Order: ASCARIDIDA

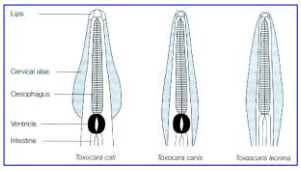
Superfamily: COSMOCERCOIDEA
Family: Atractidae (Genus: *Probstmayria*)

ASCARIDOIDEA
Family Ascarididae
Subfamily: Ascaridinae (Genera: ***Ascaris, Baylisascaris, Parascaris, Toxascaris***)
Subfamily: Toxocarinae (Genus: ***Toxocara***)
Family: Anisakidae (Genera: ***Anisakis, Contracaecum, Pseudoterranova, Terranova, ...***)

HETERAKOIDEA
Family: Heterakidae ***Heterakis***
Ascaridiidae ***Ascaridia***

Ascaroidea (Ascarids)

- adult – whitish, spindle-shaped, medium-sized to large worms up to 40 cm long and 5 mm wide;
- Oral opening is mostly surrounded by 3 lips;
- anterior end of several genera have cervical alae;

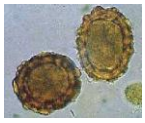


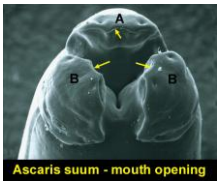
- males – 2 spicules, posterior, **without bursa**, sometimes with small lateral alae;

- **LC – direct or indirect**

Ascaris suum (large roundworm of pigs) / Ascariosis in swine

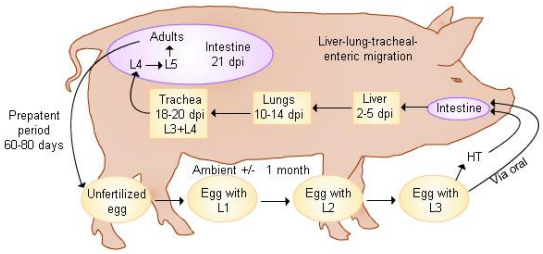
- Ascariosis is one of the most frequent helmintic infections of pigs with considerable economic impact;





- *Ascaris suum* is closely related to *Ascaris lumbricoides* (zoonotic importance)

Life cycle - direct



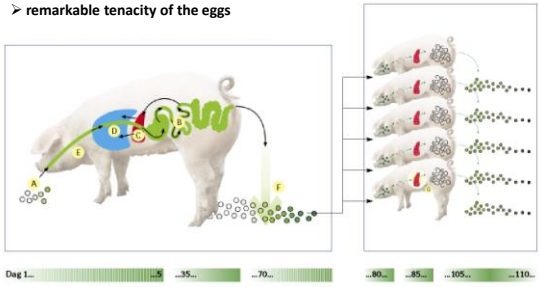
Epidemiology

- Female worms may produce as many as **2 million eggs daily** during her life time in the definitive host which can be a year or more.

- These thick-shelled eggs are resistant to freezing and drying and can therefore survive for as long as **five years** under most farm environments.

- However, exposure to sunlight and prolonged exposure to drying will destroy them. Overall, these factors ensure that the normal swine environment will be well contaminated with Ascaris eggs, ensuring that all young pigs, especially those raised on soil, will become infected.

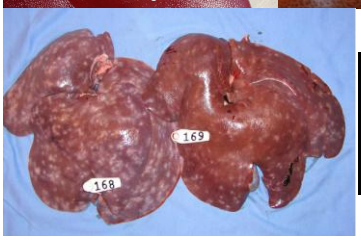
- **high reproductive potential (daily production of >200,000 eggs per female)**
- **slow exogenous development**
- **remarkable tenacity of the eggs**

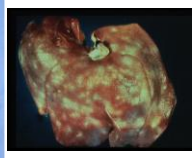


- in moist soil eggs remain infectious for 5 years; most of them die within 1 year;
- survive in raw pig slurry at 18 °C – 3 months; at 25 °C up to 8 months;

Pathogenesis and clinical signs

- Larvae cause significant physical damage in the organs and tissues through which they migrate.
- Migrating larvae in the liver cause an inflammatory reaction, intralobular **tissue destruction** and **hemorrhage**.
- This is followed by an **intense infiltration of eosinophils** and **collagen production**.
- These lesions are **visible at necropsy** on liver surfaces as whitish areas and are commonly called **"milk spots"** since they resemble splashes of milk. In the absence of reinfection these lesions will begin to regress after larvae migrate beyond the liver and **will be healed completely after 4 to 6 weeks**: therefore their presence at necropsy is an indication of recent infections.
- In pigs that experience multiple reinfections during their lifetimes, **livers become markedly fibrotic**.
- Lungs – haemorrhages; cellular eosinophilic infiltrations, local oedemas.
- Intestine – cell hyperplasia, swelling of tunica muscularis; **atrophy of the villi**.



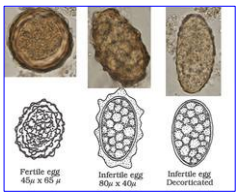


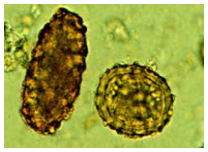
- infections – asymptomatic;
- reduction food intake and stunted weight gain;
- health impairment + liver condemnation at slaughter = economic loss (depending on the intensity of the infection);
- migrating larvae of *A. suum* may also affect liver and lungs of other animals (sheep, calves) and humans (zoonotic importance).

Cross-infections of humans with *A. suum* and of pigs with *A. lumbricoides* are confirmed.

Diagnosis

- Coprological examination
- 3 types of eggs:
- **fertilisated**
- **unfertilisated**
- **decorticated**





Deworming programme

Piglets: in case of heavy infection, deworm immediately after weaning (1) deworm, all piglets after transfer to the fattening station (2).

Fatteners: deworm all pigs 7 weeks after transfer (3) Deworm 6 weeks later (4), only when the pigs are not slaughtered before 16 weeks after regrouping.

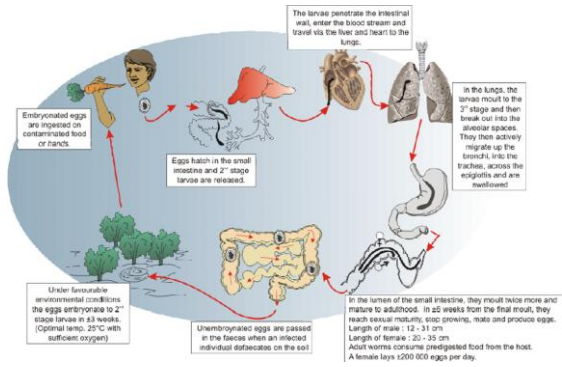
Sows and boars: deworm ALL pigs at least 3 times a year.

- **Levamisole; benzimidazoles; macrocyclic lactones**

Ascaris lumbricoides

Definitive Host: Humans
Intermediate Host: None
Geographic Distribution: Cosmopolitan
25% of world population is infected
Has been known as human parasite for over 2000 years
Location: small intestine

Life cycle



Pathology and Symptoms

Ascariosis
- Depends on the number of worms
- Worms subsist on liquid content of small intestines
  Do not suck blood or graze on mucosa.
- Small to medium infections are usually asymptomatic
  May cause "sensitization phenomenon"
  Allergic reaction to worm waste.
  Rashes, eye pain, asthma, insomnia, restlessness
  - **Acute intestinal obstruction**

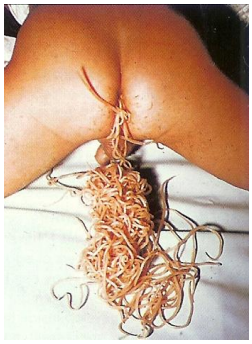
Pathogenesis:

1. "Verminous" pneumonia, lung tissue damage due to migratory larvae.
2. Bowel obstruction - too many adult worms.
3. Parasite secretes trypsin inhibitor, **prevents host from digesting proteins.**
4. **Aberrant migration** of "irritated" adult worms to:
   a. Appendix
   b. Common duct
   c. Liver
   d. Pharynx
   e. Peritoneum

A. lumbricoides

Pathology and Symptoms

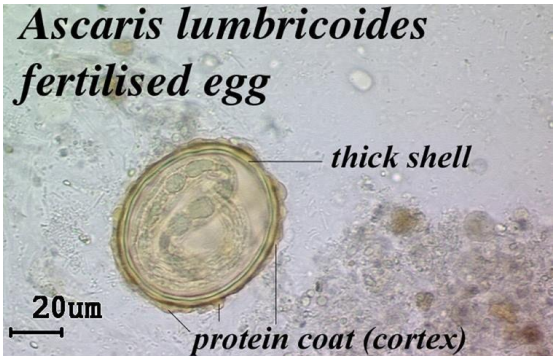
- **Wandering adults are dangerous.**
- **Overcrowding can lead to wandering**
  Upstream to pancreatic or bile ducts
  Downstream to appendix or out anus
- **Females like to crawl through small spaces**
  It may crawl through nose, ear, or any other opening
  Aspiration of worm can cause death



Diagnosis:
Eggs in feces.
Juveniles in sputum.
Difficult to identify to species.
Dead adults may be found in faeces

Treatment:
- Mebendazole
- May need to repeat treatment

Dead adults usually pass out through the anus



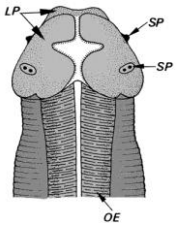
Therapy

Mebendazole
Pyrantel pamoate
Albendazole
Piperazine

Pyrantel pamoate in a single dose, albendazole in a single dose, or mebendazole for 3 days is recommended for treatment of asymptomatic and symptomatic infections.
Piperazine paralyzes the worms, allowing them to be excreted with intestinal peristalsis.
Piperazine should not be used with pyrantel pamoate because the two drugs are antagonistic.

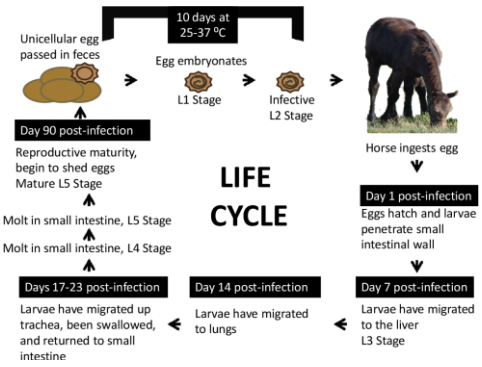
2. Sanitary disposal of feces.
3. Hygienic habits such as cleaning of hands before meals.
4. Health education

Parascaris equorum/Parascariosis in equines



- The females may be as large as 50 cm in length (18-50) and 8 mm in diameter.
- Males are smaller, ranging from 15 to 28 cm long.
- The mouth opening is typically ascarid with three prominent lips.

- Infections are found mainly in nursing and weaned foals less than a year old.



Pathogenesis

- Larval migrations through the lungs are associated first with the apearance of *petechial hemorrhages* followed by intense infiltrations of eosinophils around alveoli, bronchioles and small blood vessels. These are later replaced by **lymphocytes**.

- Beginning approximately four weeks after infection, lymphocytic nodules begin to appear under the pleura. They are raised, greyish-green in colour and contain dying larvae and lymphocytes surrounded by a fibrous capsule.

- These nodules are more common in older foals that have experienced **multiple natural reinfections** and **may indicate a strong immune response to migrating larvae.**

Clinical signs

- In **foals up to 6 months** old, coughing and greyish-white nasal discharges are seen during larval migrations through the lungs. Endoscopic examinations of the trachea, at this time, show a frothy mucus in the upper air passages.

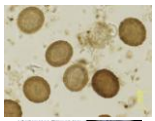
- Heavy infections may result in gut impactions and death following rupture of the small intestine.

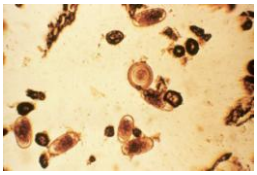
Diagnosis

Clinical signs

➢ A coughing foal with a nasal discharge is a likely indication of a prepatent infection with *P. equorum*.

Coprological examination





Therapy and control

➢ Benzimidazoles
➢ Macrocyclic lactones (resistance to ivermectin)
➢ Pyrantel derivates

➢ Regular cleaning of the stables
➢ Weekly removal of the faeces from pastures combined with strategic treatment are suitable control measures

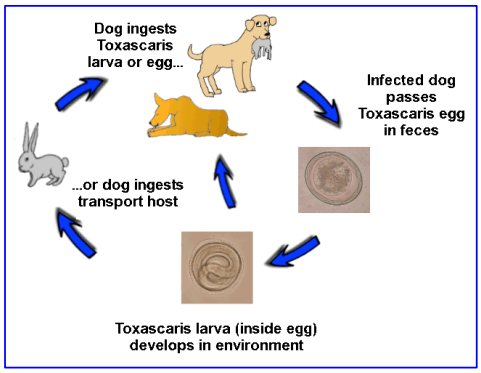
Genus: *Toxascaris*

Toxascariosis in dogs and cats or other carnivores

Ascaridioid type - Characterized by a **histotrophic phase** confined to the mucosa of the small intestine, followed by **re-entering of the larvae into the lumen, and development to the adult stage**. **THERE IS NO MIGRATION OF POST INFECTION LARVAE**

Toxascaris leonina

Toxascaris leonina



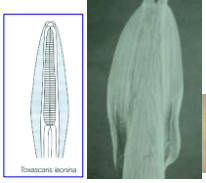
➢ small intestine of carnivores
➢ prepatency: 7-10 weeks

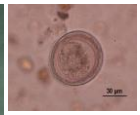
➢ worldwide distribution
➢ felids (lion, tiger, leopard, cheetah, ...)
➢ canids (foxes, racoon dog, wolf, jackal, dingo, ...)

➢ low pathogenicity, heavy infections – **enteritis**;

➢ Dg: coprology

➢ Te: as in toxocarosis;



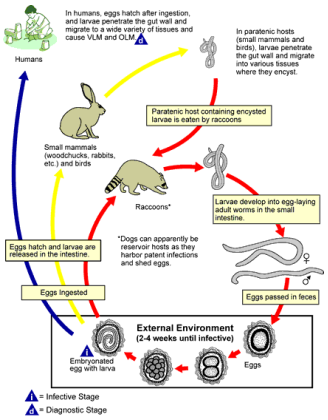


Baylisascaris procyonis/baylisascariosis

Risk to man

Baylisascaris melis
Baylisascaris transfuga

➢ Raccoons; dogs, cats;
➢ Humans may become infected by accidental uptake of larvated eggs;
➢ Migration characterized by a **entero-hepato-pulmonary**



- in paratenic hosts, migrating larvae have an affinity for the brain = death
- LMO
- LMV

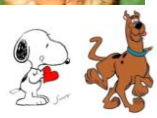
1000 µm

- Dg: coprology

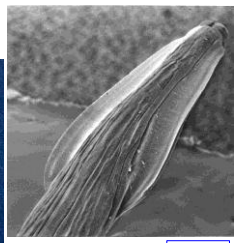
- Te: as in toxocarosis

Genus: *Toxocara*

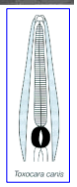
Toxocara canis
Toxocara cati
Visceral and ocular larva migrans



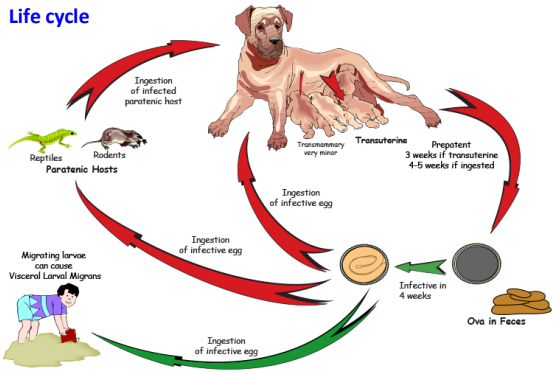
Toxocara canis

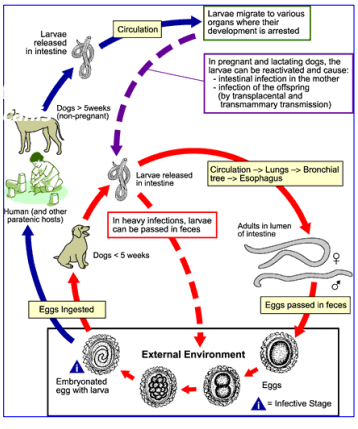


- Males are 10-12 cm long
- Females are 12-18 cm long
- 3 lips
- long and narrow cervical allae
- oesophagus with ventricle
- male with 2 spicules



Life cycle





MIGRATION ROUTES IN DEFINITIVE HOSTS

- Tracheal (lung-tracheal) migration
- Somatic migration
  - Prenatal (transplacental) transmission
  - Lactogenic (transmammary) transmission
- Faeco-oral transmission of *T. canis* stages **from puppies to the bitch**

- **Transmission of infective larvae in paratenic hosts**

Toxocara canis

➢ *Toxocara canis* has a **complete toxocaroid life cycle**.

Dogs may become infected by four routes:

1. **Direct transmission**, by ingesting infective eggs.
2. **Paratenic host transmission**, by ingesting infected mice.
3. **Transmammary transmission** in which nursing pups ingest L3s in their mother's milk.
4. **Prenatal transmission** where pups are born infected as a result of L3s migrating from tissue reservoirs in the pregnant bitch - across the placenta and through the umbilical vein to the foetal liver, where they remain until birth.
   They then resume migration to the lungs of the newborn pups.

Epidemiology

➢ Most young puppies and approximately 20% of adult dogs are actively infected with *Toxocara canis.*

➢ Puppies between 3 weeks and 3 months of age excrete large numbers of eggs and constitute the **greatest hazard to the environment**.

Pathogenesis and clinical signs

Toxocara infections can cause different types of disease in definitive hosts (dogs) and paratenic hosts.

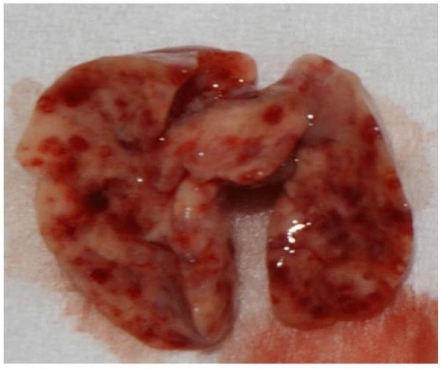
Dogs

➢ Light to moderate intestinal infection in puppies and older dogs: asymptomatic infection or occasional vomiting, diarrhoea, mucous faeces, retarded development in young animals.

➢ Heavy infections in puppies:

1. **Tissue damage**
2. **Intestinal infection**
3. **Infection of older dogs** by somatic larvae: **generally asymptomatic**.

Figure 1: Puppy showing pendulous abdomen with obstipation due to gut obstruction by *Toxocara canis*



Haemorrhagic lesions in the lung of a mouse infected with *T. canis* on day 2.

Diagnosis

➢ coprology – eggs, sometimes larvae or adult worms;
➢ USG

THERAPY – *T. canis* (adult dogs)

Active substance	Dose	Administration
Emodepsid	1 mg/kg/day	p.o.
Fenbendazol	50 mg/kg/day	p.o. 3 days
	or 100 mg/kg	p.o.
Febantel	10-15 mg/kg/day	p.o. 3 days
Flubendazol	22 mg/kg/day	p.o. 2 days
Milbemycín	0,5-1 mg/kg/day	p.o.
Moxidektín	2,5 mg/kg	**spot-on**
Pyrantel	5 mg/kg/day	p.o.
Oxibendazol	10-20 mg/kg/day	p.o. 2-3 days
Selamektín	6 mg/kg	**spot-on**

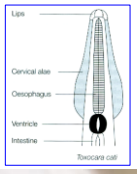
Protection of puppies

Preimaginal dehelmintization

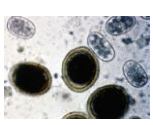
- every month to 6 months of age
- quarterly to the age of 1-2 years

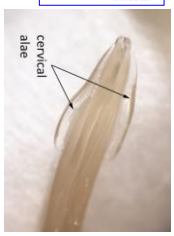
Adult animals - targeted dehelmintization based on coprological examination.

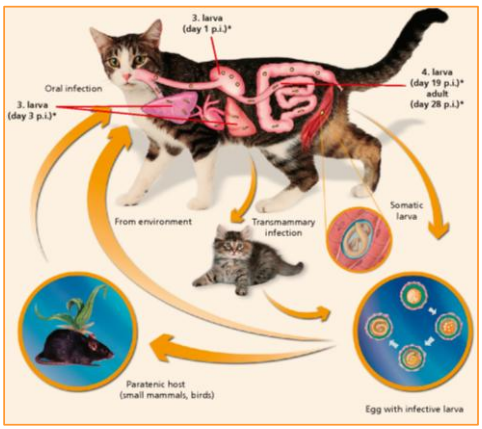
Toxocara species in felids
Toxocarosis in felids



- ***Toxocara cati*** (syn. *Toxocara mystax*)
- *T. malayensis* – Malaysia, China
- cervical alae are broader and more convex giving the anterior end the appearance of an arrow head;
- small intestine of cats and wild felids



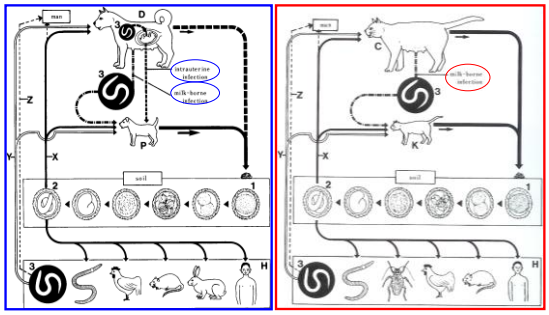




Transmission and migration routes

- Ingestion of eggs containing L_3. **Tracheal migration**
  Prepatency: 8 weeks; larvae may invade striated muscles
- **Somatic migration** and **lactogenic infection** of kittens
- **Transmission of L_3 in paratenic hosts**

Toxocara canis ***Toxocara cati***



Clinical signs

- Respiratory disorders (cough)
- Gastrointestinal disturbans (catharal enteritis, mushy faeces, vomiting, abdominal bloating)
- General symptoms (dehydration, anaemia, scrubby coat, emaciation)
- Peripheral eosinophilia

THERAPY – *T. cati* (adult cats)

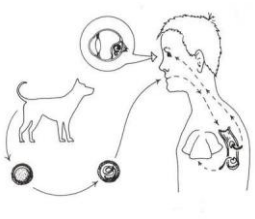
Active substance	Dose	Administration
Emodepsid	3 mg/kg	spot-on
Fenbendazol	50 mg/kg/day resp. 100 mg/kg	p.o. 3 days
		p.o.
Flubendazol	22 mg/kg/day	p.o. 2 days
Milbemycín	2 mg/kg/day	p.o.
Moxidektín	1 mg/kg	spot-on
Pyrantel	20 mg/kg/day	p.o.
Selamektín	6 mg/kg	spot-on

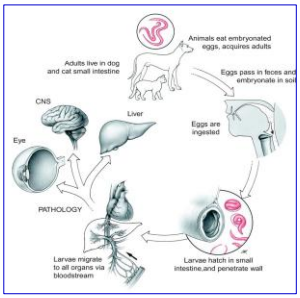
Protection of kittens

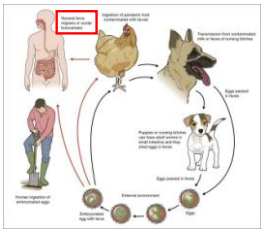
- **at the age of 3, 5, 7 and 9 weeks**
- **every month to 6 months of age**
- **quarterly ("outdoor cats"), i. 4x a year**
- **"Indoor cats" - according to coprological examination**

ZOONOSIS

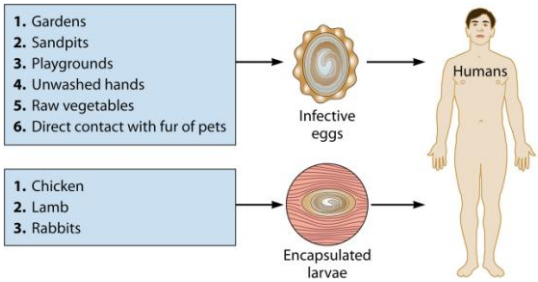
Human toxocarosis







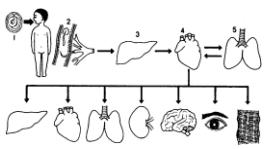
Ways of infection

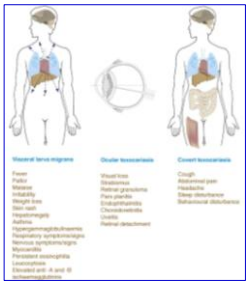


Visceral and Ocular Larva Migrans (LMV, LMO)

Two distinct patterns of larva migrans infection are recognized:

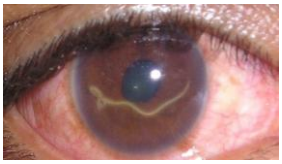
- **visceral larva migrans**
- **ocular larva migrans**
- These clinical syndromes result from the **systemic migration** of the **larval forms** of animal helminthic parasites.
- *Toxocara species*, the common roundworms of dogs and cats, are the usual cause.
- The disease affects mainly children.





Visceral Larva Migrans

Cutaneous manifestations such as chronic urticaria, chronic pruritus, and miscellaneous eczema, in patients with *Toxocara* antibodies.

Ocular Larva Migrans

Pathogenesis

➢ **The parasite cannot complete its life cycle in humans** as it does in the animal host.

➢ The larvae persist in tissue, where they evoke a granulomatous reaction and eventually die.

➢ The **clinical manifestations depend on the amount** of tissue damage caused by the invading larvae and **on the associated immuno-mediated inflammatory response**.

Diagnosis

➢ The diagnosis of visceral larva migrans is usually suggested by the clinical findings of visceral involvement in association with hyper gammaglobulinemia, leukocytosis, and eosinophilia.

➢ Liver biopsy may be diagnostic, although the larvae are difficult to find even in the presence of eosinophilic granulomas.

Treatment in Humans

VML-antiparasitic drugs (albendazole or mebendazole) usually along with anti-inflammatory medications.

Control

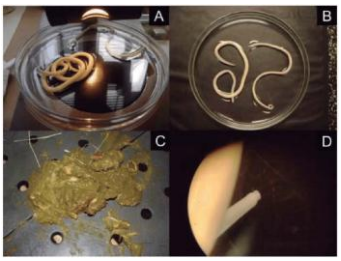
Removal of dog and cat droppings
Personal Hygiene

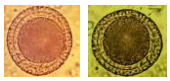
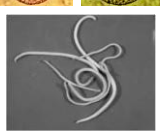


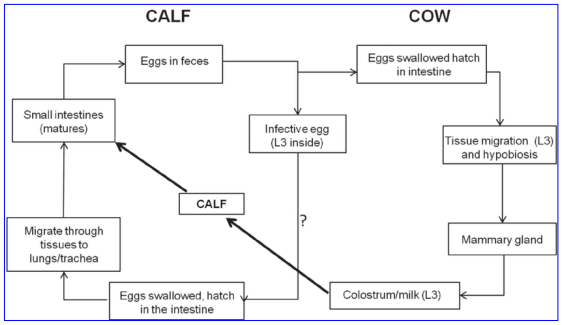

Toxocara vitulorum

/ Toxocarosis in cattle and buffaloes

➢ male: 6-25 cm
➢ female: 8-30 cm
➢ small intestine
➢ common in tropical areas, rare in Central Europe





- disease - intensity of infection;

- a cough
- colic pain
- convulsions
- lassitude movement disorders
- butt paralysis (already in the 2nd week)
- acetone odor of exhaled air and urine - seeping flesh = confiscate.
- lymphocytosis, eosinophilia, neutropenia

Family: Anisakidae (Genera: ***Anisakis, Contracaecum, Pseudoterranova, Terranova,***

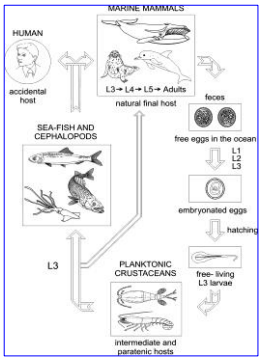
Anisakoid type

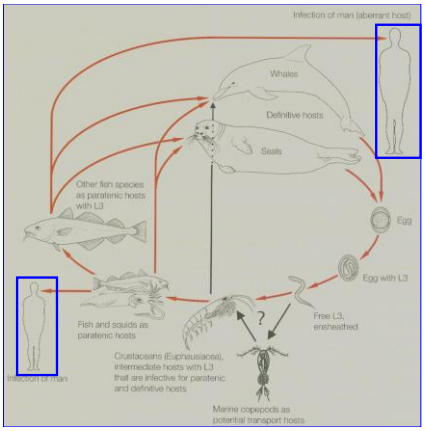
- **Life cycle is indirect**;
- FH **marine mammals**;
- IH - first: planktonic invertebrates (crustaceans);
- Paratenic host: marine fish and squid (herring, mackerel, salmon, cod, tuna, haddock);

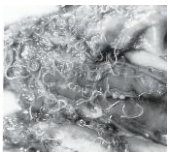
Contracaecum rudolphii, Anisakis pegreffii, A. physeteris, A. schupakovi, A. simplex, Pseudoterranova decipiens ...

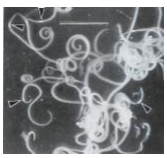
Anisakis simplex / ANISAKIDOSIS

- IH: aquatic crustaceans;
- Paratenic H: fish, cephalopods;
- FH: Marine mammals
- Danger to humans - larvae - strong allergen;
- Human-consumed live larvae can penetrate the stomach wall into the internal organs;

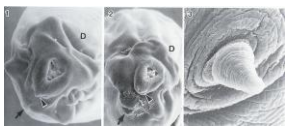


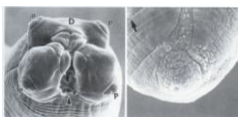






Anisakis simplex





Pathology and Symptoms:
- Usually asymptomatic.
- If larvae penetrate stomach, can cause abdominal pain, nausea, sometimes vomiting.
- If larvae wander, mimics other diseases.

Diagnosis: Frequently seen with endoscope. Immunodiagnosis.

Treatment: Removal with biopsy forceps. No drug treatment.

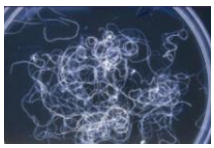
Prevention: Don't eat undercooked, raw, salted, or pickled fish. Can be prevented by blast freezing or cooking.

The European Food Safety Authority (EFSA) has been asked by the European Commission to assess food safety resulting from possible allergic reactions to parasites in fish products and to assess the method to reduce the risk of infection.
The **EFSA Biohazard Panel** (BIOHAZ) concluded in its opinion that the only parasite in fish products for human consumption that could cause allergic reactions is Anisakis, a parasitic worm whose larvae can be found in fish meat.

Allergic reactions to Anisakis include **gastroenteritis**, **rheumatological** and **dermatological symptoms**.

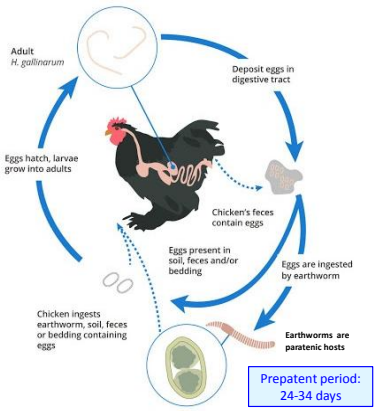
Superfamily: **HETERAKOIDEA**
Family: **Heterakidae** ***Heterakis***
Ascarididae ***Ascaridia***

- Veterinary interest
- Predominantly as poultry parasites
- Caudal alae and a precloacal sucker at the posterior end of the males

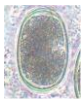


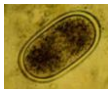
Heterakis gallinarum
Heterakiosis in poultry/chickens, Galliformes
H. dispar /water poultry
H. spumosa /rodents

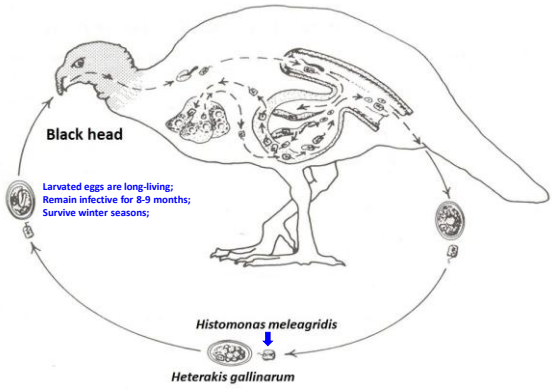
➢ **caeca**



63-85x41-48 µm

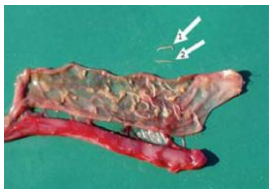






Pathogenesis and clinical signs

- digestive disorder;
- typhlitis with haemorrhages and formation of nodules in mucosa;
- inapetence;
- diarrhoea;
- weakness;
- lagging in growth;
- reduced egg production;



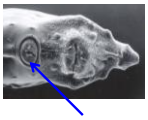
Diagnosis, therapy, prevention

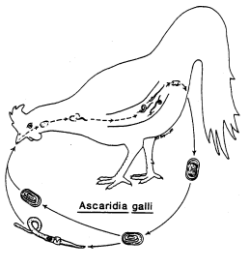
- flubendazole, levamizole

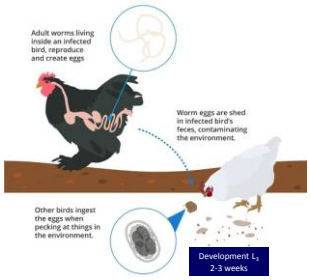
Genus: *Ascaridia*

Ascaridia gali; A. columbae; A. dissimilis /Ascaridiosis

- chicken, turkey, pigeon, pheasant, peacock, ...;
- small intestine;
- yelowish adults;

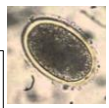




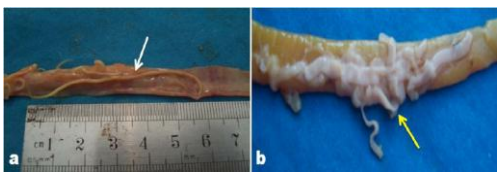


Prepatent period: 29-50 days

Infection by ingestion of larvated eggs, or by paratenic hosts;
Ascaridioid type of development, **no migration of post infection larvae**!!



77-94x43-55 µm
Thick shelled with smooth surface



Ascaridia galli

Pahogenesis and clinical signs

- Epithelial and tissue damage (destruction of glands of Lieberkühn);
- Enteritis;
- Obstruction the intestine;
- Young animals are predominantly affected;
- Food intake, reduced weight gain;
- Anemy;
- Immature and adult worms may migrate up to stomach, the cloaca, into oviductus, liver,...

When the infection is over, the poultry is immune.

Diagnosis, Therapy, control

- Coproscopic detection of eggs; inspection of worms at necropsy;
- Reduce the risk of re-infection from the environment;
- Change litter regularly;
- Never store animals on an old - infected bedding;
- Keep the litter dry;
- Never feed animals directly from the litter;
- Use a targeted deworming program to prevent excessive egg contamination;
- **Piperazine** - single dose, 50 mg / animal (> 6 weeks), 100 mg / animal (> 6 weeks), in feed 0.2% to 0.4% or in drinking water 0.1% -0.2%; it can be given to the marrow at a dose of 100 mg / animal (older than 12 weeks) or 200 mg / animal (older than 12 weeks).
- Combination of **piperazine** (0,11%) and **fenothiazine** (0,50 - 0,56%) jedno rázové ošetrenie;
- **Levamizol**;