# *Toxocara vitulorum/Neoascaris vitulorum*

**Host:** Cattle and Buffalo (sometimes sheep and goat)

**Site:** Small intestine

**Distribution:** Worldwide, Sporadic, Mainly in tropical and worm regions

**Disease:** Bovine Toxocariasis/Round-worm disease of cattle

**Morphology:**

**Adult**

- It is the largest intestinal parasite/worm of cattle
- Male: 25cm and Female: 30cm in length
- Has 3 lips and 3 to 4.5mm long oesophagus. The base of the oesophagus has a ventriculus
- Male tail usually form a spike like appendage
- 5pairs of post cloacal papillae. There is also pre-cloacal papollae which are variable in number
- Vulva opens at about $1/8^{th}$ of the body length to form anterior end
- It is a thick worm, pinkish when fresh and the cuticle is rather transparent so that the internal organs can be seen.

**Egg:**

- Sub-globular
- Thick pitted shell
- Almost colorless
- 70-90 μm in size

## Epidemiology:

- Young are most susceptible (Below 6 months of age)
- Egg production is very high ($8X10^6$ Egg/female/day)
- Infected Dam may act as carrier
- Specific feature:

○ The reactive larvae of the dam's serve as the source of milk-borne infection of calves which are exposed to infection from the first day of life

- Most Patent infection occurs in calves younger than 6 month of age.

## Pathogenesis and clinical signs:

- Infection with migrating/dormant larvae are of low pathological significance and asymptomatic. So, in adult cattle, Clinical signs are absent. The main effects of the infection appears to be caused by adult worms in the intestine of calves upto 6 months old.
- Anorexia, Wasting, Poor coat
- Diarrhoea (intermittent), Steatorrhoea. The feces yellowish, sometimes normal
- Intestional colic associated with strong butyric or acetone odour found in Animal breath
- Intestinal obstruction
- Perforation of Bowel
- Pot belly appearance
- Grinding of teeth

Eggs in faeces of infected calf

↓

Infective larva ($L_2$) developed in egg within 15 days

↓

The eggs ingested by the host, Hatch out the larva in the lumen of SI

↓

In older calves/adults (≥6months) the $L_2$ by piercing intestinal wall/mucosa enters the portal circulation

The larvae reach to various somatic organs

↓

The larvae are stored there. The hypobiotic or dormant larvae in the Mucosa, connective tissue or elsewhere, persisting for long periods

↓

In DAM, Resumption of development of the larvae occurs in late pregnancy

Larvae that reach the Mammary gland undergo ecdysis/molt in the blood vessel to form $L_3$

↓

$L_3$ break blood vessel and enter into the milk alveoli and pass out the Milk

↓

In calf, When newborn calf suckles Dam's milk/colostrums, the $L_3$ enter into the Abomasum of the calf

↓

3-7 days post infection, $L_3$ moult to $L_4$ which stays in SI, Final ecdysis occurs in SI to form the Adult worm

18-24 days of birth, Adult female lay eggs in the SI

**Treatment:**

- Anthelmintics
- Piperazine compounds: @ 100-200mg/kg BW twice one week apart
- Levamisole @ 7.5mg/kg body weight
- Benzimidazole

## *Ascaridia columbae* (Pigeon), *Ascaridia galli* (Bird), *Ascaridia dissimilis* (Turkeys)

**Disease:** Ascaridiosis or Round worm Disease of Birds

**Host:** Domestic and wild birds

**Site:** Small intestine

**Distribution:** World-wide

## Morphology:

- Largest nematode of poultry
- Typically fleshy Ascarids
- Stout and densely white
- Female up to 12cm and male 7.5cm long
- Has 3 large lips and oesophagus has no posterior bulb
- In male, has circular pre-cloacal sucker with a thick circular rim
- Male also bears caudal alae and papillae
- In female, vulva is located just anterior to the middle of the body

## Egg:

- Distinctly oval/ellipsoid, 80-95μm in size
- A thick smooth shell
- Filled with the dark zygote

## Epidemiology:

- The infection is most prevalent and heaviest in about 3 month old chicks
- Adult birds are symptomless carrier
- The infection may be transmitted by earthworm as paratenic host
- The larvated eggs may remain viable for one year
- Dietary deficiency (vit. A and protein deficiencies) predisposes to infection
- Intestinal Coccidiosis is a predisposing factors.

## Pathogenesis and clinical findings:

- Domestic fowl of different breeds show different susceptibility to infection
- Moderate infection are frequently in apparent
- The main effect is seen during the prepatent phase, when the larvae are in the mucosa\
- **Heavier infection may cause-**
- Curtailment of food
- Intoxication
- Catarrhal or hemorrhagic enteritis even death

**Life cycle:**

- The life cycle is direct
- The parasitic phase is Non-migratory, i.e. Larvae spend time in the intestinal mucosa and lumen of host's gut.
- PPP in young birds is 4 & 6 weeks for *A. galli* and *A. cloumbae*, respectively. In adult, it is 8wks or more.
- Infective stage is $L_2$ containing eggs
- Longevity up to 1 year
- The infection is by ingestion of larvated eggs ($L_2$) or by eating paratenic host (Earthworm)

The eggs in the feces of the infected host (bird)

The egg becomes infected at optimal temperature in a minimum of 3 week

The larvated eggs ingested by the host

The eggs hatch in small intestine of the host

In 8-17 days of infection, $L_2$ in intestinal mucosa

8 days post infection, $L_3$ in the lumen of intestine followed by $L_4$ and $L_5$

Adult in the lumen

**Diagnosis:**

- Clinical History
- Clinical findings
- Coproscopy for Egg identification, Fecal egg count

The eggs of *A. galli* can be distinguished form those of *Heterakis gallinarum* by their larger size (80μm or more)

**Treatment:**

Anthelmintics

Single dose:

1. Piperazine salt
   Orally in feed 250mg/kg or in DW (0.8%) for one day
2. Mebendazole: @ 40-50mg/kg
3. Flubendazole: @5mg/kg
4. Febantel: @25mg/kg
5. Tetramisole: @50mg/kg

Levamisole: @20-30mg/kg

## *Heterakis dispar, Heterakis gallinarum, Heterakis isolonche, Heterakis gallinae*

**Disease:** Caecal worm disease of Bird/Heterakiosis

**Host:**

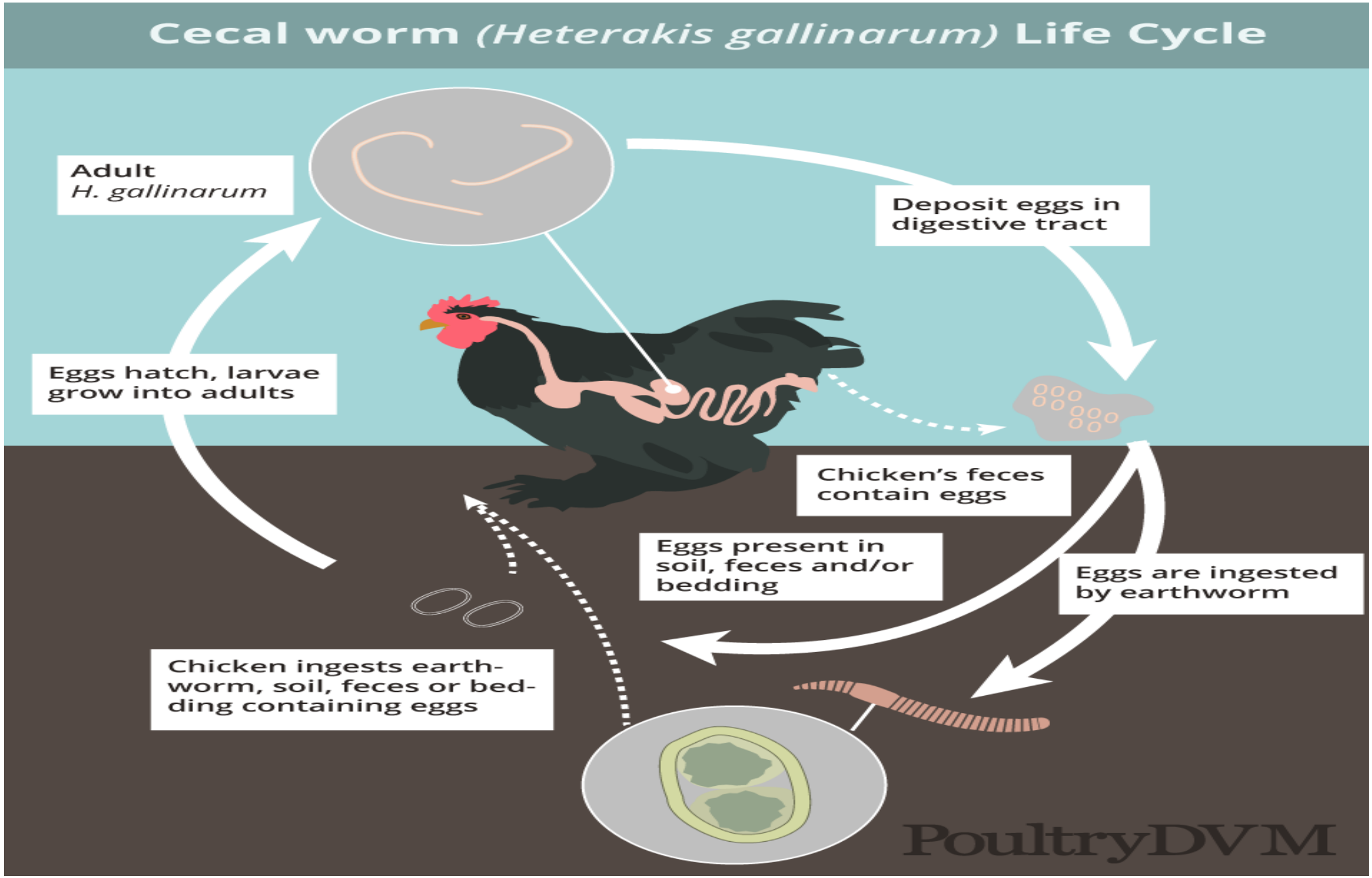
- *Heterakis gallinarum*: Domestic Fowl, GF, Turkey, Pigeon, Pheasant, Duck, Goose. (Commonest nematode of poultry)
- *Heterakis dispar:* Duck, Goose
- *Heterakis isolonche*: Pheasant, wild birds

**Site:** Caeca (large intestine)

**Distribution:** World wide

**Morphology:**

- Whitish worms
- Male is 7-13mm and Female is 10-15mm in length
- Has 3 lips
- Large lateral alae extending some distance down the sides of the body
- Oesophagus has a strong posterior Bulb
- Female has an elongated, sharp, pointed tail. Male tail has prominent caudal alae supported by large caudal papillae, has large precloacal sucker.
- Spicules are unequal in length (Right-2mm, left –0.7mm)
- *H. dispar* is larger than *H. gallinarum* and *H. isolonche*


Cecal worm (Heterakis gallinarum) Life Cycle
Adult
H. gallinarum
Deposit eggs in digestive tract
Chicken's feces contain eggs
Eggs present in soil, feces and/or bedding
Eggs are ingested by earthworm
Chicken ingests earthworm, soil, feces or bedding containing eggs
Eggs hatch, larvae grow into adults
PoultryDVM

## Pathogenesis and clinical signs/Pathogenesis:

### *H. gallinarum:*

- Usually non pathogenic but commonest nematode parasite in poultry
- But in heavy infection may cause thickening of the caecal mucosa with a number of petechial haemorrhage on the surface.
- The main important as a vector of Protozoa-Histomonas meleagridis, The causal agent for Balck Head or infectious Enterohepatitis in Turkey or Histomonosis or Typhlohepatitis. The protozoa remain viable in the eggs of *H. gallinarum* for long time.
- Re-infection may cause Verrucous Typhilitis associated with formation of nodule.

### *H. isolonche:*

- Pathogenic parasite
- In Phesants, it causes mucosal lesions resulting form markedly projecting nodules from mucosal and peritoneal surface containing adult worms.
- In heavy infection:
  - Typhilitis (inflammation of Caeca)
  - Ulceration of Mucosa
  - Diarrhoea, Unthriftiness
  - High mortality is found in heavily infected birds

## Diagnosis:

- Clinical History
- Clinical findings
- Presence of worm in caecal lumen or mucosal nodules of caeca at necropsy followed by microscopic examination of Adult worms.
- Coproscopy for Egg identification

## Treatment: Anthelmintics

- Piperazine compounds: @ 100-200mg/kg BW twice one week apart
- Levamisole @ 7.5mg/kg body weight
- Benzimidazole

## Control:

- Control is most necessary when Histomonosis is a problem in turkey
- The segregation of Turkey from other domestic poultry
- The removal and disposal of litter from poultry houses
- Hygienic management

## Superfamily: Spiruroidea

The precise classification of a number of genera currently assigned to this superfamily is controversial, but there are five of significance in veterinary medicine: Spirocerca, Habronema, Draschia, Thelazia and Gnathostoma. A major characteristic of this group is the **tight spirally coiled tail** of the **male**. The life cycles are indirect involving **arthropod intermediate hosts**.

## Spirocerca:

- The adult nematodes are found in **large granulomatous nodules** in the wall of the **oesophagus.**
- These may cause a variety of clinical signs including, infrequently, those of **oesophageal osteosarcoma.**

## Spirocerca lupi

## Identification

- The appearance of the granulomatous lesions, up to **golf-ball size**, is usually sufficient for identification.
- Numerous **pink worms** may be seen on section of the granulomas but these are difficult to extricate intact since they are coiled and up to 8.0cm long.

| Major Specie | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| ***Spirocerca lupi*** | Dog, occasional-ly cat | Coprophagous beetles | adults found in granulomatous lesions in the wall of the oesophagus and stomach, larva produce lesions in the wall of the aorta | **Tropical and subtropical areas** |
| ***Habronema muscae*** | Horse and Donkey | Muscid flies | Stomach | **Worldwide** |
| ***Draschia megastoma*** | Horse and Donkey | Muscid flies | Stomach | **Worldwide** |
| ***T. rhodesii,*** ***T. gulosa and*** ***T. skrjabini*** | Cattle | Muscid flies, particul-arly Musca, Fannia and Morellia | Ocular region especially the conjunctival sac and lachrymal duct | **Worldwide** |
| ***Gnathostoma hispidum*** | Pig | 1st I/H: aquatic crustacean<br>2nd I/H: vertebrate mammals, birds, reptiles, fish, amphibians | Stomach. | **Europe, Africa, Asia, Australia** |
| ***G. doloresi*** | Pig | | | |
| ***G. spinigerum*** | Cat & dog, erratically in man | | | |
| ***Gongylonema sp.*** | **Domesticated mammals,** | **Coprophagous beetles & cockroaches** | **upper alimentary tract, Oesophagus, forestomachs, and the of birds** | |

**Life Cycle**

The thick-shelled elongate egg, containing a larva, is passed in the faeces or vomit

↓

Eggs hatched, develops and encysted to the $L_3$ after ingestion by a dung-beetle (I/H)

↓

Paratenic hosts (domestic chicken, wild birds and lizards) may also be involved if the dung-beetle is ingested by them. In these the $L_3$, becomes encysted in the viscera.

↓

On ingestion of I/H or paratenic host by the final host the $L_3$ are liberated, penetrate the stomach wall and migrate via the coeliac artery to the thoracic aorta

↓

About three months later, they cross to the adjacent oesophagus where they provoke the development of granulomas as they develop to the adult stage in a further three months.

## Pathogenesis and Clinical Signs

- The migrating **larvae** produce **scarring of the internal wall of the aorta** which, if particularly severe, may cause **stenosis or even rupture**.
- The oesophageal granuloma **upto 4.0cm in size** associated with the **adult worms** may be responsible for a variety of clinical signs including **dysphagia** and **vomiting** arising from obstruction and inflammation.
- Two further complications are, first, the development of **oesophageal osteosarcoma** in a small proportion of infected dogs. These may be highly invasive and produce metastases. Secondly, also relatively rare, is the occurrence of **spondylosis** of the thoracic vertebrae or of hypertrophic pulmonary osteopathy of the long bones. The aetiology of these lesions is unknown.
- However, despite the potential pathogenicity of this parasite, many infected dogs do not exhibit clinical signs even when extensive aortic lesions and large, often purulent, oesophageal granulomas are present.

## Diagnosis

- Eggs may be found in the faeces or vomit if there are fistulae in the oesophageal granulomas. Otherwise diagnosis may depend on endoscopy or radiography.

## Treatment

- Treatment is rarely practical but levamisole, disophenol and albendazole have been reported to be of value. Disophenol is given subcutaneously at 7mg/ kg body weight and the treatment is repeated after seven days.

## Control

- This is difficult because of the ubiquity of the intermediate and paratenic hosts.
- Dogs should not be fed uncooked viscera from wild birds or from free-range domestic chickens.

# Habronema

Members of this genus and the closely related another genus *Draschia* are parasitic in the stomach of the horse: *Habronema* may cause a **catarrhal gastritis**, but is not considered an important pathogen while ***Draschia*** **provokes the formation of large fibrous nodules** which are occasionally significant. The chief importance of these parasites is as a cause of **cutaneous habronemosis** or **'summer sores'** in warm countries.

| Major Specie | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| ***Habronema muscae***<br>***H. microstoma (syn. H. majus)*** | Horse and Donkey | Muscid flies | Stomach | **Worldwide** |
| ***Draschia megastoma*** | **Horse and Donkey** | **Muscid flies** | **Stomach** | |

## Identification

- Slender **white worms** 1.0-2.5 cm long.
- In the male the tail has a **spiral twist**.
- It is unlikely to be confused with other nematodes in the stomach since *Draschia* is associated with characteristic lesions and *T. axei* is less than 1.0cm in length.
- The elongated eggs are thin shelled and larvated when laid.

**Epidemiology**

The seasonality of cutaneous lesions is related to the activity of the fly vectors.

**Life Cycle**

Eggs or $L_1$, are passed in the faeces of F/H

↓

$L_1$ are ingested by the larval stages of various muscid flies including *Musca, Stomoxys* and *Haematobia*

↓

Development to $L_3$ occurs synchronously with the development to maturity of the fly I/H

↓

When the fly feeds around the mouth of the horse the larvae pass from its mouthparts on to the skin and are swallowed. Alternatively infected flies may be swallowed whole.

↓

Development to adult takes place in the glandular area of the stomach in approximately two months

# Pathogenesis

- The adults in the stomach may cause a **mild catarrhal gastritis** with excess **mucus production**. More important are the **granulomatous lesions of cutaneous habronemosis**, commonly known **as 'summer sores'** and the **persistent conjunctivitis with nodular thickening and ulceration of the eyelids associated with invasion of the eyes**. Larvae have also been found associated with small lung abscesses.

## Clinical Signs

- Clinical signs are usually absent in gastric habronemosis.
- Lesions of cutaneous habronemosis are most common in areas of the body liable to injury and occur during the fly season in warm countries. During the early stages, there is **intense itching** of the infected wound or abrasion which may cause further **self-inflicted damage**. Subsequently a **reddish-brown non-healing granuloma** develops which protrudes above the level of the surrounding skin and may be up to **8.0cm in diameter**. Later the lesion may become more fibrous and inactive, but will not heal until the advent of cooler weather when fly activity ceases.
- Invasion of the eye produces a **persistent conjunctivitis with nodular ulcers** especially at the medial canthus.

## Diagnosis

- This is based on the finding of non-healing, reddish cutaneous granulomas.
- The larvae, recognized by spiny knobs on their tails, may be found in material from these lesions.
- Gastric infection is not easily diagnosed since *Habronema* eggs and larvae are not readily demonstrable in the faeces by routine techniques.

## Treatment and Control

- Any broad spectrum anthelmintics have activity against the **adult parasites** in the stomach.
- Cutaneous lesions are best treated with ivermectin.
- The use of insect repellents has some benefit and radiation therapy and cryo-surgery have been used in more chronic cases.
- Obviously any measures taken to prevent injuries and to control fly populations will be beneficial.

# Thelazia

| Major Specie | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| *T. rhodesii,* *T. gulosa and* *T. skrjabini* | Cattle | Muscid flies, particul-arly Musca, Fannia and Morellia | Ocular region especially the conjunctival sac and lachrymal duct | Worldwide |
| *Thelazia lacrymalis* | Equines | | | Europe and North America |
| *T. californiensis* | Dog, cat, sheep and occasionally man | | | North America |

## Identification

- Small thin white worms 1.0-2.0 cm long.
- A mouth capsule is present and the cuticle has prominent striation at the anterior end.

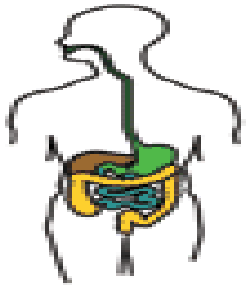
## Epidemiology

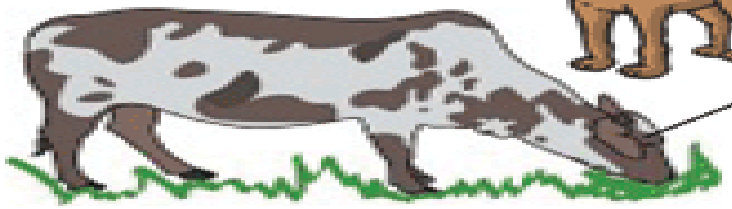
- *Thelazia* infections occur seasonally and are linked to periods of maximum fly activity.
- The parasite can survive in the eye for several years, but since it is only the young adult which is pathogenic a reservoir of infection may persist in symptomless carrier cattle.
- Survival of larvae also occurs in the pupal stages of flies during the winter.

6 Humans become incidental hosts when the insect intermediate host feeds on their tears

i = Infective Stage

d = Diagnostic Stage





Adults in conjunctival sac.

5 d

i

4 Cattle, horses and dogs serve as the primary definitive hosts and become infected when the insect intermediate host feeds upon their tears.

1 Adults shed larvae by ovoviviparity in the conjuctiva of the definitive host.

2 First-stage larvae are ingested by the insect intermediate host as it feeds on the tears of the definitive host.

3 Larvae develop into infective L3 larvae in the intermediate host.

## Pathogenesis

- Lesions are caused by the serrated cuticle of the worm and most damage results from movement by the **active young adults** causing **lachrymation**, followed by **conjunctivitis**.
- **In heavy** infections the cornea may become **cloudy and ulcerated**.
- There is usually complete recovery in about two months although in some cases areas of corneal opacity can persist.

## Clinical Signs

- Lachrymation, conjunctivitis and photophobia.
- Flies are usually clustered around the eye because of the excessive secretion.
- In severe cases, the whole cornea can be opaque.

## Diagnosis

- This is based on observation of the parasites in the conjunctival sac.
- It may be necessary to install a few drops of local anaesthetic to facilitate manipulation of the third eyelid.

## Treatment and Control

- Treatment was at one time based on manual removal of the worms under a local anaesthetic, but this is now replaced by administering an effective anthelmintic such as levamisole or an avermectin (applied topically as a 1 % aqueous solution.
- Prevention is difficult because of the ubiquitous nature of the fly vectors.

# Gnathostoma

Like most spiruroids, *Gnathostoma* inhabits the **upper /alimentary tract**, occurring in **nodules in the stomach wall** of omnivores and carnivores. It is exceptional in **requiring two intermediate hosts**.

| Major Specie | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| ***Gnathostoma hispidum*** | Pig | **1st I/H: aquatic crustacean**<br>**2nd I/H: vertebrate mammals, birds, reptiles, fish, amphibians** | **Stomach** | **Europe, Africa, Asia, Australia** |
| ***G. doloresi*** | Pig | | | |
| ***G. spinigerum*** | **Cat & dog, erratically in man** | | | |

## Identification

- *Gnathostoma* is a thick-bodied worm, and the females are up to 3.0cm long.
- The presence of the worms in **gastric nodules** is sufficient for generic diagnosis, and confirmation is easily made with a hand lens when the swollen anterior end covered with rows of small hooks will be seen.

## Pathogenesis

- As in many spiruroid infections, the most obvious effect of gnathostomosis is the **presence of fibrous growths (**3-4 cm in diameter/variable size**) on the stomach wall**.
- Ulceration and necrosis of the stomach wall are often present.
- The most pathogenic species is G. *spinigerum,* which in cats may cause fatal **gastric perforation and peritonitis.**
- In some cases a number of larvae will migrate from the stomach to other organs, most commonly the liver, in which they burrow, leaving necrotic tracks.
- When **visceral larva migrans** due to *Gnathostoma* occurs in humans G. *spinigerum* is the species usually involved, and the commonest source of infection is domestic poultry acting as second intermediate hosts. The worms never become fully adult, and the immature forms are most commonly found in subcutaneous nodules which appear and disappear irregularly as the parasites wander in various parts of the body.

## Clinical Signs

- Except in the cat, in which acute abdominal signs may be present, *Gnathostoma* infection is usually inapparent.

## Diagnosis

- The infection in the living animal can only be diagnosed by the finding of the greenish, oval eggs, which have a thin cap at one pole, in the faeces. Often, however, eggs are not present in faeces.

## Treatment and Control

- Treatment has not been investigated. With ubiquity of the first and second intermediate hosts complete control cannot be achieved, but partial limitation is possible by the thorough cooking of all food.

# *Parafilaria bovicola*

## Identification

- Slender white worms 3.0-6.0cm in length.
- Anteriorly, there are numerous papillae and circular ridges in tri cuticle.
- In the female the vulva is situated anteriorly near the simple mouth opening.
- Small embryonated eggs are laid on the skin surface where they hatch to release the microfilariae or $L_1$ which are about 200 µm in length.

## Epidemiology

- In Europe, bovine parafilariosis occurs in spring and summer,
- In tropical areas it is seen mainly after the rainy season.
- A high prevalence of 36% in cattle has been reported from some endemic areas in South Africa and the disease is now present in Sweden, an area previously free from infection.
- Parafilaria infection may be introduced by the importation of cattle from endemic areas, but its spread will depend on the presence of specific fly vectors.
- It has been estimated in Sweden that one 'bleeding' cow will act as a source of infection for three other animals.

**Pathogenesis**

- When the gravid female punctures the skin to lay her eggs, there is a **haemorrhagic exudate** or 'bleeding point' which streaks and mats the surrounding hair and attracts flies.
- Individual lesions only bleed for a short time and healing is rapid.
- there is **inflammation and oedema** at the predominant sites (on shoulders, withers and thoracic areas), which at meat inspection, resemble subcutaneous bruising
- Sometimes the lesions extend into the intermuscular fascia. The affected areas have to be trimmed at marketing and further **economic loss** is incurred by rejection or downgrading of the hides.

## Clinical Signs

- The signs of parafilariosis are pathognomonic.
- Active bleeding lesions (pathognomonic) along with fly intermediate host.

## Diagnosis

- Clinical signs
- Laboratory Diagnosis
  - Embryonated eggs or microfilariae may be found on examination of exudate from bleeding points.
  - The demonstration of eosinophils in smears taken from lesions is also considered a constant diagnostic feature.
  - Serodiagnosis (ELISA)

## Treatment

- For Beef and non-lactatiing cows: Ivermectin (single dose) or Nitroxynil (Two doses at an interval of three days).
- For lactating cattle less effective levamisole may be tried.

# Stephanofilaria

The worms of this genus inhabit the dermis and are responsible for chronic dermatitis in cattle and buffalo in the tropics and subtropics.

| Major Species | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| ***Stephanofilaria stilesi*** | Cattle and buffalo | Muscid flies<br>Musca conducens | Skin of lower abdomen | **USA, Russia** |
| ***S. assamensis*** | | | Hump region | **Asia.** |
| ***S. zaheeri***<br>***S. kaeli***<br>***S. okinawaensis*** | | | **Skin of head, legs and teats** | **India and the Far East** |

## Identification

- Very small worms, less than 1.0cm in length.
- Microscopically the mouth opening is surrounded by a **spiny collar**.

## Epidemiology

- In endemic areas, the incidence of infection may be as high as 90% and the occurrence is to a great extent influenced by the type of herbage.
- Succulent grazing produces soft, moist faeces which are more suitable breeding sites for the flies than the hard crumbly faeces deposited on sparse dry grazing.
- Hence irrigation of pasture may result in an increase of stephanofilariosis.
- Though the lesions subside in cooler weather, the **damage to the hide** is permanent and may result in considerable **economic loss**.
- Milk yield may be severely diminished from the pain of the lesions and the irritation of cattle by the flies.

## Life Cycle

- The fly vectors are attracted to the open lesions in the skin caused by the adult parasites, and ingest the microfllariae ($L_1$) in the exudate. Development to $L_3$ takes about three weeks, and the final host is infected when the flies deposit larvae on normal skin.

## Pathogenesis and Clinical Signs

- Lesions begin to appear within two weeks of infection.
- In the case of *S. stilesi*, the flies congregate on the shady underside of the abdomen and it is in this area that the most severe damage occurs;
- *S. assamensis* lesion is found on hump which is commonly termed 'hump sore'.
- In all species, the lesions are usually localized to the preferred biting areas of the vectors.

- The skin is at **first nodular**, but later there is **papular eruption** with an exudate of blood and pus. In the centre of the lesion there may be **sloughing of the skin**, but at the **margin** there is often **hyperkeratosis**.
- The condition is essentially an **exudative, often haemorrhagic, dermatitis** which attracts the fly vectors.

## Diagnosis

- Though adult worms and microfilariae are present in the lesions they are often scarce and many scrapings prove negative. Diagnosis is therefore usually presumptive in endemic areas, and is based on the appearance and site of the lesions.

## Treatment

- Organophosphorus compounds applied topically as an ointment have proved effective.
- Avermectins are likely to be effective.

## Control

- This is rarely feasible because of the ubiquity of the vectors, but would have to be based on the use of insecticides or repellents.

# Dirofilaria

- The adults (Macrofilaria) which are found in the **right side of the heart** and **adjacent blood vessels** of dogs are responsible for a debilitating condition known as **canine heartworm disease**.
- Although primarily a problem of warm countries where the mosquito intermediate host abounds, the disease has become much more widespread in the past decade and the problem in North America is now so extensive that special heartworm clinics have been created.

| Major Species | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| ***Dirofilaria immitis*** | Dog, occasionally cat and rarely man | Mosquitoes | Cardiovascular system; | **warm-temperate and tropical zones throughout the world** |
| ***Dirofilaria repens*** **(Little pathogenic significance)** | **Dog, cat** | **Mosquitoes** | **adults in S/c tissues and the microfilariae in the blood and lymph** | **Mediterranean basin, the Middle East, Africa and Asia** |

## Identification

### Gross:

- Long slender worms **20-30 cm long**.
- Female up to 30cm and Male upto 15cm long
- The male tail has the typical loose spiral common to the filarioids.
- .Immature adult ($L_5$): 1-5cm long.
- The size and site are diagnostic for *D. immitis*

### Microscopic:

- The **microfilaria ($L_1$, motile)** in the blood **are not ensheathed** and are 307-332 µm in length by 6.8 µm wide.
- They have a tapered anterior end and blunt posterior end. (in case of *Dipetalonema reconditum*, posterior end is coiled)

### Location of Macrofilarae (adult):

- Right ventricle of heart (Right atrium)
- Pulmonary artery
- Posterior Venacava
- Rarely also in the other blood vessel or organ (eye, CNS)

### Location of Microfilaria:

- Peripheral circulation

## Epidemiology

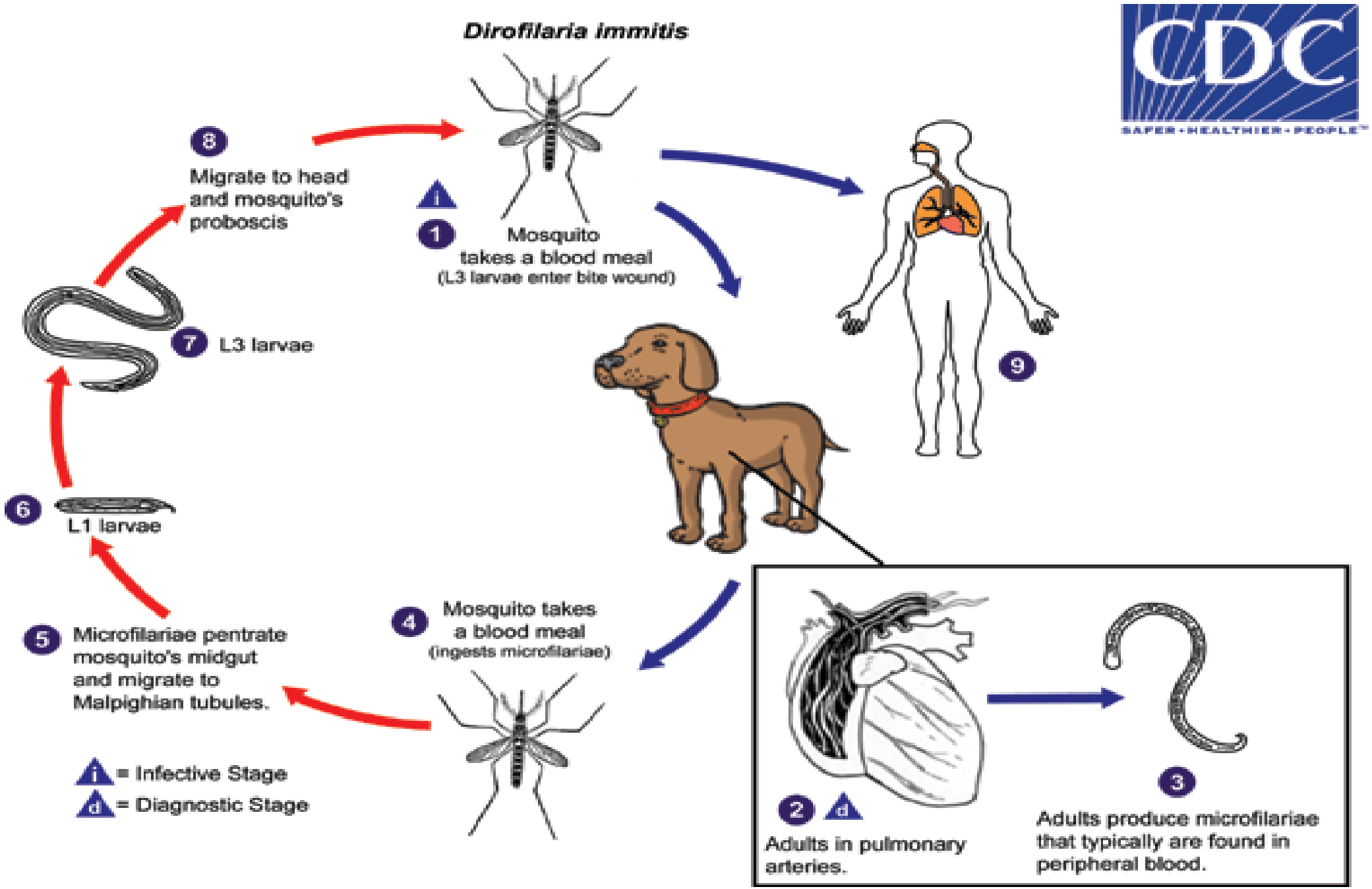
- Prevalent in warm temperate and tropical zones throughout the world
- Sporadic in Southern Europe
- It is only found in imported dogs in Britains.
- The important factors in the spread of Heartworm diseases includes

## A. Those affecting the Host:

- **Host factors** include a high density of dogs in areas where the vectors exist,
- The lengthy patent period of up to five years during which time circulating microfilaria are present, and the lack of an effective immune response against established parasites.
- Infection usually occurs in Dogs older than one year of age. Although intrauterine transmission of microfilaria to pups of infected bitches also possible
- Seasons: Features of Microfilariaemia are the seasonal , highest levels in Spring and Summer.
- Period: Features of microfilaraemia are diurnal periodicities (evening or night). Maximum number of larvae in the peripheral blood from 1800 to 2200 hour (synchronously with the period of diurnal mosquito's activity). Minimum numbers around 600hours.

## B. those affecting the Vectors:

- The ubiquity of the mosquito intermediate hosts,
- Their capacity for rapid population increase and the short development period from microfilaria to $L_3$.
- Larval development ceases at below $18^oC$


Dirofilaria immitis
CDC
SAFER • HEALTHIER • PEOPLE™
8
Migrate to head and mosquito's proboscis
i
1
Mosquito takes a blood meal
(L3 larvae enter bite wound)
9
7
L3 larvae
6
L1 larvae
5
Microfilariae pentrate mosquito's midgut and migrate to Malpighian tubules.
4
Mosquito takes a blood meal
(ingests microfilariae)
i = Infective Stage
d = Diagnostic Stage
2
d
Adults in pulmonary arteries.
3
Adults produce microfilariae that typically are found in peripheral blood.

## Pathogenesis

- Pathogenesis is associated with the adult parasites.
- Many dogs infected with **low numbers** of *D. immitis* show **no apparent ill effects**
- In **heavy infections** that circulatory **distress occurs**, primarily due to **obstruction** to normal blood flow leading to **chronic congestive right-sided heart failure**.
- The presence of a mass of active worms can cause an **endocarditis** in the heart valves and a **proliferative pulmonary endarteritis (**Inflammation of the inner lining of an artery**)**, possibly due to a response to parasite excretory products.
- In addition, **dead or dying worms** may cause **pulmonary embolism**. After a period of about nine months the effect of the developing **pulmonary hypertension** is **compensated by right ventricular hypertrophy** which may lead to **congestive heart failure** with the usual accompanying signs of **oedema and ascites**. At this stage the dog is listless (Lethargic) and weak.
- A mass of worms may lodge in the **posterior vena cava** and the resulting **obstruction** leads to an acute, sometimes fatal, syndrome known as the **venacaval syndrome**. This is characterized by **haemolysis, haemoglobinura, bilirubinaemia, icterus, anorexia and collapse**. Death may occur within 2-3 days.
- Very occasionally there is **blockage of the renal capillaries** by microfilariae leading to a **glomerulonephritis**, possibly related to the deposition of immune complexes.

**Clinical Signs**

- In heavily infected dogs:
  - Listless, gradual loss of condition and exercise intolerance.
  - Chronic soft cough with haemoptysis
  - Become dyspnoeic, oedema and ascites during later stages of disease.
- In lightly infected dogs:
- Poor performance during periods of sustained exercise.
- Acute vena caval syndrome is characterized by
  - Haemoglobinuria, icterus and collapse.
- Lighter infections in working dogs may be responsible for

**Diagnosis**

- Clinical signs of cardiovascular dysfunction
  - Radiography: thoracic radiography may show the thickening of the pulmonary artery
  - Angiography: may also be used to demonstrate more clearly the vascular changes
- Wet smear : by observing the movement of the microfilaria
- Thin/Thick smear followed by Giemsa staining
- Modified Knott's techniques: (Mainly for Differential diagnosis)
  - Take 1ml blood +9ml 2% formalin and centrifuge 5minutes in 1500rpm
- Add 0.1% methylene blue with sediment and observe under microfilaria

| *Dirofilaria immitis* | *Dipetalonema reconditum* |
|---|---|
| • Microfilaria has tapered anterior end and blunt posterior end<br>• 307-332 µm in length by 6.8 µm wide. | • Microfilaria has blunt head and a hooked posterior end<br>• <300 µm in length |

Note: More precise differentiation may be achieved by using histochemical stains for acid phosphatase activity. *D. immitis* show distinct red acid-phosphate positive spots at the excretory pore and anus, while D. reconditum stains pink overall.

Mcropore filter techniques:

Blood forced through micropore filter; stainded and examined.

6. Buffy Coat Method

7. Immunodiagnostic tests:

**Antigen detection ELISA:** Can identify the mature worm antigen to confirm the most mature infection but not microfilaraemia.

**Antibody diction ELISA:** Not Realiable

## Treatment

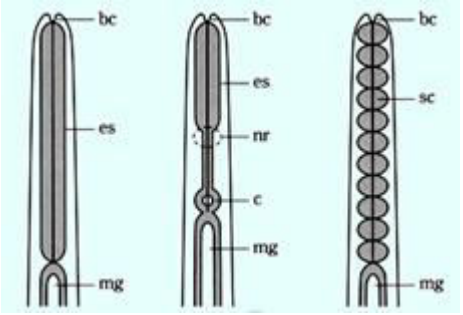
- Treatment should not be undertaken without a physical examination of the dog and an assessment of heart, lung, liver and kidney function. Where these functions are grossly abnormal it may be necessary to give prior treatment for cardiac insufficiency.
- The usual recommendation is that infected dogs are first treated intravenously with thiacetarsamide twice per day over a three day period to remove the adult worms;
- Toxic reactions are not uncommon following this treatment due to the dying heartworms and resultant embolism; activity of the dog should be restricted for a period of 2-6 weeks. This drug should be used with extreme care.
- A further treatment with a different drug is then given six weeks later to remove the microfilariae which are not susceptible to thiacetarsamide treatment. Several drugs are now available for this purpose; the traditional one was dithiazanine and either this or levamisole given orally over a 10-14 day period has proved effective.
- The avermectins are also highly efficient against microfilariae With all of these drugs there is a risk of adverse reactions to dying microfilariae.
- In some severe cases, heartworms have been removed surgically rather than risk adverse reactions following drug therapy.
- Following treatment it is usual to place dogs on a prophylactic programme.

# Superfamily: Trichuroidea

**Common features:**

Stichosome esophagus which is composed of a capillary like tube surrounded by a single column of cells.

Infective stage is $L_1$




**Genus:** 1. Trichinella-viviparous-Direct L/C –Larva in muscles

2. Trichuria-oviparous-Direct L/C - Egg in feaces

3. Capillaria-oviparous-Direct/Indirect L/C-Egg in feaces

4. Trichosomoides

| Major Species | F/H | I/H | Location/Site | Distribution |
|---|---|---|---|---|
| ***Trichinella*** | | | | |
| ***Trichinella spiralis*** | **Mammals-Pig, Man, Rat, Dog** | **Same animal** | **Small intestine** | **Worldwide** |

| *Trichuris* | | | | |
|---|---|---|---|---|
| ***Trichuris ovis*** | Sheep,Goat | - | **Large Intestine: Caecum & Colon (Embeded in the mucosa by anterior end)** | **Worldwide** |
| ***T. globulosa*** | Cattle | | | |
| ***T. suis*** | Pig | | | |
| ***T. vulpis*** | Dog,Fox cat | | | |
| ***T. serrata*** | Cat | | | |
| ***T. trichuria*** | Man | | | |
| ***T. skrjabini*** | Sheep, Goat & Camel | | | |
| | | | | |

## *Trichinella* (Muscle worm)

**Species**: *Trichinella spiralis*

**Host**: Mammals-Pig, Man, Rat, Dog

**Site:** Adult worm-Between the villi of Small intestine (After fertilization, the males die while the female burrow into the glandular crypts of the mucosal epithelium in the proximal part of the SI).

Larvae-

- Skeletal musle, Diaphragmatic muscle , Intercostal muscle and Masseter muscle
- Tongue, larynx, eye-muscle

**Distribution:** Worldwide

**Morphology:**

**Adult**:

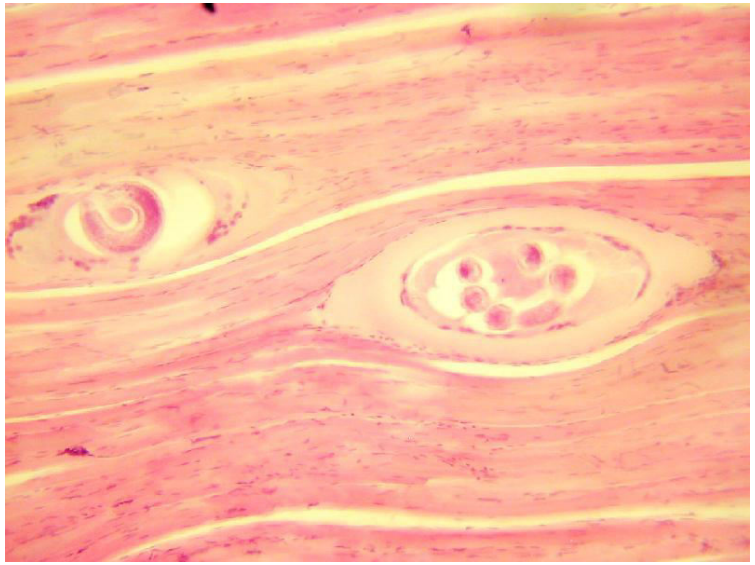
- Horn like tiny worms.
- Male is about 1 mm long & Female is 3mm long
- In male, the esophagus is at least one third of the total body length & the tail has two small cloacal flaps, but no spicule.
- In female, the uterus contains developing larvae.

**Larvae**:

- It presents in muscle as coiled appearance.

**Cyst**:

- Lemon shaped
- 0.3-0.8x0.2-0.4 mm in size
- Usually transparent
- The wall of old cysts may become calcified



**Disease**: Thrichinellosis/Muscle worm disease

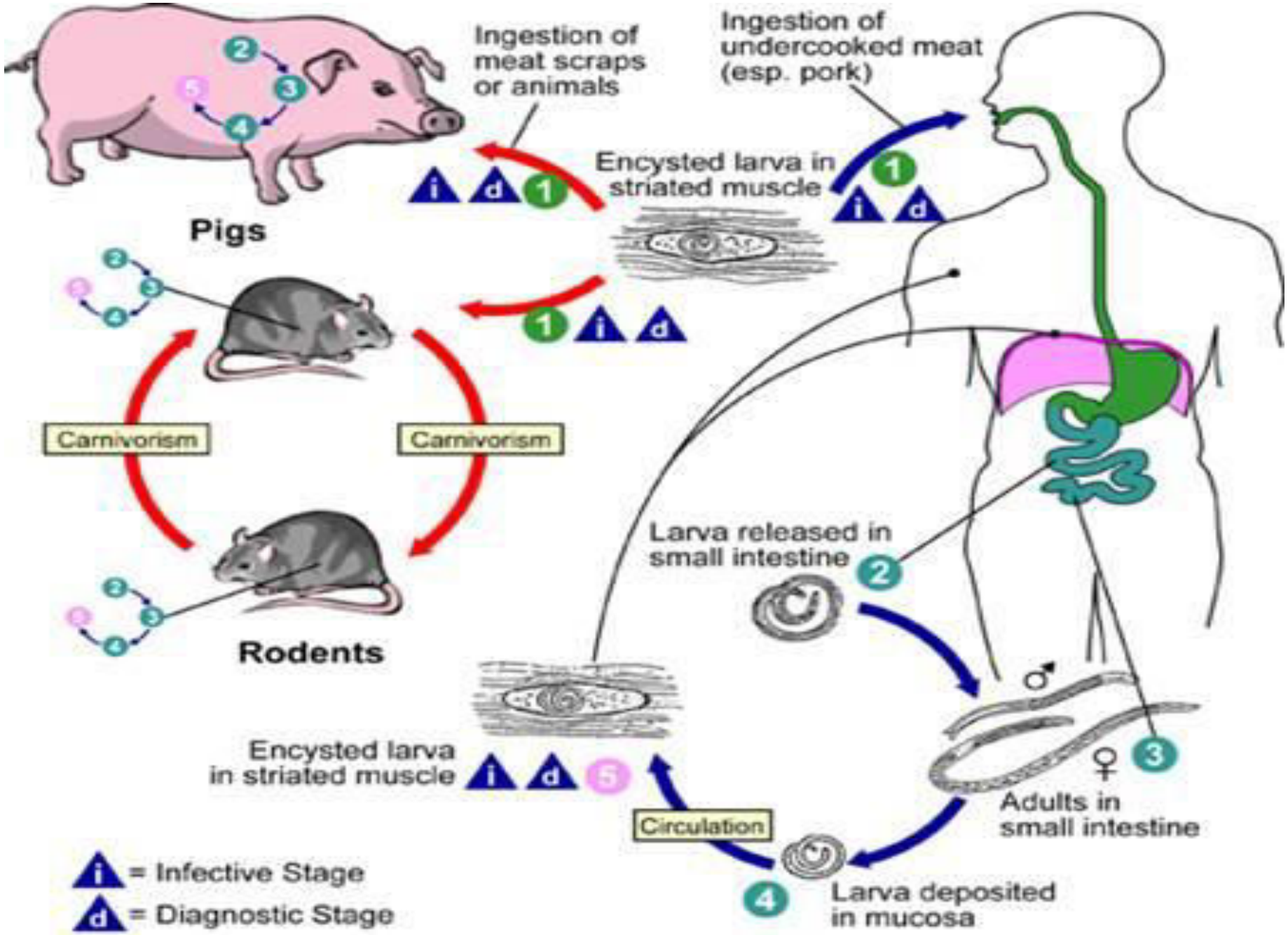
- Veterinary significance is moderate
- **Public health significance/Zoonotic significance** is high (worldwide zoonosis)

**Epidemiology**:

- It is a typical Feral (wild) & worldwide endemic disease.
- There are sylvatic and synanthropic cycle.
- In sylvatic or Feral cycle-
  - The infection is transmitted within some wild life populations (e.g, rats) and ascends in food chains (fox in UK).
  - Carnivores and omnivores mammals are particularly vulnerable.
  - But herbivores can be infected if they accidentally ingest by infected meat (e.g.- remain of dead mouse in hay)
  - In this way, from the natural sylvatic cycle, the infection is transformed to the Synanthropic cycle.

**In synanthropic/Domestic cycle**-

- The main source of infection for pigs is uncooked swill containing kitchen waste.
- Humans are infected by eating uncooked pork or pork products (or, less commonly, other infected material)


Ingestion of meat scraps or animals
Ingestion of undercooked meat (esp. pork)
Encysted larva in striated muscle
Pigs
Carnivorism
Carnivorism
Rodents
Larva released in small intestine
Encysted larva in striated muscle
Adults in small intestine
Circulation
Larva deposited in mucosa
= Infective Stage
= Diagnostic Stage

**Pathogenesis and Clinical Signs**:

**Pig**:

- Infection in domestic animals (pigs) is invariably light and clinical signs do not occur.

**Rat, Dog & Cat:**

- Much more sensitive to the toxic & mechanical damage presented by the infection-
  - Catarrhal enteritis
  - Diarrhoea
  - Acute myositis
  - Myalgia

**Human:**

- Ingestion of 500 larvae may result in severe /**acute Trichinollosis**.
- Internal disorders: Diarrhoea, Vomition, Ascitis
  - High fever
  - Headache
  - Marked blood eosinophilia
  - Muscular pains
  - Sweating
  - Ascitis
  - Oedema of the eyelids, periorbital & facial region
  - Toxic neurological symptoms
  - Cardiomyopathy
  - Paralysis of the respiratory muscle
  - Death (at 4-7 weeks)

**Diagnosis:**

1. Clinical history
2. Clinical signs (human)
3. Biopsy sample (e.g in pig-tongue)
4. Muscle digestion in pepsin/HCL & sediment examined microscopically.
5. Meat inspection includes direct squash or pressure glass technique.
6. Immunodiagnostic test-ELISA, FAT, HA

**Treatment:**

- Rarely called for treatment, infected carcasses should be condemned during meat inspection.
- Thiabendazole: 25 mg/kg twice daily for 5-10 days.
- Mebendazole: 200mg three daily for 10 days.
- Fenbendazole, Cambendazole

**Control**:

- The main principles of control are –
- Pigs must not be allowed to scavenge
- Meat inspection
- Destruction of infected carcasses
- Protects human health
- Reduces risk of contaminated swill-pig
- Boil swill adequately (compulsory in UK)
- Pork products must be well cooked or otherwise treated before human consumption.

# Some species of Trichenella:

| Species | Host |
|---|---|
| *Trichenella spiralis* | Mammals,pig,Rat,Man |
| *T. nativa* | Wild carnivores,Senl,White bear |
| *T.nilsoni* | Wild carnivores & Omnivores |
| *T.britovi* | Wild carnivores,Fox,Wild boars, Horse,Man |
| *T.pseudospinalis* | Mammals,Bird<br>(Lacks the ability to form a nerve cell remain unencysted in the muscle) |

**Trichuris** (whip worm)

(Syn.Trichocophalus)

| Species | Host | Location |
|---|---|---|
| *Trichuris ovis* | Sheep,Goat | Large Intestine: Caecum & Colon (Embeded in the mucosa by anterior end) |
| *T. globulosa* | Cattle | |
| *T. suis* | Pig | |
| *T. vulpis* | Dog,Fox cat | |
| *T. serrata* | Cat | |
| *T. trichiuria* | Man | |

**Distribution**: Worldwide

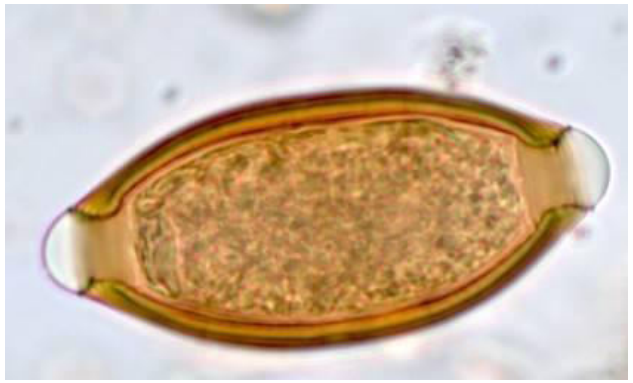
**Identification**:

**Adult**:

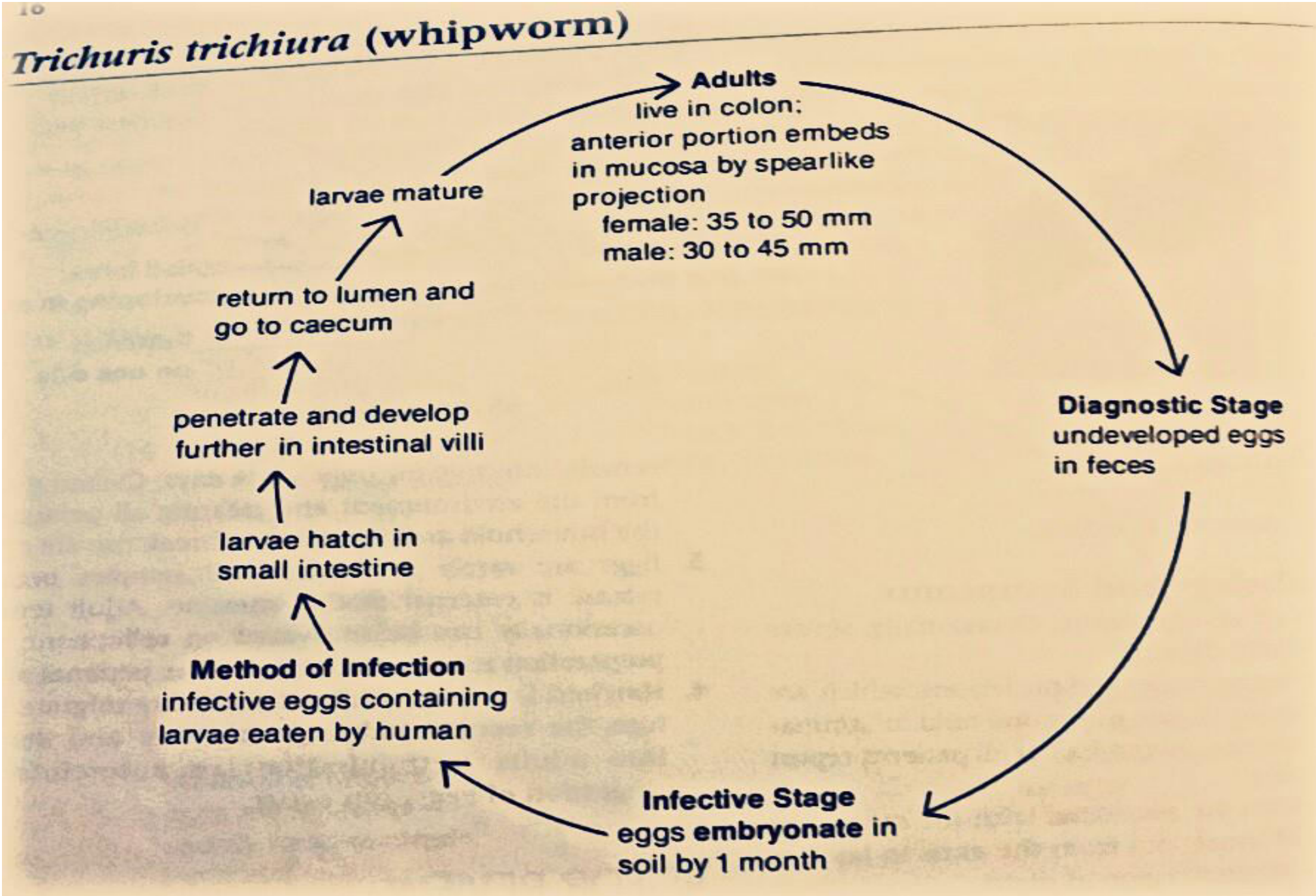
- 3-8 cm long, white worms
- The anterior part of the worm is tapered and whip-like and the posterior part is broad and handle like(Hence the name **"whipworm"**
- The tail is curved in female and coiled in males.
- The male tail possesses a single spicule in a sheath, which is armed with fine cuticular spines(flower like)
- In female, vulva is situated at the beginning of the wide part of the body.

**Egg**:

- Barrel or lemon shaped 50-80 in size.
- A thick smooth shell.
- Conspicuous opercula(plugs) at both ends (protrude markedly)
- Yellow or brown in color with dark content
- In the single cell stage passed in feaces.
- **Disease**: Trichuriosis/Whip worm disease.
- **Vet significance:** Moderate/High
- **Zoonotic significance**: Moderate/High



# *Trichuris trichiura* (whipworm)



## **Pathogenesis and Clinical Sign**:

- Adult are haematophagus and more pathogenic than mucosal larvae-
- Non pathogenic in ruminants and may be pathogenic in Pig, Dog & Man.
- Light infections are asymptomatic, heavier infection may cause clinical disease especially in young animals
- Possible symptoms are acute or chronic, characterized by:
  - Catarrhal or hemorrhagic **typhilitis**
  - Colitis/Enteritis
  - Watery diarrhea
  - Hypoprotenemia
  - Anaemia, weakness and death
- In severe cases, the mucosa of the large intestine is inflamed
  - Hemorrhagic with ulceration & formation of corpous-diptheritic membranes.
  - Can provoke severe diarrhea or dysentry by providing a portal of entry for microbial organisms (particularly spirochaetes)
  - (In adult man, symptomatic infection rarely occur, more common in small children, especially in the tropical/subtropical regions)

## **Diagnosis**:

1. Clinical history
2. Clinical signs
3. Coproscopy-Finding of characterstics egg in faeces.
4. Spontaneously dislodged adult worms may be recongnized in the faeces.
5. Necroscopy finding.

## **Treatment:**

Anthelmentic therapy-

Eg. Pig-Mebendazole: 30 mg/kg food for days orally

Cattle-Fenbendazole: 7.5 mg/kg for single dose orally

Sheep-Fenbendazole: 5 mg/kg for single dose orally

Dog-Fenbendazole: 50 mg/kg for 3 days orally

## **Control:**

1. Prophylactic treatment
2. Good hygienic management

(Houses should be cleaned & disinfected or sterilized by wet or dry heat)

## **Epidemiology**:

The most important feature is the longevity of the eggs which can survive 3 or 4 years as a reservoir of infection in piggeries or kennels. On pasture, this is less likely since the eggs tend to be washed into the soil.

# Capillaria (Hair Worm)

| Species | Host | Site | L/C |
|---|---|---|---|
| ***Caplliana contorta*** | Domestic & wild Birds | Oesophagus, Crop | **Direct/ Indirect** |
| ***C. annulata*** | Do | Do | **Indirect** |
| ***C. obsinata*** | Domestic fowl. Turkey, Pigeon | SI | **Direct** |
| ***C. caudinflata*** | Domestic Fowl, Turkey Goose | SI | **Indirect** |
| ***C. bursata*** | Domestic Fowl, Turkey Pheasant | SI | **Indirect** |
| ***C. retusa*** | Gailiform & Anseri form Birds | Caccum | **Indirect** |
| ***C. Anatis*** **(Bargladesh)** | **Duck, Goose, Domestic fowl, G. fowl, Turkey** | **Caecum** | **Direct** |

## Mammals:

| | | | |
|---|---|---|---|
| ***C. bovis*** | **Large and small Ruminants** | **SI** | **Direct** |
| ***C. longipes*** | Small Ruminants | SI | **Direct** |
| ***C. aerophila*** | Fox(Dog, Cat) | SI | **Direct** |
| ***C. plica*** | Fox (Dog, Cat, wolf) | Unitary bladder, pelvis of kidney | **Indirect** |
| ***C. feliscati*** | Cat | Urinary bladder | **Indirect** |
| ***C. hepatica*** | Rodent, Leopards (Dogs Cat, man) Natural parasite | Liver Parenchyma | **Direct** |
| ***Trichosomoides crassicauda*** | Rat | Urinary bladder | **Direct** |
| ***C. bilobata*** **(Bangladesh)** | Cattle, Goat | Abomasums, SI | |
| ***C. Philippinensis*** | **slam** | **SI** | **Indirect** |

**Distribution:** World wide

**Identification:**

**Adult:**

- Very fine filamentous hair like worms between 1-5 cm long, hence it is called Hair worm
- Stichosome some esophagus occupying half the body length.
- Males have a long thin specule & often possess a primitive bursa like structure.
- The front end is slightly thinner than the tail end.
- *C. contorta* , Male : 15-25 mm long
- Female: 27-38 mm long
- *C. annulata,* Male: 15-25 mm long
- Female: 37-80 mm long
- *C. caudinflata, C. obsinata, C. anatis:* 6-35mm long
- The parasites anterior end is buried in the mucosa.


**Egg:**

- Barrel or lemon shaped 50-75µm in size.
- Colorless, with thick, slightly striated shell (in trichuris it is not present)
- Polar plug at both ends (Operculated)
- When passed in the faces it contains a single cell that is pale & granulated.

**In case of Direct life cycle:**

Adult worms in the Predilection site of the Host

↓

Adult female lays eggs in the Predilection site

↓

Eggs in the faces of the host.

↓

$L_1$ develop within the egg in environment

↓

The other potential host gets infection by ingesting the egg containing $L_1$.

↓

The eggs hatch at their predilection site develops into $L_2$, $L_3$, $L_4$& $L_5$ and become adult

↓

Adult female lay eggs

**In case of indirect life cycle:**

Adult worms in the predilection site of the F/H

↓

Adult lays eggs in the site

↓

Eggs in the feces of the F/H.

↓

The egg ingested by I/H, Earthworms in 2-4 weeks

↓

$L_1$ developed within the Earthworms

↓

The potential F/H gets infection by ingesting the Earthworm

↓

$L_1$ develop to Adult in their predilection site in the F/H.

↓

Adult female lays eggs.

**PPP:** In Avian species: 3 to 4 weeks

In Mammalian species: 6 wks

In case of ***C. hepatica*:**

Adult worms in lever parenchyma of the host.

↓

Adult female lay eggs in the site

↓

The eggs are encapsulated by host tissue in the liver parenchyma. (Thus eggs can't normally be released from the host)

↓

The potential host is infected by the ingestion of infected liver

↓

Then, the eggs will be disseminated through the faeces of the animals.

- Eggs can be freed by decomposition of after the death of the infected host.
- In Mice, embrocated eggs adhering to their fur or feet which are being ingested during preening.

**Pathogenesis and Clinical Sings:**

**In Poultry:**

- In light infections
- May be asymptomatic
- Light thickening of Mucosa of crop & Esophagus
- Lowered weight gain & egg production
- In heavy infections: usually in young birds are infected
- Marked thickening of their infected mucosa with catarrhal or diphtheritic inflammation and sloughing of the mucosa of the Esophagus, crop, Sl.
- Inappetence, emaciation, diarrhea (may be bloody) anemia & death.
- *C. Obstinate* may cause high n mentality in fowl & pigeons.

**Mammals:**

*C. aerophila*:

- Rhinotracheitis
- Bronchitis

*C. plica:*

- Low Pathogenic significance
- secondary bacterial infections may cause cystitis ( urinary bladder infection)

*C. hepatica*:

- Irregular tacks & granulomas formed around egg masses in the liver parenchyma
- In severe infections acute or sub acute hepatitis, cirrhosis and ascites.
- Death may occur.

**Diagnosis:**

- Cl. History
- Cl. Sings
- Charactionstics eggs in feaces
- Microscopic exam of mucosal scrapings squeezed between 2 glass slides.
- Contents of effected organs or intestinal larvae should be washed through fine sleeve, retained material resuspended in water or saline & examined against a black background. Squash preparation from liver lesions can be investigated microscopically
- Necropsy findings.

**Treatment:**

- Levamisole: With DW
- Benzimidazoles: With feed

- *Capillonia hepatica:*
- Mebendazole, Oxfendazole: 12.5 /kg, 5 day course of onal daring.

**Control:**

- Regular deforming
- Moving the animals/birds to clean ground after treatment.
- Control of earthworms & prevention of contact with earthworms e.g. housing the birds after anthelmintics medication.

**Super family:** Dictophymatoidea

**Genus:** Dioctophyma

**Species:** *Dioctophyma renale* (Kidney worm)

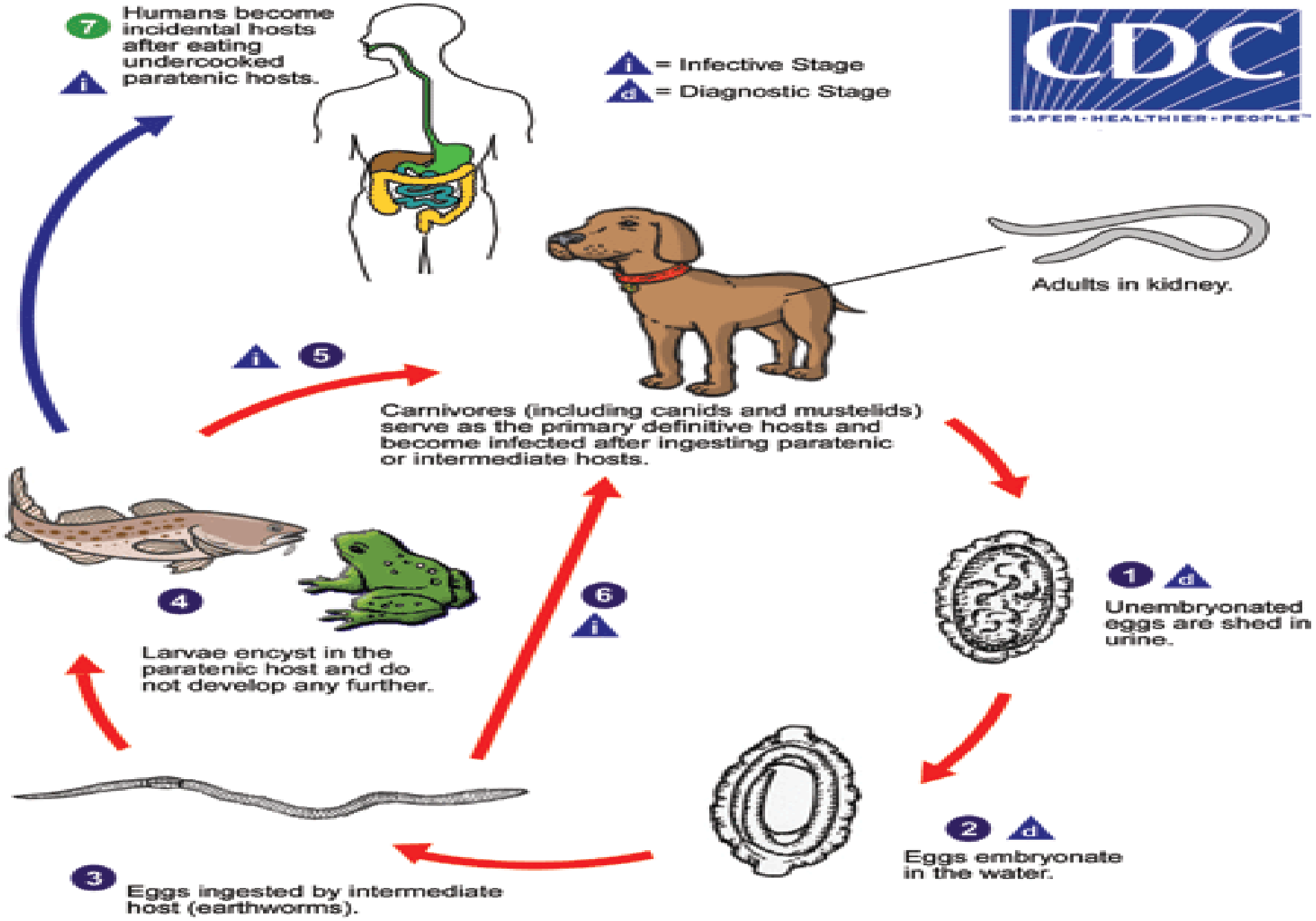
**Common name:** Kidney worm/Giant kidney worm/Largest parasite of nematodes

**Disease:** Dioctophymatoidoses/ Dictophymosis

| Species: | Final Host (F/H) | Intermediate Host (I/H) | Location in F/H | Distribution |
|---|---|---|---|---|
| *Dioctophyma renale* | Dog, fox, mink, occasionally horse/cattle/man. | Aquatic annelids. *Lumbriculus variegatus* | Kidney parenchyma | Temperate and sub arctic areas |

**Identification:**

- Largest nematode of domestic animals.
- Females are 60-130 cm long & 1.0 cm wide.
- Worms has blood red color.
- Male has a cup-shaped copulatory bursa without bursal rays and a single spicule.
- Eggs are barrel shaped, 60-80µm in size, has a thick brownish yellow pitted shell and bipolar pulgs


7 Humans become incidental hosts after eating undercooked paratenic hosts.
i
i = Infective Stage
d = Diagnostic Stage
CDC
SAFER · HEALTHIER · PEOPLE
Adults in kidney.
i 5
Carnivores (including canids and mustelids) serve as the primary definitive hosts and become infected after ingesting paratenic or intermediate hosts.
4
Larvae encyst in the paratenic host and do not develop any further.
6
i
1 d
Unembryonated eggs are shed in urine.
2 d
Eggs embryonate in the water.
3
Eggs ingested by intermediate host (earthworms).

**PPP:** 2years

**Pathogenesis:**

- Destruction of the infected kidney. Usually Unilateral (specially Rt kidney) infection is occured
- Destruction of kidney parenchyma, leaving only the capsule as a sac containing the worms.
- Usually no clinical sign as other kidney serve the functions.
- Rarely, the worms may occur in the abdominal cavity and in the subcutaneous connective tissue.
- Developing and migrating worms may cause peritonitis, destruction of the liver tissue, atrophy and fibrosis of the kidney, secondary hydronephrosis, associated with haematuria, uremia or not apparent symptoms because of unilateral infection.

**Clinical Signs:**

- Usually completely asymptomatic, even when one kidney has been completely destroyed.
- The main sings are dysuria (difficult micturation) with some haematuria especially at the end of micturation.
- In few cases there is lumbar pain/Flank pain
- Retention of urine & death from uremia when bladder or urethra is blocked.
- In man, renal colic, pyuria, haematuria may occur.

## Diagnosis:

- Clinical History
- Clinical signs
- Characteristic eggs in urine
- Size and predilection is also sufficient for identification.

## Treatment:

- Surgical removal of worms.

## Control

- Avoid raw fish from the diet.

**Super Family: Dracunculoidea**

Genus: Dracunculus

Disease: Guinea worm Disease /Dracunculosis/Madina worm disease/ Dracontiasis

It is a neglect disease especially in remote rural areas.

| Species: | Final Host (F/H) | Intermediate Host (I/H) | Location in F/H |
|---|---|---|---|
| ***Dracunculus medinensis*** **(Guinea worm/Medina worm/Little Dragon in Medina)** | Man, Dog, Horse, Cattle | **Copepod Crustacean e.g. Cyclops spp.** | **Sub-cutaneous connective tissue (Adult)** |
| ***Dracunculus insignis*** | **Dog, Wild Carnivores, Mink, Ferret** | | |

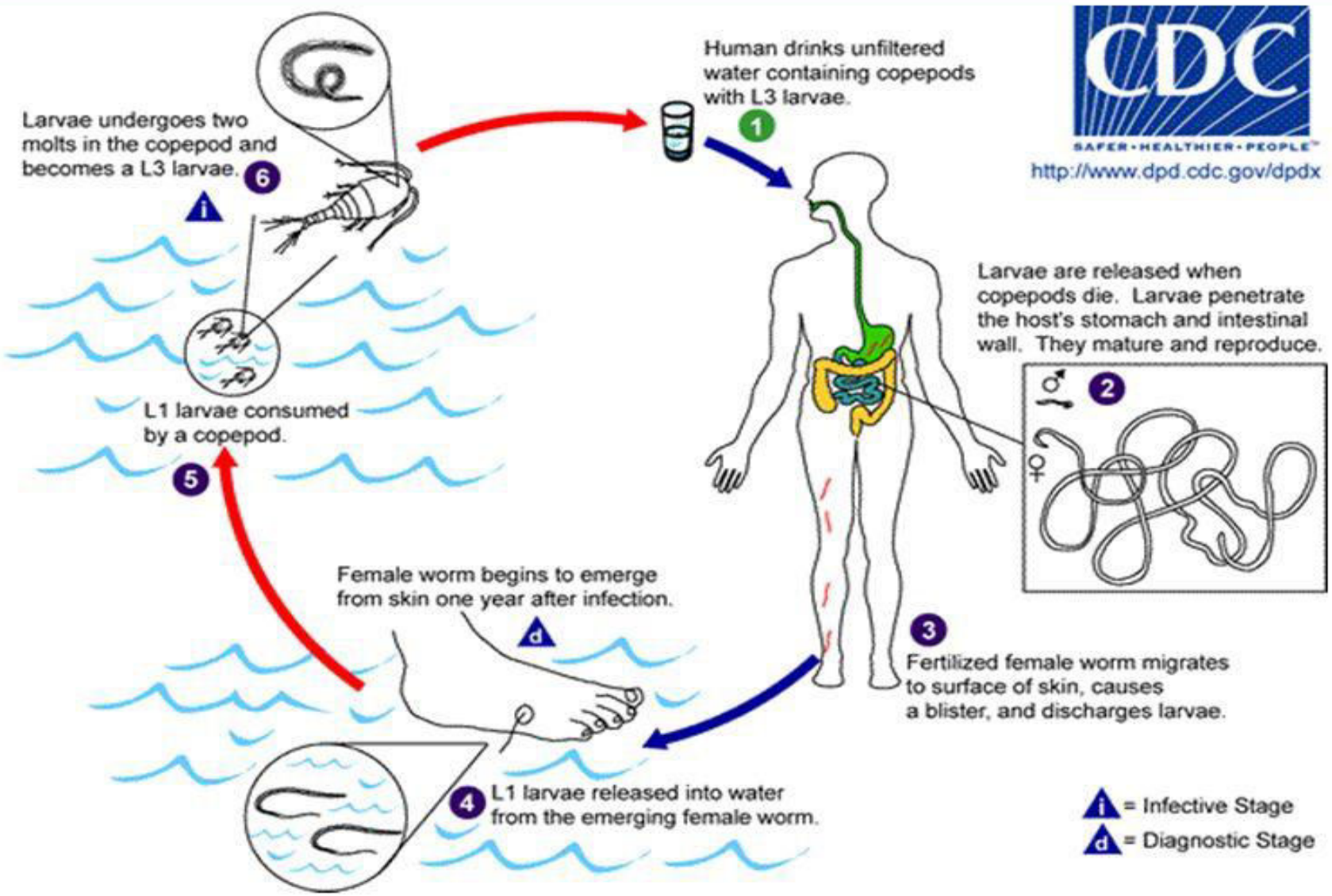
**Morphology:**

- Largest nematode in Man and Animals.
- Adult: Anterior end with Helmet
- Males: 3-4cm and Female : Up to 80cm in length

**Distribution:**

- Formerly, frequent in rural areas of Tropical Africa, Arabia, Near-East, India, Pakistan etc.
- Still highly prevalent in Sudan

# Dracunculus medinensis



## Pathogenesis and Clinical signs:

- Adult worms developed in deep connective tissue.
- Gravid female move to S/c tissues of the extremities of the infected host at 8-10months post infection without causing appreciable signs of disease.
- At the anterior end of the worm, a **papule** developed followed by **painful blister** especially on the lower limbs
- When blister come in contact with water, they burst resulting release of $L_1$ larva from the **ulcer masses**.
- Secondary infection

## Diagnosis:

- Clinical History
- Clinical Signs: (Blisters on skin)
- Examination of Blister fluid under microscope for $L_1$ larva.
- However, Adult worm can be seen in naked eyes in the ulcerated area.

## Treatment:

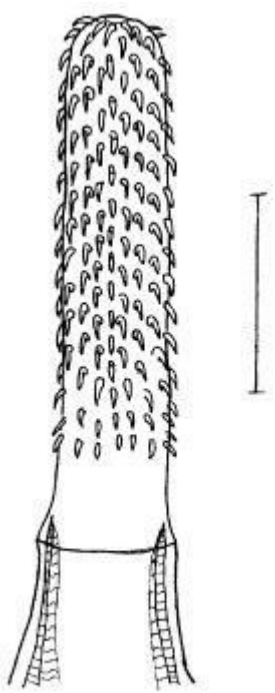
- There is no medicine (Anthelmintic therapy) or vaccine to treat or prevent the disease
- Gradual removal of the worm alive from the ulcerated area wrapped by a piece of gauze or rolling a strict (rounded) on it. This is long, painful process can take up to a month.
- Anthelmintic therapy, can be used thiabendazole @ 50mg/kg body wt. for 2-3days
- Secondary infection should be checked

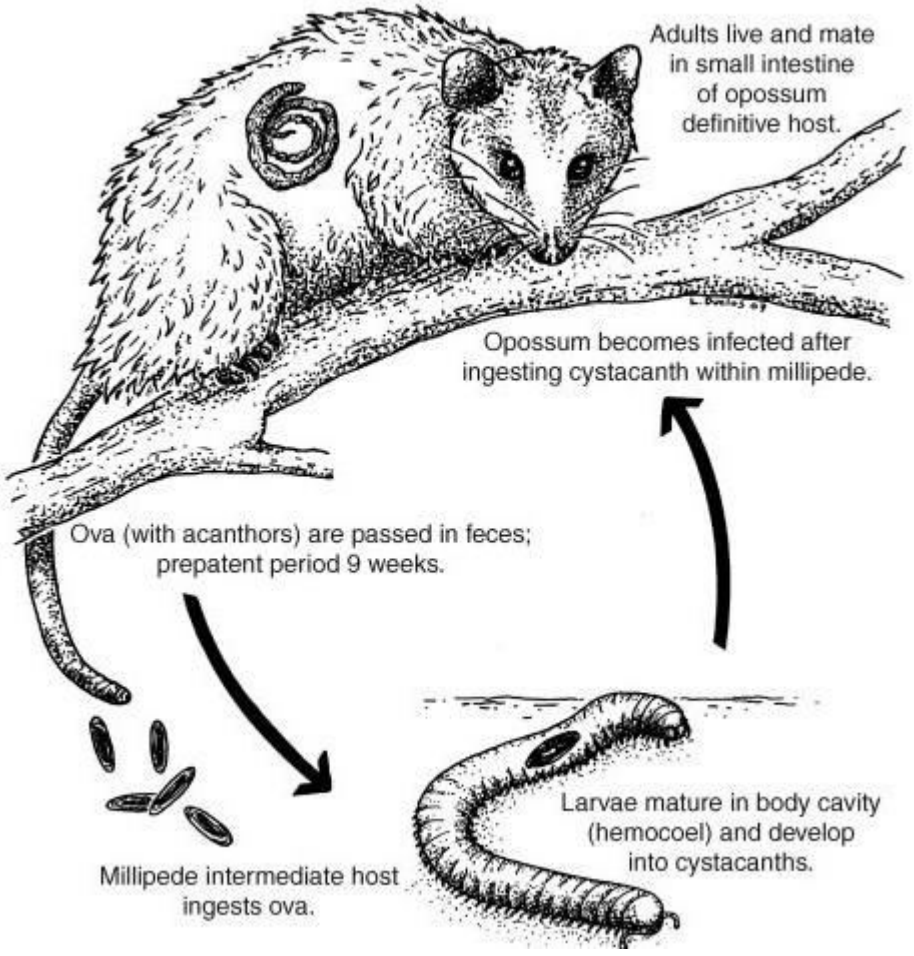
**Prevention and Control:**

- Educating people on the pattern of transmission of the disease
- Provision of safe water supply (underground water)
- Removal of cyclopes form drinking water simply by sieving it through a double layer of ordinary shirting material/fine –mesh cloth like nylon.
- Prevent persons/animal with an emerging Guinea worm from entering ponds and well used for drinking water
- This is the only parasitic disease of human that may be completely eradicated in the near future.

# Phylum: Acanthocephala

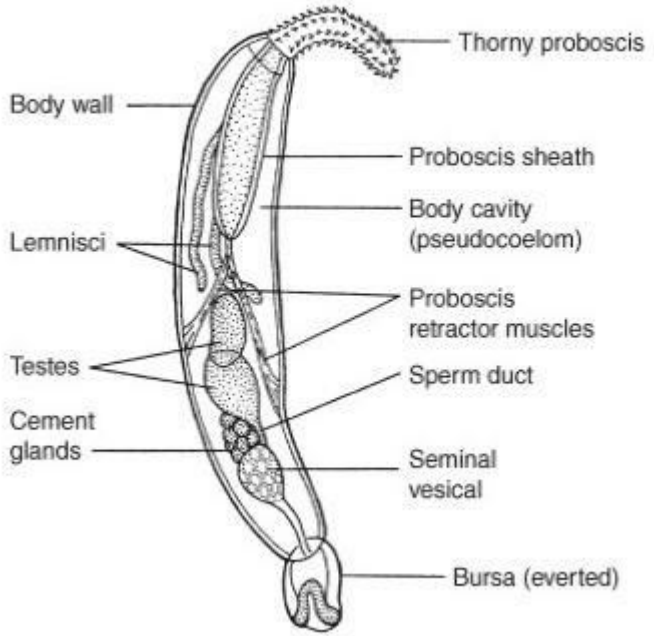
- This is a separate phylum, closely related to the Nematoda, which contains a few genera of veterinary importance.
- They are generally referred to as **'thorny-headed** worms' due to the presence **anteriorly hook-covered proboscis** and most are parasites of alimentary tract of vertebrates.
- The hollow probosis armed with recurved hooks, which aid in attachment, is retractable and lies in a sac-**Proboscis receptacle**.

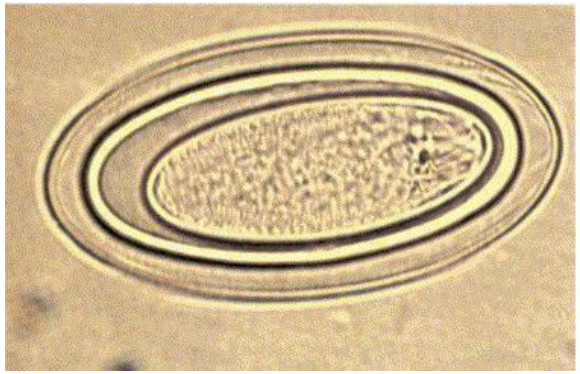


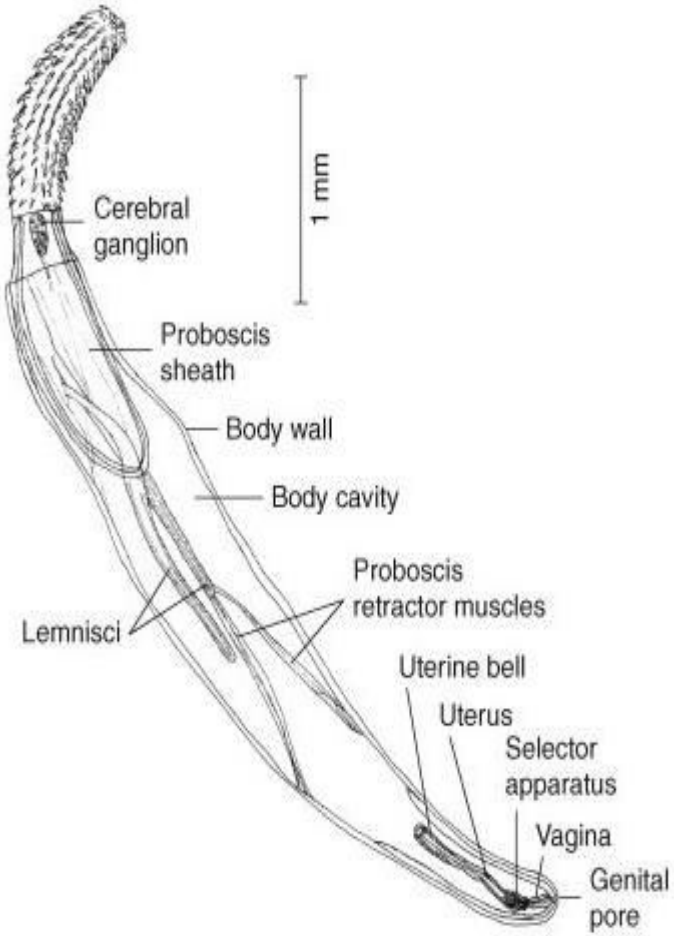


- There is no alimentary canal, absorption taking place through the thick cuticle, which is often folded and invaginated to increase the absorptive surface.

- The sexes are separate, male are being much smaller than females. Posteriorly, male has a muscular bursa and penis and after copulation, eggs, discharged by ovaries into the body a of the female, are fertilized and taken up by a complex structure called **the uterine bell** which allow only all mature eggs to pass out. These are spindle-shaped thick-shelled and contain a larva which has an anterior circlet of hooks and spines on its surface and is called an acanthor.





# Macracanthorhynchus

| Species: | Final Host (F/H) | Intermediate Host (I/H) | Location in F/H | Distribution |
|---|---|---|---|---|
| *Macracanthorhynchus hirudinaceus* | Pig | Various dung beetles | | Worldwide except Western Europe |

**Identification**

**Gross:**

- Adults resemble *A. suum*, but taper posteriorly.
- The males are up to 10 cm and the females up to 65 cm length.
- When placed in water the spiny proboscis protruded, thus aiding differentiation from Ascaris.

**Microscopic:**

The egg is oval, 110µm by 65 µm, with a thick brown shell and contains the **acanthor larva when laid.**

**Life Cycle**

Adults, attached to the small intestinal mucosa, lay eggs which are passed in the faeces. These are produced in large numbers, are very resistant to extremes of climate and can survive for years in the environment. After ingestion by dung beetle larvae, the acanthor develops to the infective cystacanth stage in approximately three months. Infection of pigs occurs after ingestion of beetle grubs and adults and the prepatent period is 2-3 months.

## Pathogenic Significance

- *M. hirudinaceus* produces inflammation and may provoke **granuloma** formation at the site of attachment in the small intestinal wall.
- Heavy infections may cause weight loss and, rarely, penetration of the intestinal wall results in a fatal peritonitis.

## Diagnosis

- This is based on finding the typical eggs in the faeces.

## Treatment and Control

- Pigs should be prevented from access to the intermediate hosts.
- In modern management systems, this may be easily achieved, but where pigs are kept in small sties the faeces should be regularly removed to reduce the prevalence of the dung beetle intermediate hosts. Although there is little information on treatment, levamisole and ivermectin are reported to be effective.

## Acanthocephalans of aquatic birds

- There are two genera which may cause enteritis in aquatic fowl, namely *Polymorphus* and *Filicollis*. The **intermediate hosts** in both cases are **crustaceans** and in all other respects they are typical of the group.

**Superfamily : Ascaridoidea**

Family : Ascarididae

# Epidemiology:

- Is a GI parasite of pig but occasionally found in cattle, sheep and man
- Sucking piglets may become infected shortly after birth; the skin of the mother may be contaminated with embryonated eggs.
- Infection is most prevalent and heaviest in 3-6 months old pigs.
- Pig acquired natural resistance with increasing age, though parasite free animal remain susceptible to infection even over 2 years of age.
- Acquired immunity plays an important role in reducing intensity and longevity of patent infection and in limiting the establishment of subsequent challenges.
- Older piglets, sows and boars may act as worm carriers
- Husbandry condition, which allow ready access to earthworms, for example grazing, garbage, heaps etc which opens the way of transmission through ingesting the paratenic hosts.
- Larvated eggs are highly resistant to drying and freezing temperature

Note: *Ascaris lumbricoides* causes Ascariosis in man, which is specific for man, is irrelevant to Vet. Medicine but previously thought *Ascaris suum* is synonymous for *Ascaris lumbricoides.* But there is morphological differences e.g. difference in the identification of lips

**Pathogenesis /Pathogenic significance:**

- Adult parasites are not highly pathogenic. Main pathologic events occur during pre-patent stage (8-9wks)
- In heavy infection, the migrating larva may cause severe damage & mechanical destruction in various organs like liver, lungs, bile duct etc.
- Hypersensitivity and allergic reaction also occurred.

**Liver**:

- The fibrotic liver after inflammation heals, gives rise to so called **"Milk spot" or "white spot**" lesions & made whole liver unfit for human consumption.
- Hemorrhage may occur in liver & in most cases lungs.

## Lungs

- In most of the cases lungs become haemorrhagic (due to breakdown of lungs capillaries). The lungs hemorrhages can be followed by desquamation of alveolar epitheliums, edema & infiltration of the eosinophils & other cells in pulmonary parenchyma.
- The migrating larval stages in large numbers may cause a transient pneumonia which is called **Ascaris Pneumonia.**
- Extensive lung lesions give rise to clinical signs of severe respiratory embarrassment (thumps) where breathing is rapid, shallow & marked by forcible expiratory efforts & coughing and pigs may die.
- Death from severe lung damage in 15 days after infection.

## Intestine

- Little apparent damage to the mucosa
- If numerous, adult parasites may form a bundle and create intestinal obstruction.
- Adult may causes diarrhea by interference with proper nutrition and normal growth

## Bile duct

- If numerous in intestine, rarely a worm may migrate into the bile duct causes biliary obstruction, biliary stasis, peritonitis, obstructive jaundice which causes condemnation of carcass.

**Clinical findings:**

- Depends on severity of infection.
- Severe respiratory distress in growing pig/piglets due to **Ascaris Pneumonia (**Interstitial Pneumonia**)** which is usually transient rapidly resolving.
- Pneumonia in heavily infected newborn pigs especially a cough & exudates into the lungs.
- Hepatic fibrosis & diarrhea, anaemia in some cases

**Necropsy findings:**

- Extensive petechial & echymotic pulmonary hemorrhage & edema indicate acute ascarosis.
- Liver shows fibrosis as "**milk spots".** These are manly whitish in color but become hemorrhagic when recent.

**Diagnosis**

**Presumptive diagnosis:**

- Clinical History
- Clinical findings
- Necropsy findings.

**Confirmatory Diagnosis:**

- Demonstration of larva in sputum in early stage.
- Identification of larva in Baermann apparatus by mincing (minced) the pieces of lung tissue.
- Presence of Ascaris egg in the faeces. (Sometimes due to coprophagia pigs may shed egg in feces without having adult in their intestine, thus false positive result can occur)

## Treatment

- At first isolate the infected animal and provide the following treatment with hygienic management

Migrating larva is not affected by anthelminties.

- For adult the following anthelmintics can be used.
  - Piperazines Orally 100-400 ng/kg BW.
  - Dichlorvos Orally @ 10 mg/kg.BW
  - Levamisole Orally @ 7.5mg/ kg BW
  - Tetramisole Orally @ 5 mg/kg BW
- Ovicidal: Fenbendazole, Orally @ 5 mg/kg BW
  - Supportive treatment
- Saline for diarrhea
- Haemtenic mixture in case of Anaemia
- Multivitamin etc.

## Control:

- The aim of control is the breakdown of parasite life cycle by appropriate measures
- Strict and constant hygiene in feeding, bedding and cleaning.
- Use of "**All in all out system"** of repopulation, specially of furrowing and fattening unites; this greatly facilitates the creation of clean pigs pen for rearing of new born piglets.
- The most relevant measures is careful mechanical cleaning, removal of fecal materials and bedding contaminated with eggs, hosing of floors, walls feeding toughs etc.
- Cleaning may be completed by disinfection by heat, hot vapor, highly potent disinfection 5% formaldehyde etc.
- Removal of larvated eggs adhering to skin and coat of gestating sows by careful washing of their body surface before moving to the furrowing pen.
- Course of treatment with medicated food for 6-7 day ensures prolonged bio-availability of drugs and more balanced uptake of the effective done by all animals. This method of treatment enhances both the curative and preventive efficacy of the drug used.
- Strategic 6-7 days treatment course of piglets should be first applied between 30-50days of age and be repeated every 2 months for as long as necessary to prevent establishment of a patent infection. Pigs should be treated for roundworms before moving them to the fattening units, young pig exposed to drug abbreviated natural infections acquire incomplete to subsequent challenge.
- In infected piggeries, breeding sows should be treated twice, at 14-17 and 3-4 days before furrowing, bedding boars should be treated yearly.
- Methods of control recommended against ascariosis are applicable also to control other nematodes of direct life cycle of Pigs such *Hyostrongylus, Oesophagostomum* and *Trichuris.*

**Short Note: Milk spot**

- Milk spot are lesions of liver of porcine Ascariosis caused by the migratory larvae $L_2$ and $L_3$ of *Ascaris sum* in pig.
- The spots appears as cloudy whitish spots of up to 1cm in diameter and represents the fibrous repair of inflammatory reactions to the passage of larvae in the liver of previously sensitized pigs.
- The spot may become haemorrhagic.
- In chronic infections, the liver may become markedly fibrotic. Infection of neonatal piglets doesn't provoke eosinophilia, which is seen in older infected animals.
- However, Milk spot on the liver of infected animals occurred at all ages. Varying degree of Pneumonia or Bronchitis may be found or only a number of petechial haemorrhage in the lungs.

***Parascaris equorum***

- Infection with *Parascaris equorum* is common through out the world is a major cause of **unthriftiness** in young foals.

**Disease:** Equine Parascariosis, Round worm disease of horse and donkeys.

**Site:** Small intestine.

**Distribution:** Worldwide

**Morphology/Identification:**

- One of the longest nematode species,rigid, whitish worm
- Male: 15-28cm and Female: 50cm in length
- Has a simple mouth opening surrounded by 3 large lips.
- The tail of male has small caudal alae.

**Eggs:**

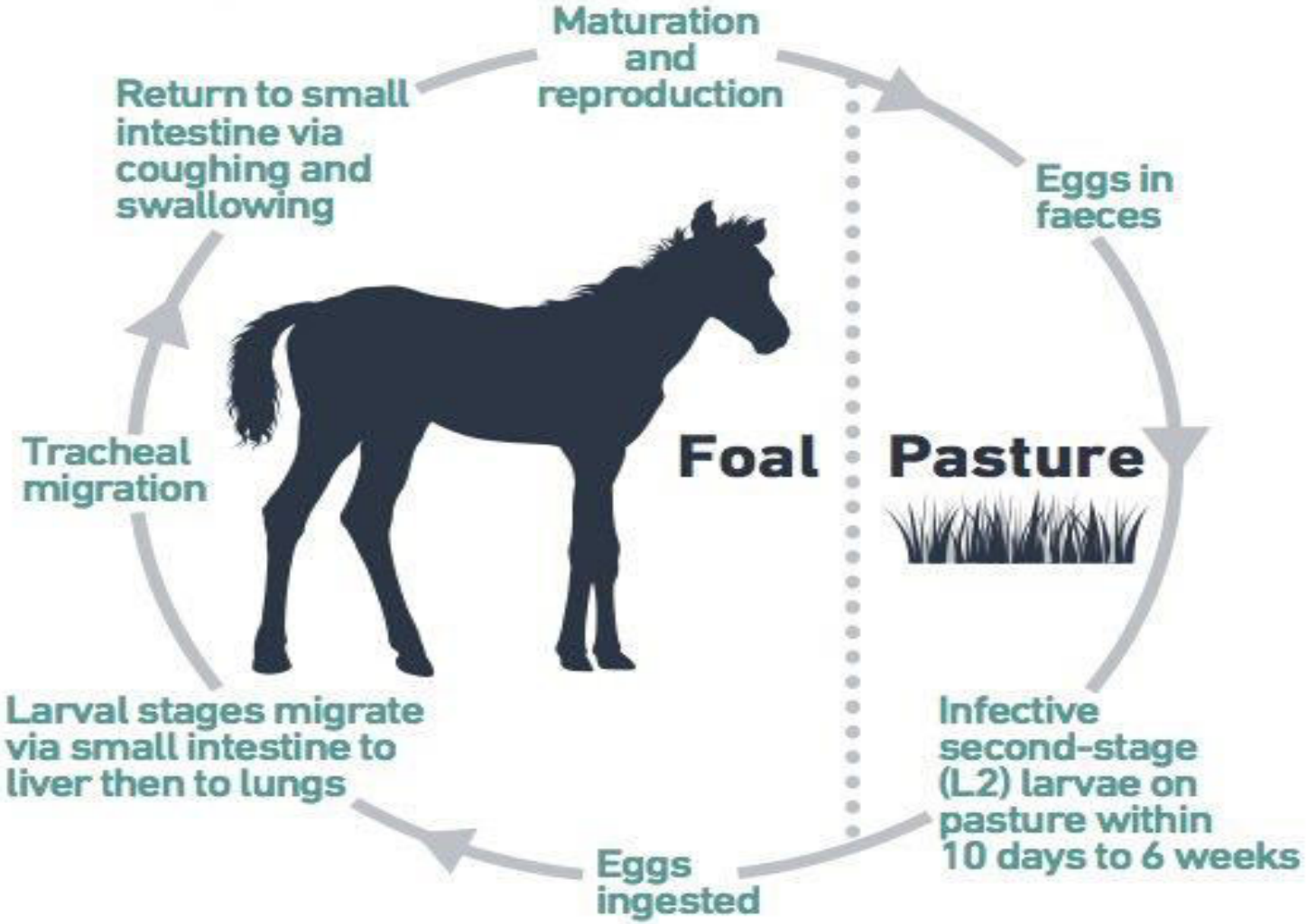
- Almost spherical in shape
- Yellowish brown in colour
- Thick shelled with an outer pitted coat
- 90-100µm in size.
- Passed out in the faeces in the one cell stage

**Epidemiology:**

- Larved eggs are resistant to desiccation and freezing temperature and may survive several grazing seasons on pasture, paddocks and stalls.
- Sucking and weaning foals have the heaviest infections and the highest prevalence peaking at the age of 16-18 weeks.
- Mature horse and donkey may be worm carriers

No prenatal infection

# Life cycle of ascarids in foals (*P. equorum*) – 10-15 weeks



**Pathogenesis**

- Gross changes provoked in the liver and lungs by migrating *Parascaris equorum* larvae

**Liver:**

- Larvae cause focal haemorrhage and Eosinophilic tract which resolve leaving whitish fibrosis

**Lungs**

- Larvae cause haemorrhage and infiltration by eosinophils which are later replaced by accumulations of lymphocytes
- Sub-pleural grayish green lymphocytic nodules developed around dead or dying larvae. These nodules are more numerous following re-infection.

**Small intestine**

- Usually not associated with any specific lesions.
- Occasionally, heavy infection causes impaction and perforation of the intestine which lead to peritonitis sometimes invagination and mechanical occlusion of SI.

**Clinical findings:**

- Heavier infection may cause coughing, changing appetite, unthriftiness or poor growth, abdominal pain (colic), diarrhea, dull coat, emaciation, severe debilitation and finally death.

**Note:** Infection elicits immunity leading to partial or total elimination of the worm burden

**Diagnosis:**

- Clinical History
- Clinical findings
- Coproscopy: the eggs may show 2 kinds of appearance
  - Typical are the eggs with thickened, medium-brown roughened outer shell
  - Atypical eggs are devoid of the darkened outer shell and appear as smooth and thick walled, colorless and translucid.
- Diagnosis may be confirmed by administration of Anthelmintic when large number of worms may be observed in the feces.

**Treatment**

- Thiabendazole @ 44mg/kg BW
- Mebendazole @ 10 mg/kg BW
- Albendazole group

**Note: Don't use Tetramisole and levamisole due to their toxicity to horses.**

**Control:**

- Regular anthelmintics treatment
- Remove feces from paddock/stable regularly
- Sanitary measures during birth of a foal
- Brood mares should be treated before foaling for Ascarid
- Safe disposal of manure
- Anthelmintc prophylaxis for the horse Strongyles will effectively control of *P. equorum* infection.
- Since transmission is largely on a foal to foal basis, it is good policy to avoid using the same paddocks for nursing mares and their foals in successive years.

Round worm disease of Carnivores: Toxascariosis

**Causal agent:** Toxascaris, Toxocara

Vet significance is low; Public health significance may be high.

***Toxascaris leonina***

**Host**: Mainly domestic carnivores but infects both canidae and felidae e.g. Dog, Cat, Lion, Fox, Jackel etc.

**Site**: Small intestine

**Distribution**: Worldwide

**Disease**: Toxoascariosis

**Morphology**:

**Adult**

- Large, white worms, 6-10 cm in length (male: 7cm and Female: 10cm)
- Almost similar to *Toxocara sp.*
- The cervical alae lanceolate in shape (*Toxocara cati*, arrow headed) and the posterior margin of the alae tapered gradually into the body.

**Eggs:**

- Sub-spherical/ slightly ovoid shape, 70-85µm size
- Light, colourless
- **A smooth thick shell**

**Epidemiology:**

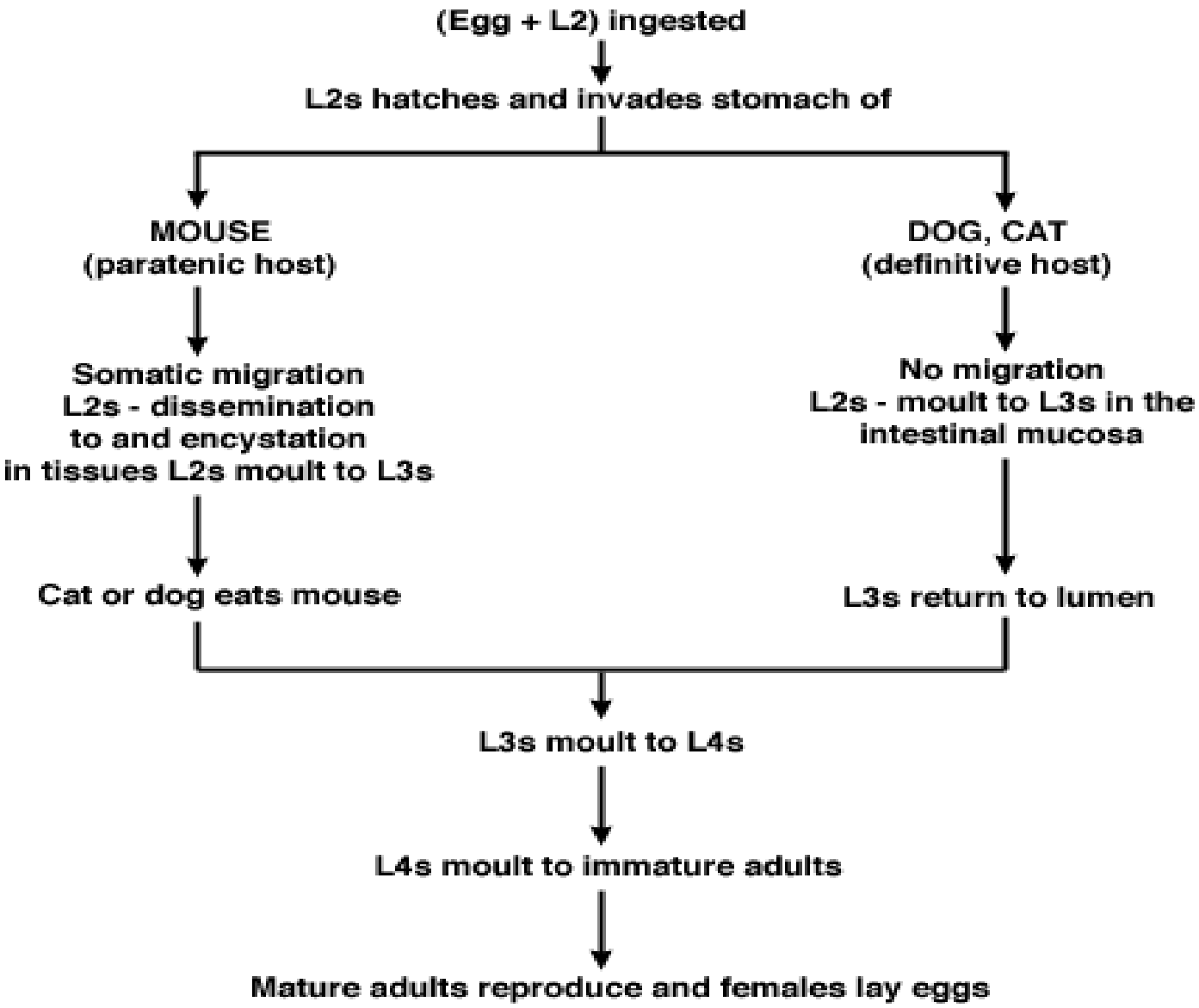
- Puppies and kittens younger than 6month are more susceptible than olders (Toxocara is far more prevalent than Toxoascaris).
- Older may serve as carriers.
- Mice may acts as paratenic host.
- Female roundworm are extremely fecund and larvated eggs may survive several years.

**Pathogenic significance:**

Though Toxascaris is more common, but it has less significance than Toxocara because its parasitic phase is **non migratory**. Adult worm may cause

- Curtailment of food
- Mechanical damage
- Changing appetite
- Dull coat, etc.

# *Toxascaris leonina* Life Cycle



**Diagnosis:**

- Clinical History
- Clinical findings
- Coproscopy for egg identification

**Treatment:**

- Anthelmintics
- Piperazine
- Levamisole
- Benzimidazole

**Control:**

- Hygienic management
- Prevent mice entrance
- Treatment for the infected animals

# *Toxocara canis*

**Host:** Dog, Fox, jackel and other canids. Unnatural host: Human

**Site:** Small intestine, stomach

**Distribution:** World-wide

**Morphology:**

**Adult:**

- Large, white; 10-18cm in length (male: 10cm, female: 18cm)
- Large cervical alae present
- The tail of male has terminal narrow appandages (a small finger like process) and caudal alae and spicule (0.75-0.95mm long

**Egg:**

- Subglobular/sub-spherical shape
- 70-90µm in size
- Dark brown in colour
- **A thick pitted shell**

## Epidemiology:

- The highest prevalence in dogs of less than 6month of age with the fewest worms in adult
- The widespread distribution and high intensity of infection with *Toxocara canis* depend essentially on the three factors-
- The female are extremely fecund**.** One female worm lay about 700 eggs/gm/day.15000 epg are not uncommon in pups. Female lay 250000 eggs per day.
- Eggs are highly resistant to climatic changes and can survive for years on the ground
- Bitch acts as constant reservoirs of infection in the somatic tissue where larvae are insusceptible to most anthelmintics.

# *Toxocara canis* Life Cycle

(Egg + L2) ingested

L2 hatches and migrates to liver, then lung via blood

**B**

PUPPY (<5 weeks old)

Tracheal migration L2s moult to L3s which then migrate up the bronchial tree

Pharynx (L3s swallowed)

Stomach (L3 --> L4)

Small intestine (L4 --> Adults)

**D**

MATURE DOGS (>6 months old)

Somatic migration L2s - dissemination via peripheral circulation to tissues where they encyst

Pregnant bitch

**A**

Transplacental migration of L2s to fetal liver

Neonatal pup lungs

Migration of L2s to mammary glands (moult to L3s)

**C**

L3s ingested by suckling pups

MOUSE (paratenic host)

Somatic migration L2s - dissemination via peripheral circulation to tissues where they encyst

**E**

Dog eats mouse Larvae released

L2 --> L3 --> L4 --> adults in the intestine of the canine host

## Pathogenesis:

- In moderate infection, the larval migratory phase is associated without any apparent damage to the tissues and the adult worms provoked little reaction in the intestine.
- In heavy infections, the pulmonary phase of larval migration is associated with **Pneumonia,** which is sometimes accompanied by **Pulmonary oedema.** The adult worms cause a **mucoid enteritis**. There may be partial or complete **occlusion of the gut** and in rare cases, **perforation** with **peritonitis** or in some instances **blockage of bile duct.**
- In man, immunologic reaction may occur with subsequent granuloma formation. Visceral and larval migrations occur in Man

**Clinical findings:**

- Most fatalities occur usually in pulmonary phase of larval migration

**In mild to moderate infection:**

- There are no clinical signs during the pulmonary phase of larval migration.
- The adult worms in the intestine may cause –
  - Pot belly appearance
  - Changing appetite
  - Dull coat
  - Failure to thrive
  - Decrease body weight
  - Occasional Diarrhoea
  - Vomition after feeding
  - Entire worms are sometimes vomited or passed in the feaces.

**In heavy infection:**

During larval migration-

- Pumonary damage- Pneumonia
- Coughing
- Increased respiratory rate
- Frothy nasal discharge
- Pups may die within a few days of birth which have been heavily transplacentally.
- Nervous signs may be seen (mechanism unknown)

**Diagnosis:**

1. Clinical History
2. Clinical findings
3. Coproscopy for egg and adult identification
4. Necropsy findings

## Treatment:

- Anthelmintics
- Piperazine compounds: @ 100-200mg/kg BW
- Benzimidazole
- Fenbendazole
- Mebendazole

## Control:

- Control of Toxocarosis in young dogs/pups is as follows:
- All pups should be dosed at 2 wks of age and again 2-3wks later, to eliminate prenatally acquired infection. The bitch should be treated at the same time as the pups
- A further dose should be given to the pups at 2 months old, to eliminate any infection acquired from the milk of the dam or from any increase in fecal egg output by the dam in weeks following whelping.
- Newly purchased pups should be dosed twice at an interval of 14 days
- Since there are likely to be a few worms present, even in adult dogs, in spite of the diversion of the majority of larvae to the somatic tissues, it is recommended that adult dogs should be treated every 3-6 months throughout their lives.
- Daily administration of high doses of Fenbendazole to the bitch from 3 weeks pre partum to 2days post partum has largely eliminated transmammary and pre-natal infection in the tissues of the bitch may persist. This regimen may be useful in Breeding kennels.

**Short Note: Visceral Larval Migrans (VLM)**

It is a pathological condition caused by the invasion of the visceral tissues of an animal by parasites whose natural hosts are other animals. Now, it refers invasion of *Toxocara canis* infective larva in human tissues (Children).

- The condition may also be caused by infective larvae of
- *Toxocara leonine*
- *Toxocara cati*
- *Cpillaria hepatica* (of Rodent),
- *Lagochilascaris minor* (wild feline),
- less frequently also by *Toxocara vitulorum* and *Strongyloides stercoralis*

Children are at high risk from pet/public parks. Usually the infection is acquired by ingesting embryonated eggs from soil or from dog fur.

This entry is characterized by

- Chronic eosinophilic granulomatous lesions in the liver, lungs, eye or elsewhere.
- Enlarged liver (hepatomegale), Pulmonary infiltration
- Intermittent fever, loss of weight and appetite and persistent cough.
- In children, the larvae migrate in somatic manners.

**In case of Ocular larval Migrans (OLM):**

- Granuloma form around the larva on retina resulting a **Retinoblastoma**
- Sometimes granuloma involves the Optic disc with total loss of vision/partial loss of vision with **endophthalmitis** or **granulomatus retinitis.**

## Diagnosis:

- Demonstration of larvae in the lesions in Biopsy material
- Immunodiagnostic tests are also valuable

## Treatment:

- Anthelmintics therapy
- Laser therapy in Ocular larval migrans

**Control:**

- Anthelmintics treatment
- Safe disposal of Dog feces
- Limitation of Dog's access to public places

# *Toxocara cati* (Syn: *T. mystax)*

Host: Cat and wild felids (lion, Tiger etc.)

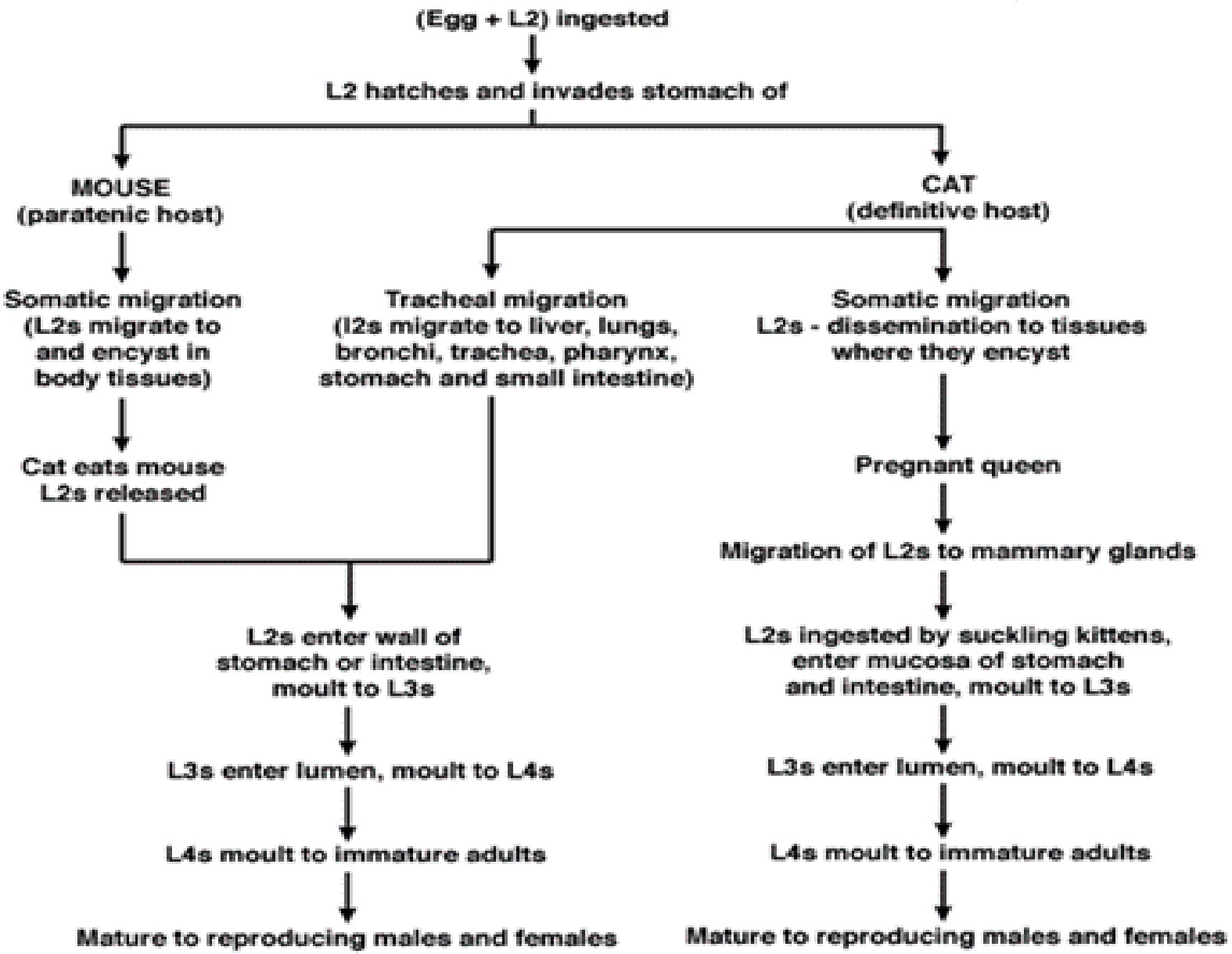
Site: Small intestine

Distribution: World-wide

Morphology:

- Large, white worm
- Male: 3-6cm, Female: 4-10cm
- The cervical alae are seen to have an **Arrow head** from with the posterior margins almost at a right angle to the body (where as those of Toxascaris taper gradually into the body).
- Spicules : 1.6 to 2mm long
  - Egg:
- Subglobular
- A thick pitted shell
- Almost colourless
- 65-75μm in size

# *Toxocara cati* Life Cycle



## Epidemiology:

- It depends lagely on a reservoir of larvae in the tissues of the dam which are mobilized late in pregnancy and excreted in the milk throughout lactation.
- The paratenic host is also of considerable significance because of the strong hunting instinct in cat
- Exposure to the latter route of infection does not occur until kittens begins to hunt for themselves or to share the prey of their Dams

## Pathogenesis and Clinical Signs:

- Migratory phase is usually absent
- So, any changes are usually confined to the intestine, showing
  - Pot belly appearance
  - Diarrhoea
  - Poor body coat
  - Failure to thrive

## Diagnosis:

- Clinical History
- Clinical findings
- Coproscopy for egg identification

**Treatment:**

- Anthelmintics
- Piperazine compounds: @ 100-200mg/kg BW
- Benzimidazole
- Fenbendazole
- Mebendazole

**Control:**

- Removal of kittens from the infected Dam and artificial rearing
- Early and repeated administration of anthelmintics to kittens along the lines recommended for *T. canis* in pups.

**Differentiation among *Toxascaris leonine, Toxocara canis* and *Toxocara cati***

The above mentioned parasites are known as **Arrow worm/Arrow Headed Worm**

| Parameter | *Toxascaris leonine* | *Toxocara canis* | *Toxocara cati* |
|---|---|---|---|
| Cervical Alae | * Lanceolate shaped<br>* The posterior margin taper gradually into the body | | *Arrow Headed<br>*The posterior margin almost at a right angle to the body |
| Spicule | | 0.75-0.95mm long | |
| Male tail | Not such as *T. canis* has | Has a terminal narrow appendages which is a small finger like process | Not such as T. canis has |
| Egg | Smooth thick shell 70-85µm | Thick pitted shell 70-90 µm size | Thick pitted shell 65-75 µm size |

| Parameter | *Toxascaris leonine* | *Toxocara canis* | *Toxocara cati* |
|---|---|---|---|
| L/C | | | |
| Parasitic pahse | Non migratory | Migratory/Non-migratory | Usually Non migratory, But Migratory may be occurred |
| Prenatal infection | Absent | Present | Absent |
| Transmammary infection | Absent | Present | Present |
| Hepatotracheal migration | Absent | Present | Present |