nematodes, specifically *Heterorhabditis* sp., undergo a process called *endotokia matricida*: intrauterine birth causing maternal death. Some nematodes, like other animals (for example segmented worms), are hermaphroditic. The hermaphroditic nematode keeps its self-fertilized eggs inside its uterus until they hatch. The juvenile nematodes will then ingest the parent nematode. This process is significantly promoted in environments with a low or reducing food supply.

The nematode model species *Caenorhabditis elegans* and *C. briggsae* exhibit androdioecy, which is very rare among animals. The single genus *Meloidogyne* (root-knot nematodes) exhibits a range of reproductive modes including sexuality (amphimixis), facultative sexuality, meiotic parthenogenesis (automixis), and mitotic parthenogenesis (apomixis) [6].

### **Nematodes Species**

Nematodes can either be free-living or parasitic [2, 7]. The free-living species feed on materials as varied as algae, fungi, small animals, fecal matter, dead organisms, and living tissues. Free-living marine nematodes are important and abundant members of the meiobenthos. They play an important role in the decomposition process, aid in recycling of nutrients in marine environments and are sensitive to changes in the environment caused by pollution. One roundworm of note is *Caenorhabditis elegans*, which lives in the soil and has found much use as a model organism. *C. elegans* has had its entire genome sequenced, as well as the developmental fate of every cell determined, and every neuron mapped.

Nematodes which are commonly parasitic on humans include ascarids (*Ascaris*), filarids, hookworms, pinworms (*Enterobius*), and whipworms (*Trichuris trichiura*). The species *Trichinella spiralis*, commonly known as the trichina worm, occurs in rats, pigs, and humans, and is responsible for the disease trichinosis. *Baylisascaris* usually infests wild animals but can be deadly to humans as well. *Haemonchus contortus* is one of the most abundant infectious agents in sheep around the world, causing great economic damage to sheep farms. In contrast, entomopathogenic nematodes parasitize insects and are considered by humans to be beneficial.

One form of nematode is entirely dependent upon fig wasps, which are a major source of fig fertilization. They prey upon the wasps, riding them from the ripe fig of the wasp's birth to the fig flower of its death, where they kill the wasp, and their offspring await the birth of the next generation of wasps as the fig ripens.

Plant parasitic nematodes include several groups causing severe crop losses. The most common genera are *Aphelenchoides* (foliar nematodes), *Ditylenchus*, *Globodera* (potato cyst nematodes), *Heterodera* (soybean cyst nematodes), *Longidorus*, *Meloidogyne* (root-knot nematodes), *Nacobbus*, *Pratylenchus* (lesion nematodes), *Trichodorus* and *Xiphinema* (dagger nematodes).

Several phytoparasitic nematode species cause histological damages to roots, including the formation of visible galls (e.g. by root-knot nematodes), which are useful characters for their diagnostic in the field.

Some nematode species transmit plant viruses through their feeding activity on roots [8]. One of them is *Xiphinema index*, a vector of GFLV (Grapevine Fanleaf Virus), an important disease of grapes.

Other nematodes attack barks and forest trees. The most important representative of this group is *Bursaphelenchus xylophilus*, the pine wood nematode, present in Asia and America and recently discovered in Europe.

Depending on the species, a nematode may be beneficial or detrimental to plant health. From an agricultural perspective, there are two categories of nematode: predatory ones, which will kill garden pests like cutworms, and pest nematodes, like the root-knot nematode, which attack plants [9].

Predatory nematodes can be bred by soaking a specific recipe of leaves and other detritus in water, in a dark, cool place, and can even be purchased as an organic form of pest control [7].

### **Location of Nematodes**

Parasitic nematodes are either located in the intestine or tissues of their hosts and are referred to as intestinal and tissue nematodes, respectively [10, 11].

#### **Intestinal nematodes**

Small intestine: *Ascaris, Ancylostoma, Strongyloides etc.*

Large intestine: *Enterobius, Trichuris etc.*

#### **Tissue nematodes**

Lymphatic system: *W. bancrofti*

Subcutaneous tissue: *O. volvulus, Loa loa, D. medinensis*

Muscles/lungs/brain: *Trichinella (larva)*  
*Toxocara canis non human species larva*  
carried in blood to liver, lung, brain, and eye.

## **GASTRO-INTESTINAL NEMATODES**

### **Intestinal Nematodes**

Intestinal nematodes are a class in the animal kingdom with more than 20,000 species described. They have managed to live in a large variety of habitats. Most of these are parasites of animal or humans. In veterinary medicine, parasitic nematodes comprise an important group of endoparasites. Their clinical importance relates to the diseases they may cause in the animal host, as well as the zoonotic potential for pet owners and others [1].

### **General Features of Intestinal Nematodes**

The general features of intestinal nematodes include [12]:

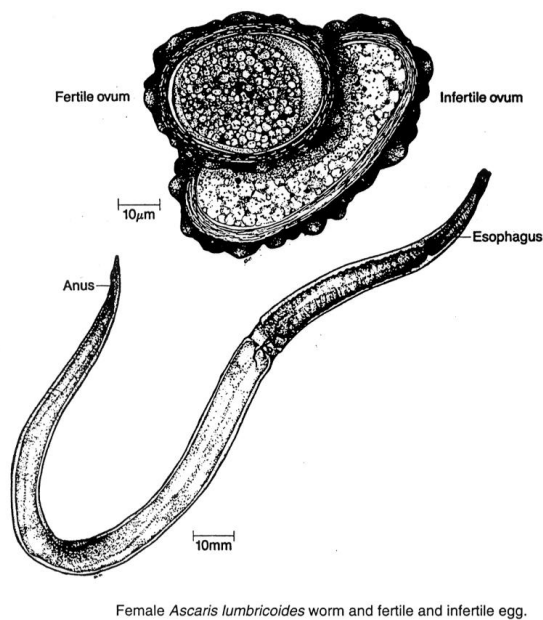
- Non-segmented, fusiform, cylindrical shape with a complete digestive system with oral and anal openings
- Separate sexes with female being larger than the male;
- Covered with a tough outer coating (cuticle) that may be smooth or textured; generally cream white in color; size ranges from millimeters to several centimeters.
- Life cycle usually involves an egg and one or more larval stages some of which may be free living.
- Diagnosis is made by observing and identifying the egg and larval stage found in feces.

### **Common Intestinal Nematode (Nematode Infections)**

Common intestinal nematodes include:

- *Enterobius (Oxyuris) vermicularis* (Pinworm, seatworm, threadworm)
- *Trichuris trichiura* (whipworm)
- *Ascaris lumbricoides* (roundworm)
- *Ancylostoma duodenale* & *Necator americanus* (hookworm)
- *Strongyloides stercoralis*

**Ascaris lumbricoides - Large Intestinal Roundworm**



**Figure 1:** *Ascaris lumbricoides*.

**Distribution:** temperate, subtropical and tropical areas with poor sanitation.

**Adults:** F 20-35cmX0.5-1cm M <30cmX0.5cm (curved tail).

**Infective stage:** embryonated egg.

**Diagnostic stage:** egg in stool (10-15 days to become infective).

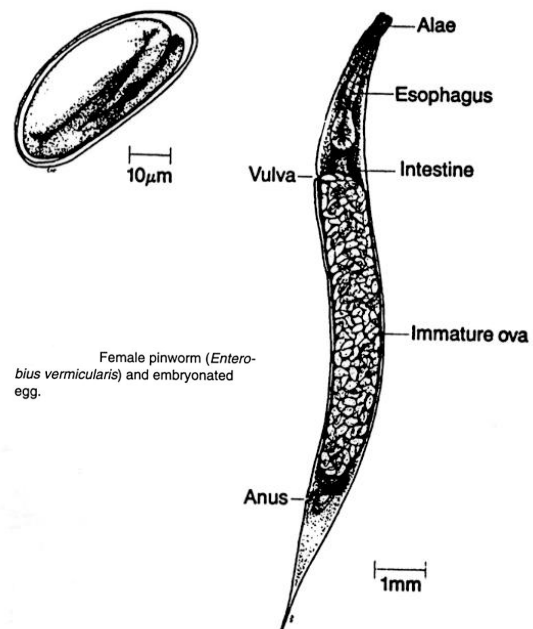
**Fertile:** round-broadly oval, 45-75µm X 35-50 µm bile stained, thick walled with round one celled embryo; may have albuminoid mammillated outer coating.

**Infertile:** more markedly oval, 40 µm X 90 µm, thinner wall surrounding undifferentiated yolk mass, may have albuminoid mammillated outer coating.

**Symptomatology:** During larval migration phase symptoms will vary depending on numbers; low numbers produce no definitive symptoms while large numbers of migrating larvae may provoke pneumonitis and asthma attacks in sensitive individuals. Low numbers of adults generally produce few if any symptoms; a large worm

burden can lead to intestinal obstruction, especially in children. Fever, ingestion of some drugs and some anesthetic agents can induce extra-intestinal migration of adult worms. Eight weeks from ingestion of egg to adult form [12].

**Enterobius vermicularis – Pinworm**



**Figure 2:** *Enterobius vermicularis*.

**Distribution:** most common in temperate areas with high sanitation standards.

**Adults:** F 8-13 µm X 0.5 µm M 2-5 µm X 0.2 µm.

**Infective stage:** embryonated egg.

**Diagnostic stage:** Egg can be observed in scotch tape mount (infective within six hours) double walled hyaline egg resembling under inflated football, containing C shaped embryo; 50-60 µm X 20-32 µm .

**Symptomatology:** migration of adult females may cause anal pruritus; migration into the vagina can produce local irritation. Majority of infected individuals remain asymptomatic. Egg ingests to adult form within 4-7 weeks [12].

## Hookworms

Hookworms can either be *Ancylostoma duodenale* (old world) or *Necator americanus* (new world) [11, 12, 13].

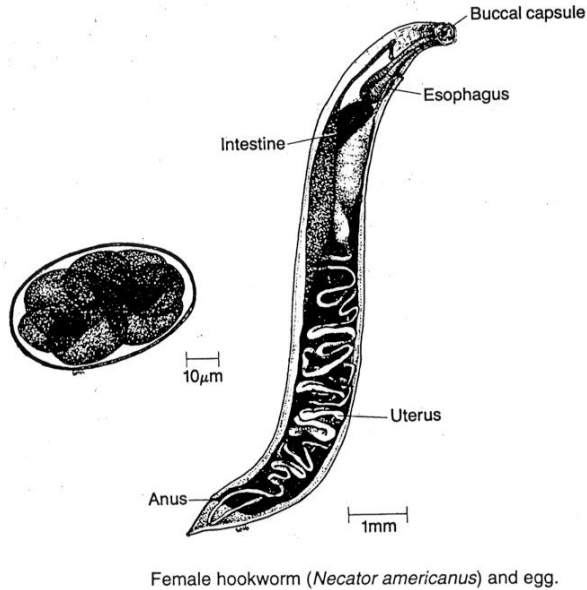


Figure 3: Hookworm.

**Distribution:** tropical and subtropical areas with sandy soil and ample rainfall; major cause of parasitic disease. Hookworms cannot be differentiated based on the appearance of the diagnostic stage (egg). Identification of species depends on the recovery and identification of the adult. This is not necessary for treatment purposes.

**Adults:** *A. duodenale* M < 1 cm X 0.5, F larger and thicker; oral cavity with two pairs of teeth. *N. americanus* M 5-9mm, F 10mm; oral cavity with two pairs of cutting plates.

**Infective stage:** filariform larva.

**Diagnostic stage:** embryonated egg (5-6 days to reach infectivity) 56-60 µm X 36-40 µm (*A.d.*) 64-76 µm X 36-40 µm (*N.a.*); oval shaped with a thin hyaline shell surrounding 2-8 celled embryo.

**Symptomatology:** localized dermatitis following larval penetration ("ground itch"); pulmonary symptoms are uncommon due to small size of migrating larvae. Adults may cause gastrointestinal symptoms depending on numbers. These can include diarrhea, flatulence,

abdominal or epigastric pain, nausea and vomiting. Chronic infections can result in significant blood loss especially for *A. duodenale*. Iron deficiency anemia may be seen among undernourished individuals. Four to seven weeks from larval penetration to adult form.

## **Strongyloides stercoralis - Thread worm**

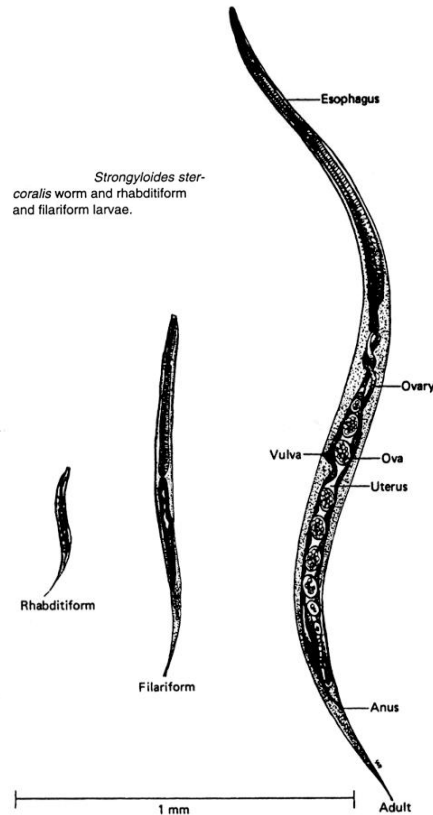


Figure 4: *Strongyloides stercoralis*.

**Distribution:** same areas as hookworm but less frequently observed.

**Adults:** F 2-2.5mm X 0.4mm.

**Infective stage:** filariform larva (3-4 days to develop from rhabditiform larva).

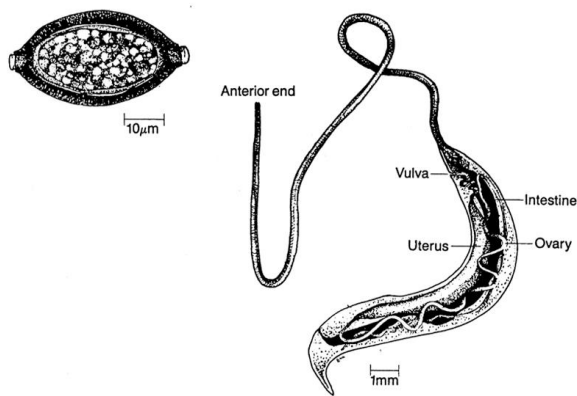
**Diagnostic stage:** rhabditiform larva (filariform larva).

**Rhabditiform:** short buccal cavity and prominent genital primordium.

**Filariform:** esophagus approximately ½ length of body, notched tail.

**Symptomatology:** localized dermatitis following larval penetration; pneumonitis possible but not common. Adults produce symptoms that vary from asymptomatic to severe diarrhea depending on numbers present. Hyperinfection can result in fever, gastrointestinal problems, dyspnea, hemoptysis and cough. Very high numbers of migrating larvae can cause damage to liver, kidney, heart and CNS; essentially disseminated strongyloidiasis. Four weeks from larval penetration to adult form [12].

***Trichuris trichiura* – Whipworm**



Female whipworm (*Trichuris trichiura*) and unembryonated egg.

**Figure 5:** *Trichuris trichiura*.

**Distribution:** worldwide, especially in area with poor sanitation and hygiene (similar to *Ascaris*).

**Adult:** both sexes are characterized by a slender anterior section and thickened posterior and resembling a whip; male posterior end is tightly coiled. Female 3-5 cm, Male <4cm.

**Infective stage:** embryonated egg (by ingestion).

**Diagnostic stage:** fertilized egg in stool (3 weeks to become infective). Barrel shaped, bile stained with hyaline polar plugs, thick walled.

**Symptomatology:** light infections are usually without symptoms; heavier infections can lead to abdominal pain, diarrhea and bloody stools. Rectal prolapse is occasionally observed in

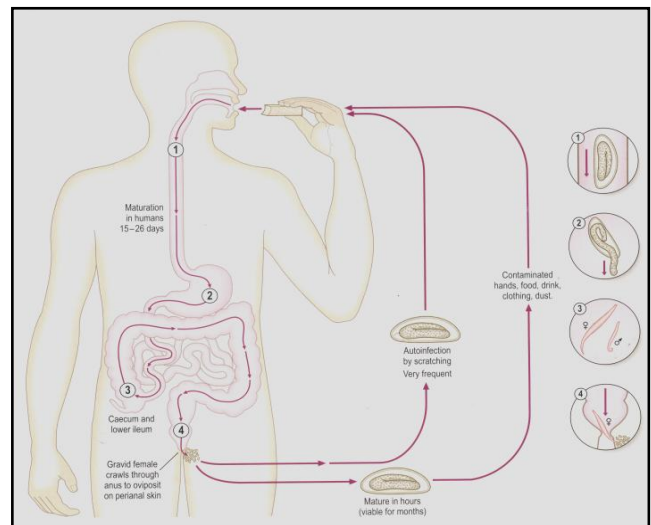
children with very heavy infections. About three months from ingestion to adult form.

**Diagnosis:** Symptomatology can include chronic nonspecific pulmonary disease, splenomegaly, eosinophilia and leukocytosis. Serologic tests are available for confirmation. Ocular toxocariasis-visual difficulties due to migration of larvae into the eye; visceral symptoms are often absent.

**Agents:** *Toxocara canis*, *T. cati*, *Ancylostoma caninum*, and *A. braziliense* [11, 12].

**LIFE CYCLE OF INTESTINAL NEMATODES**  
***Life cycle of Enterobius vermicularis (Oxyuris)***

This parasite is cosmopolitan. There is no intermediate host. Infection is via ingestion of eggs. They accumulate in the ileocaecal region. After copulation the males die. The females migrate via the colon to the anus and lay their eggs chiefly at night, as they creep over the perianal skin. This explains the nightly itching. In rare cases there is vaginal itch because the females can also hide there. Sometimes the parasites are found in the vermiform appendix. The eggs must be sought not only in the faeces, but also on the peri-anal skin (using Scotch tape or other transparent sticky tape). In women the eggs may be found in the urine, due to contamination. Sometimes a small number of adult worms are found in the vagina. Apart from the itch there are few problems [13].

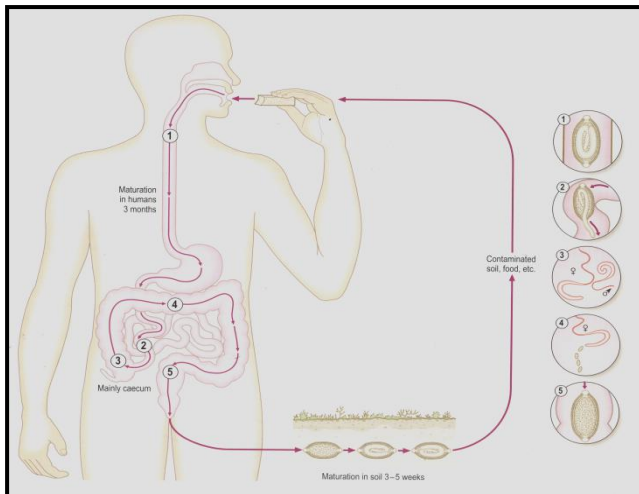


**Figure 6:** Life Cycle of *Enterobius vermicularis* (*Oxyuris*) [11].

**Treatment:** Mebendazole 100 mg (Vermox®), to be repeated after 1 and 2 weeks. Albendazole is also effective. Since the eggs can adhere to all objects e.g. underclothing, sheets and so on, these should be changed. In a family it is best to treat all the family members, even those without symptoms. Vanquin® (pyrvinium) may also be used as an alternative to mebendazole. The faeces may discolor red.

### **Life Cycle of *Trichuris trichiura* (Whipworm)**

Whipworms are cosmopolitan. The eggs are eliminated with the faeces. Infection is via the oral route (direct anus-hand-mouth as in *Enterobius* or after maturation in the outside world). In one week it becomes an adult worm measuring 3 to 5 cm. Egg laying begins 2 months after infection. The adult worm has a thin whip-like head with which it buries itself in the mucosa of the large intestine. The worm survives for several years. The parasite is possibly the same as *Trichuris suis*, a parasite of pigs [13].



**Figure 7:** Life Cycle of *Trichuris trichiura* (Whipworm) [11].

**Symptoms:** Most infected humans remain asymptomatic. Only in severe infections (> 1000 worms; >10,000 eggs per gram of faeces) do symptoms occur: these include diarrhea (dysentery type), malnutrition or anaemia. In undernourished children with chronic diarrhea and tenesmus there is sometimes prolapse of the

rectum, in which the worms can be seen on the prolapsed mucosa.

**Diagnosis:** Diagnosis is based on faecal examination. No concentration technique is necessary for clinically relevant infections. Sometimes the worms can be seen on the rectal mucosa (rectoscopy or during anal prolapse).

**Treatment:** The use of Mebendazole (100 mg BD x 3 days) and albendazole.

### **Life Cycle of *Ascaris lumbricoides***

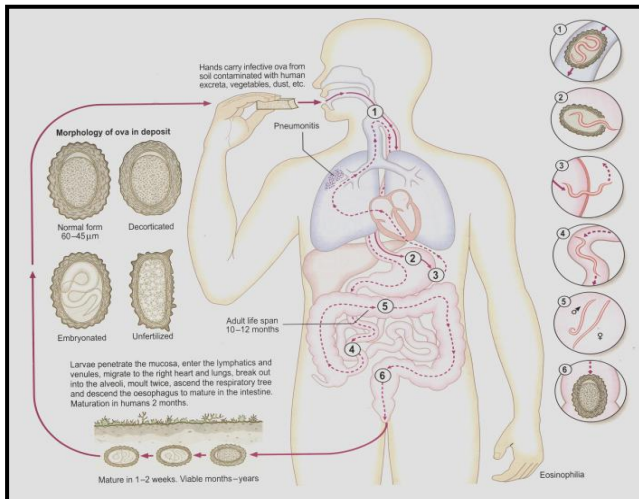
The eggs pass on to the ground via the faeces. Fertilized eggs require 10 to 40 days in the outside world to mature before they become infectious. Direct self-infection is thus ruled out. Once they are mature the eggs are taken up once more (faeco-oral transmission) via infected food, drink, dirty hands or fingernails.

In the intestine small larvae emerge from the eggs, and these bore through the intestinal wall. In this way they reach the blood (portal vein system). They are carried with the blood, through the liver to the lungs (lung passage occurs 3 to 14 days after ingestion).

In the lungs the larvae make their way to the bronchial lumen and climb via the respiratory branches into the throat. They are swallowed, and in this way they again reach the intestine. They grow into adult worms in the jejunum. They do not damage the intestinal wall.

Egg-laying begins two months after infection. The adult worm survives on average for 1 year. The creatures reach 15 to 40 cm. There is no animal reservoir. Occasionally infections with *Ascaris suum* occur (parasite of pigs), this worm resembles *Ascaris lumbricoides* very closely and some think the parasites are identical [13].

**Epidemiology:** This is the most common worm infection in humans. It has a cosmopolitan distribution. Children are most often infected. The eggs are very resistant, which makes it possible in certain circumstances for them to survive for a long time in the outside world. The number of eggs which can be found in the soil is a measure of the hygiene standard and degree of sanitation of an area (faecal pollution of the ground).



**Figure 8:** Life Cycle of *Ascaris lumbricoides* [11].

**Symptoms:** The vast majority are asymptomatic. Some people have various forms of intestinal discomfort or allergic symptoms. Serious complications are rare. Nevertheless, in view of the large number of infected persons, the morbidity and mortality should not be disregarded.

The larvae undergo lung passage. This produces symptoms of mild to severe cough, dyspnoea, thoracic pain, some fever. The clinical picture is similar to asthma or pneumonia. On chest X-ray migratory infiltrates are observed. Eosinophilia is present. This whole phenomenon is called Loeffler's syndrome. The sputum contains many eosinophils, Charcot-Leyden crystals and sometimes also larvae. The symptoms last for some days or weeks.

When numerous adult worms are present, they may form a tangle and cause mechanical intestinal obstruction manifested by a bloated abdomen, increased peristalsis with clangour, colicky pain, vomiting (bile, faecaloid) and dilated intestinal lumen on an abdominal X-ray. Migration into the biliary tract may lead to biliary obstruction (cholestasis) with possibly infection (cholangitis, liver abscess, pancreatitis). Sometimes there is migration to the appendix with inflammation (appendicitis). Sometimes an adult *Ascaris* is present in vomitus. Occasionally, an adult can penetrate the lacrimal duct. Recent surgical intestinal sutures can be breached by an inquisiting adult *Ascaris*, leading to bowel perforation and peritonitis. Infection with *Ascaris*

*lumbricoides* also plays a role in the development of pigbel (necrotising enteritis).

**Malnutrition:** *Ascaris* itself does not cause malnutrition. In borderline malnutrition the presence of numerous worms can have a negative effect, however. It is also important to know that many patients suffer from anorexia. Humans infected with *Ascaris* are best treated before they undergo intestinal surgery. Migration of an *Ascaris* through an intestinal suture line is a serious event. Pre-operative de-worming is advised in endemic areas.

**Diagnosis:** Since an adult female lays up to 200,000 eggs per day, as a rule no concentration technique is necessary to detect eggs in the faeces. If infection is solely with one or more male worms, then of course no eggs will be detected. During lung passage there is significant eosinophilia. After lung passage there is no longer appreciable eosinophilia. X-ray of the intestine may show one or more adult worms. The worm forms a long, thin dark area if using barium contrast. Sometimes a central longitudinal radio-opaque line can be seen; this is the intestinal tract of the worm. Such a line is absent in tapeworms. An ultrasound of the pancreas (Wirsung duct) or of the biliary tract and gall bladder sometimes shows an ectopic migrating adult *Ascaris*.

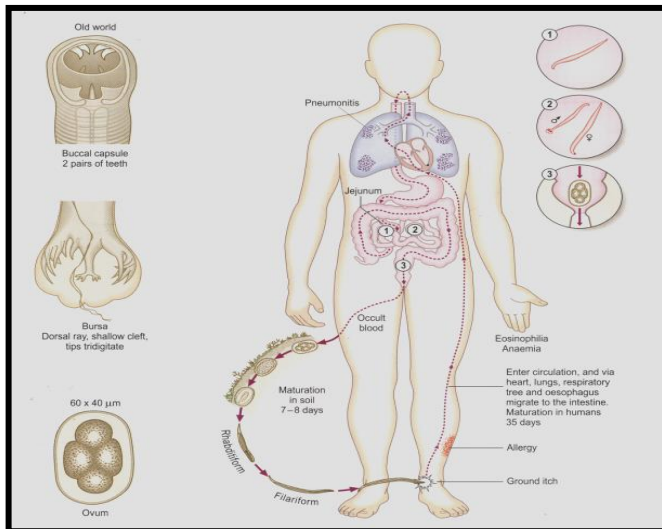
**Treatment:** Mebendazole (Vermox®): 100 mg BD x 3 days, effective broad spectrum, Flubendazole (Fluvermal®): 100 mg BD x 3 days, effective, narrow spectrum, Albendazole, effective, broad spectrum, Piperazine (Adiver®): narrow spectrum and Pyrantel (Antiminth®, Combantrin®)

### Life Cycle of Hookworms

The adult worms are found in the small intestine. They measure approximately 1 cm. Adult hookworms survive for several years, *Necator* longer than *Ancylostoma*. A few weeks or months after infection eggs can be found in the faeces. Once the eggs arrive in the outside world with the faeces, they take one week to mature to infectious larvae. At first they are rod-shaped = rhabditiform, later thread-shaped = filariform. They may survive for weeks or months (at an optimal temperature and humidity for as much as 2 years). A soil with neutral pH is optimal for their development, as is shade and a sufficiently high temperature (23°C to 30°C is ideal). If the faeces mix with urine the eggs die.



Frost, direct sunlight and a soil saturated with salt or water, are unfavorable for the development of the young parasites. Infection occurs via the mouth (*A. duodenale*) or via the skin (*A. duodenale* and *N. americanus*). If they enter through the skin, the young parasites have to pass through the lungs. A new dimension in the epidemiology of hookworm disease emerged when it was found that insufficiently cooked meat from paratenic hosts such as pigs, cattle, rabbits and sheep can be responsible for transmission. The adult hookworms bore a hole in the mucosa of the duodenum and the small intestine and suck blood. They adhere with hooked teeth in their mouth (*Ancylostoma*) or with two buccal cutting plates (*Necator*). *A. duodenale* sucks 5 to 10 times more blood than *N. americanus* (approximately 30  $\mu$ l per day for *Necator* and 260  $\mu$ l for *Ancylostoma*). It is estimated that the life span of adult worms is 5 to 15 years [13].



**Figure 9:** Life Cycle of Hookworms [11].

**Symptoms:** At the site where the hookworms penetrate, the skin develops a rash and itch. This is short-lived and rarely noticed. Lung passage also rarely produces symptoms, but may be accompanied by Loeffler's syndrome. There are few intestinal symptoms. Significant infections (>1000 worms) may result in pronounced anemia. The haemoglobin level may sometimes be very low. Children and pregnant women in whom the iron supplies are already low, are particularly affected. Hypoproteinaemia may also occur and results in oedema. Protein deficiency also has consequences for the production of immunoglobulines. Some patients exhibit

geophagia. In history, certain regions in the USA were famed for their "quality" clay and people would cover great distances to eat this iron-containing soil. In 1920 someone even began a mail order business to send clay to people with hookworms.

**Diagnosis:** The eggs are found in fresh faeces. In an old stool (>24 hrs), the eggs will have hatched and rhabditiform larvae can be seen (Gr. rhabdos = rod). There is mild eosinophilia. Since an adult hookworm lays approximately 25,000 eggs per day, as a very rough estimate 100 eggs per gram of faeces corresponds to 1 adult worm.

**Differential diagnosis:** Differentiation from *Strongyloides* larvae is based chiefly on the difference in morphology of the "head" end. The mouth is elongated in ancylostomes and shorter in *Strongyloides*. Sometimes, if intestinal transit has been swift, eggs of *Strongyloides stercoralis* may be found in the faeces. These too should be differentiated from hookworm eggs. *Ternidens deminutus* is a nematode which is generally non-pathogenic, and which strongly resembles the hookworm (although *Ternidens* eggs are somewhat larger). Eggs of *Oesophagostomum* are morphologically identical to those of hookworms. Identification of the latter parasite can only be made by coproculture (identification of the typical stage 3 larvae).

**Treatment:** Mebendazole 2 x 100 mg/day for 3 days, Pyrantel 10 mg/kg for 3 days. Also give iron supplementation and folic acid in anemia. *Necator* is less sensitive to ivermectin, unlike *Ancylostoma duodenale*. Albendazole may be used in treatment and is generally effective.

**Prevention:** Mass chemotherapy, together with health education and sanitary provisions are strategies which are often used. The most heavily infected individuals are the chief target group. Wearing footwear only partly prevents infection because oral infection is also important for *Ancylostoma duodenale*. Children are the main victims, rarely wear shoes and their whole skin is a portal of entry.

### Life Cycle of *Strongyloides stercoralis*

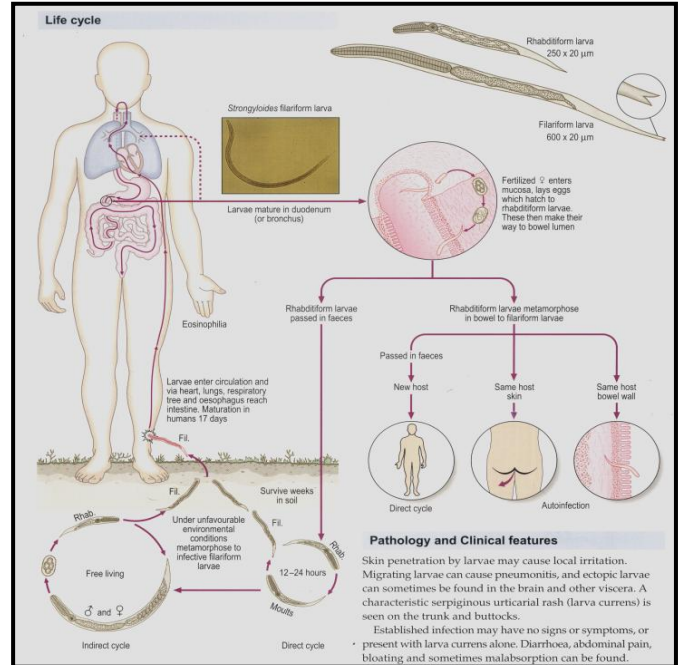
The adult female worm, (average 2.7 mm) is found in the mucosa of the small intestine. Males cannot penetrate the intestinal mucosa and

perish. Reproduction is asexual via parthenogenesis. The females lay eggs after 2-3 weeks, from which larvae are quickly produced. Initially the larvae are described as rhabditiform. These quickly develop into filariform (infectious) larvae. These larvae may:

- either penetrate back into the intestinal mucosa.
- or pass to the perianal skin and from there again penetrate the body (auto re-infection). In auto re-infection there is always another lung passage. In this way an infection with *Strongyloides* may persist for a very long time (more than 30 years).
- or pass to the outside world with the faeces. From there, after molting, they may go in either of two directions. The larvae either again penetrate the skin of a human (sometimes even via the mouth) or they develop to adult worms in the outside world. They may then via sexual reproduction in their turn lay eggs, from which new larvae develop. The worm can thus survive without a host.

Some related parasites which seldom cause infections in humans include:

- I. *Strongyloides fuelleborni* (*S. fuelleborni*) in sub-Saharan Africa and Papua New Guinea. Larvae actively penetrate the skin. There is probably also transmission via breast milk. In severe infections the patient may suffer heavy loss of protein via the intestine, leading to so-called protein-losing enteropathy. *Strongyloides kellyi* is a nematode which is only known in Papua New Guinea. Transmammary transmission is very probable.
- II. *Strongyloides papillosus*, *S. ransomi* and *S. westeri*: cosmopolitan. Larvae may be found in the skin.
- III. *Strongyloides canis*, *S. cebus*, *S. felis*, *S. myopotami*, *S. planiceps*, *S. procyonis* and *S. simiae* can cause experimental infections. Natural infections with these parasites are (as yet) unknown [13].



**Figure 10:** Life Cycle of *Strongyloides stercoralis* [11].

**Symptoms:** Mild infection is generally asymptomatic. In severe infections there may be intestinal discomfort or diarrhea. During lung passage symptoms may occur, depending on the number of larvae. Auto re-infection via the skin may give rise to significant itching, chiefly perianal. Migration of the larvae in the skin leads to itching red swollen lines (on the rump, arms, face, etc.). These lines may occur anywhere and progress swiftly (up to 10 cm per hour). The swelling is the result of an urticarial reaction to the migrating larva (the larva itself is only 0.5 mm long). These lesions disappear spontaneously a few hours later, to reappear once more at a different site.

Immune suppression (especially HTLV-1 infection), achlorhydria, haematological malignancies including lymphoma, cytotoxic medication, nephrosis, burns and especially the long-term use of systemic corticoids, all increase the risk of hyperinfection. In such cases there is extensive multiplication with spread of the larvae to all organs. Symptoms include purpura-like skin lesions (initially often peri-umbilical), severe diarrhea, pulmonary symptoms (dyspnoea, bronchospasms, bloody sputum) and meningo-encephalitis.

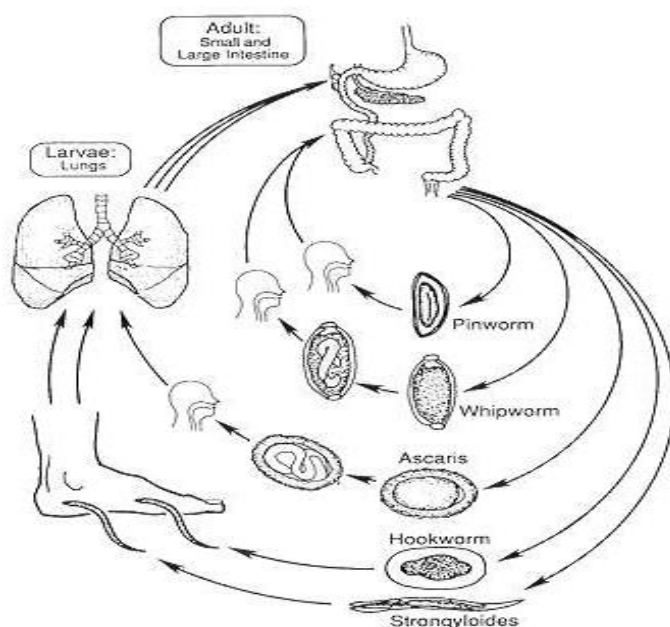
Hyperinfection with *Strongyloides stercoralis* may be accompanied by bacterial septicaemia. Usually Gram-negative bacteria are involved. Mixed infection may occur. This probably depends on mechanical damage to the colon wall, adhesion of intestinal bacteria to the outside of migrating larvae and excretion of bacteria from the intestinal system of the parasite. Hyperinfection has a high mortality (75%). In chronic and persistent infection, an underlying infection with HTLV-1 or use of glucocorticoids should be considered. There have been fewer hyperinfections in AIDS patients than one would expect at first sight.

**Diagnosis:** The eggs hatch very rapidly in the intestine and are often not found in a faecal specimen. Larvae are found in the faeces. Often the numbers are not so high and a concentration technique, called the Baermann method, needs to be used. Larvae can also be detected via duodenal intubation. Differentiation from hookworm larvae is necessary. Eosinophilia is almost always present, except when immune suppression exists. A history of larva currens is suggestive of strongyloidosis and is enough to start treatment even if no larvae are found in the faeces. In hyperinfection larvae may be found in the sputum or in broncho-alveolar lavage fluid. The sputum must be regarded as infectious. If this sputum is cultured on blood agar, bacterial colonies can be seen which form a curvilinear pattern, reminiscent of a pearl necklace. This follows the migration of a larva on the agar plate, with translocation of the bacteria.

**Therapy:** Thiabendazole was used in the past, but had many side effects. Albendazole is only moderately effective. Mebendazole is not active. Ivermectin is easy to use and effective and at present is the first line treatment. If immunosuppression is present, the cure rate with ivermectin is lower, certainly if cortisone has been taken. It should be mentioned that there are parenteral ivermectin formulations for veterinary use. They are not (as yet) registered for use in humans, but anecdotal case reports mention success with them. Cyclosporin A is an immunosuppressive agent, which among other indications is used for transplant patients. It has an anthelmintic action on *strongyloides stercoralis*. Transplant patients who take cyclosporin A have a greatly reduced risk of *strongyloides* hyperinfection. In hyperinfection it is important not to forget to use antibiotics, in view of the risk of severe septicaemia.

## CONCLUSION

In conclusion, Nematodes constitute a very homogeneous group as far as anatomical and morphological features are concerned, but exhibit very diverse living habits. Nematodes are either located in the intestine or tissues of the host and are referred to as intestinal and tissue nematodes respectively. Many of them live as parasites; especially large intestinal roundworm, pinworm, hookworms, threadworm, whipworm and other roundworms are enemies and detrimental to Man and domestic animals. The generalized life cycle of intestinal nematodes could be represented in Figure 11 below [14]:



**Figure 11:** Generalized Life Cycle of Intestinal Nematodes.

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