

ORDER ASCARIDIDA

I. Intro:

A. Morphology: Large adult worms; host specific; mouth surrounded by 3 fleshy lips. Eggs are thick-walled, distinctive looking, and contain a single cell.

B. Life cycle: There are many superfamilies with diverse lifecycles in this Order.

Examples:

Toxocara canis in dogs, *Toxocara cati* in cats

Toxascaris leonina in dogs and cats *Toxocara vitulorum* in cattle *Parascaris equorum* in horses

Ascaris suum in pigs

Baylisascaris procyonis in raccoons

Ascaridia galli in birds, and *Heterakis gallinarum*, cecal nematode of poultry

Anisakis in marine mammals

In general, ascarids infecting terrestrial host species, have thick-walled, highly resistant, eggs that can survive for years in the environment. Hosts are infected by direct ingestion of the egg (containing infective larva), or by ingesting an infected paratenic host; for some ascarid species transplacental or lactogenic routes lead to very early infections in neonates.

II. *Toxocara canis*: found in the small intestine of dogs.

A. Morphology: white, thick-bodied worms, 50 to 180 mm long, expanded **cervical alae** at the anterior end (=‘arrowhead’ worms). Adult female worms produce large numbers of eggs that are brown, single-celled and have rough pitted surfaces.

In the environment, the infective L2 larva matures within the sturdy egg shell in ~ 4 weeks, and remains dormant until the ‘egg’ is ingested.

B. Life cycle: The routes of infection are determined by the behavior and age (immune status) of the host, and whether it is a definitive or paratenic host.

Adult dog Infection can occur by (1) direct ingestion of the infective egg which hatches in the stomach. The larva penetrates the intestinal wall, reaches the liver via the portal vein. After migrating for several days in the liver, the larva gets to the caudal vena cava, to the heart and the pulmonary artery. Most of the larvae within an adult dog will be transported by systemic circulation to various organs and somatic tissues where they encyst as **arrested infective larvae** (SOMATIC MIGRATION). Some of the larvae will leave the pulmonary artery, enter the alveoli in the lungs, move up the trachea (TRACHEAL MIGRATION) to the pharynx, where they are swallowed and will mature into adult worms in the small intestine. Adult dogs can also be infected (2) by ingestion of a paratenic host (in paratenic host, ingested egg hatches to release the larva which encysts and arrests in the somatic tissue). Most of these larvae reactivate in the dog’s stomach and mature into adult worms in the small intestine.

Neonates & Immunocompromised dogs Infection can occur by (1) direct ingestion of an infective egg, or (2) by ingestion of a paratenic host. Most of the larvae will display TRACHEAL MIGRATION and will return to the intestinal system and mature into adult worms in the small intestine. Neonates are also infected (3) by transmission of infection from the mother to the pup; arrested larvae in the somatic tissues of the female are reactivated during late pregnancy, and migrate via the uterus into the tissues of the developing puppies (TRANSUTERINE). A low level of larvae may also be acquired via the milk (TRANSMAMMARY) as seen with *Ancylostoma caninum* hookworm infection in puppies.

WHY ARE MOST NEWBORN PUPPIES INFECTED WITH *Toxocara* and *Ancylostoma*?
HOW COULD THIS BE PREVENTED?

Prepatent period is ~ 4 to 5 weeks if infected by ingestion of eggs.

Prepatent period is ~ 3 weeks if infected prenatally (*in utero*).

- C. Pathogenesis: Young puppies experience gastroenteritis from developing larvae and adult worms in the small intestine; rarely do migrating larvae cause pneumonia.
- D. Zoonosis: In humans, especially children, visceral and ocular larva migrans is still a major public health concern in the U.S. Why is it called 'visceral' larva migrans as opposed to the 'cutaneous' larva migrans seen with *A. caninum* infection? Ova are not infective until 3 – 4 weeks after being laid. 14% of U.S. population is positive for *Toxocara* antibodies.
- E. Diagnosis: Puppies show abdominal pain, potbellied conformation, poor coat condition and often a fetid, mucoid diarrhea; eggs appear on fecal exam only after patency (~3 to 5 weeks). Adult worms or larvae may be vomited or shed in the feces as a consequence of large burden. Differentiate the mature nematode from the stomach nematode *Physaloptera*.
- F. Treatment and Control: Many anthelmintics are effective against adults and larvae in the intestinal lumen; few drugs are effective against arrested or migrating larval stages.

Manage and treat pregnant dogs to reduce prenatal infection of feti. Clean the environment, remove feces.

Treat puppies with fenbendazole, or pyrantel pamoate **at 2-3 weeks of age and repeat every 2 weeks** until 2 months old (when monthly treatment regime begins) to control contamination of environment with eggs and prevent reinfection of mother and puppies, and decrease risk of visceral larva migrans in children.

Also, to prevent transuterine and transmmary transmission of *Toxocara canis* infection from the **pregnant female**, fenbendazole can be given daily at 50 mg/kg starting at the 40th day of gestation and continuing until two weeks after whelping. Selemectin, and possibly other monthly topical prophylactic drugs used for heartworm prevention, effectively kill larvae that have migrated to puppies when mother is treated at or near parturition.

WHY SHOULD PUPPIES BE REPEATEDLY TREATED AT 2 WEEK INTERVALS?

III. *Toxocara cati* (new name *T. mystax*): found in the small intestine of cats.

- A. Morphology: Stout bodied, 30 to 120 mm long, smaller than *T. canis*. Cervical alae are very prominent and striated.
- B. Life cycle: Unlike *T. canis*, there is **no transuterine infection**. The major routes of infection include
 - (1) direct ingestion of infective eggs; larvae released from the eggs can undergo tracheal or somatic migration (arrested larvae). Unlike *T. canis* in dogs, both routes of migration are common throughout the life of a cat.
 - (2) **transmammary transmission** is important during lactation if the queen is infected during late pregnancy resulting in transfer of L3 via the milk to the nursing kittens. These larvae mature into adult worms in the intestine of the kitten without migration.
 - (3) ingestion of infected paratenic hosts. Larvae mature into adult worms without migration.

Prepatent period is ~ 8 weeks if tracheal migration occurs but is much shorter if larvae mature directly into adult worms in the small intestine.

- C. Pathogenesis: *T. cati* causes a less severe gastroenteritis than *T. canis*. In children, visceral larva migrans is a public health concern as with *T. canis*.
- D. Clinical signs: These are similar to *T. canis*, but not as severe; potbellied conformation, poor coat condition, and intermittent diarrhea.
- E. Treatment and Control: The treatment is the same as for *T. canis* except that treatment can be started at 6 weeks of age (earlier in puppies due to transplacental infection). Pyrantel and praziquantel (Drontal) approved for use in kittens ≥ 4 weeks. Sanitation; prevent access to paratenic hosts.

- IV. *Baylisascaris procyonis*: found in the small intestine of raccoons, rarely in dogs.
- A. Morphology: Adults are 100 to 220 mm long with the typical ascarid body shape (larger than *Toxocara* or *Toxascaris*, but cervical alae not grossly visible. Eggs are smaller than *Toxocara* eggs, are brown and have a finely granular surface rather than the rough surface of *Toxocara*. Important to distinguish *Baylisascaris* from *Toxocara* and *Toxascaris* because it carries a much greater risk of lethal visceral larva migrans in all vertebrates including man and it is being found sporadically as a patent infection in dogs.
 - B. Life cycle: Infection by (1) direct ingestion of eggs or by (2) ingestion of infected paratenic host. In the raccoon, ingestion of eggs is followed by tracheal migration.
 - C. Pathogenesis: The main concern is a **lethal form of visceral larva migrans** that has been reported in many animal species, including man.
 - D. Clinical signs: Neurological symptoms from larvae migrating in the CNS of aberrant hosts.
 - E. Treatment and Control: Piperazine causes the expulsion of larvae and adults (these can be used to confirm a suspected diagnosis in dogs); in dogs, the common anthelmintics including the combined heartworm preventative formulations appear to be effective. Prevent exposure to raccoon feces, e.g. contaminated hay from barn storage.

- V. *Parascaris equorum*: found in the small intestine of equids, especially <2 year olds.
- A. Morphology: Adults are very large, up to 500 mm long. Typical thick-bodied ascarid; location and host specificity aid in easy identification.
 - B. Life cycle: Only ONE route of infection i.e. **by ingestion of infective egg (containing L2)**. Eggs hatch in the small intestine to release the larvae which penetrate the gut wall, enter the portal vein and are carried to the liver. Larvae migrate in the liver for 2 to 4 days before traveling to the lungs via the heart. Within the pulmonary capillaries, the L3 larvae penetrate the alveoli, migrate up the trachea and are coughed up and swallowed -- return to the small intestine 2 to 4 weeks after initial infection. Prepatent period ~ 80 days. Egg requires 10-14 days to become infective. One adult can produce up to 200,000 eggs/day.
 - C. Pathogenesis: In young horses, the lungs can be congested by repeated infections due to allergic reactions to migrating larvae. In the intestine, catarrhal enteritis, occlusion and perforation can occur (especially with heavy worm burdens - remember the size of each adult worm!). Hypoproteinemia. Can jeopardize full developmental potential of the foal.
 - D. Clinical signs and diagnosis: Foals with diarrhea or feces with fetid odor, potbellied conformation, rough coat; signs may appear before eggs are detectable in feces. Respiratory problems occur when repeating infections sensitize the lungs to the potent ascarid allergen. Ultrasonographic diagnosis of ascarid impaction (see assigned reading).
 - E. Treatment and control: Prevent exposure to infective eggs i.e. sanitary environment, clean the teats and udder of brood mares -- **eggs are highly resistant, long-lived and sticky!** Treat foal at 2 months of age, every 2 months until 1-year-old using a broad spectrum anthelmintic. With heavy infections (well past prepatent time at 3-6 months of age), a highly efficacious drug (e.g. benzimidazole, moxidectin or ivermectin) should NOT be used for the first time as it may lead to death of the foal (from impaction or anaphylactic reaction) -- begin treatment with a mild drug e.g. piperazine, and use mineral oil. **Ivermectin resistance has been documented for *Parascaris equorum*.**

ASSIGNED READING: "What Is Your Diagnosis?" JAVMA 239: 435-436 (2011): see supplemental course materials:

<http://parasitology.cvm.ncsu.edu/http://parasitology.cvm.ncsu.edu/vmp930/supplement.html>

VI. *Ascaris suum*: found in the small intestine of pigs; most economically important helminth infection in swine. Closely related (same species?) *Ascaris lumbricoides* in humans.

- A. Morphology: Adults are 150 to 400 mm long, with three prominent lips at the anterior end, but no cervical alae. Eggs are thick shelled, rough, brownish-yellow, oval.
- B. Life cycle: Female lays 200,000 or more single cell eggs/day. Infective larva develops within egg in ~10 days. Infection is ONLY by ingestion of the infective egg. Larvae hatch within the intestine and burrow out of the gut into the liver where they migrate for a few days before continuing on to the lungs via the heart; larvae can be found in lung alveolar spaces, and bronchioles 5-7 days after ingestion of the egg. Larvae move up the bronchi and trachea, are swallowed and return to the small intestine by 7-8 days post infection, where they mature. Prepatent period is 60 days.
- C. Pathogenesis: Severe lesions can occur in the lungs of piglets due to repeating larval migrations that produce small hemorrhages, edema, and infiltration of the pulmonary parenchyma by eosinophils and other inflammatory cells. Focal fibrosis '(milk spots') occurs in the liver of repeatedly infected pigs. In the small intestine, adults may cause hypertrophy of the tunica muscularis and interfere with nutrient absorption.
- D. Clinical signs and diagnosis: Piglets may show signs of pulmonary disease with coughing ("thumps"=rapid, shallow, audible expiratory effort); may find larvae in the sputum. Growing pigs may be stunted. Diarrhea may be seen with heavy infections; may occur before patency/appearance of eggs in feces.
- E. Treatment and Control: Eggs are numerous, very resistant and sticky -- therefore difficult to eliminate the problem in pigs exposed to pasture. To protect piglets, treat sows coming off dirt 2 weeks prior to farrowing and wash them thoroughly before entering the farrowing area, to remove adhering eggs. Clean frequently to wash the eggs away before they become infective. Fenbendazole, levamisole & ivermectin are all effective against adult worms; **only pyrantel tartrate kills newly hatched larvae** and is given as a continuous feed additive. Infection is NOT a problem in well-managed confinement units (sows not on dirt).

VII. *Ascaridia* sp: *A. galli* is found in the small intestine of chickens, turkeys and many birds. *A. dissimilis* is found in the small intestine of domestic and wild turkeys.

- A. Morphology: 50 to 100 mm long. Males have a pre-anal sucker.
- B. Life cycle: The only route of infection is by ingestion of infective eggs. Larvae lie within the mucosa; adults in the lumen of the small intestine. Prepatent period is 4 to 8 weeks.
- C. Pathogenesis: Lesions usually seen in young (under 3 months) birds; i.e. **hemorrhagic enteritis, anemia and diarrhea, intestinal blockage**. Can occur during phase of larval development before the infection is patent (i.e. no eggs in feces).
- D. Treatment and Control: Hygromycin B in feed kills larvae as they hatch from eggs in the intestine. Keep young birds separated. Clean up the environment to remove eggs.

VIII. *Heterakis gallinarum*: found in the cecum of chickens, turkeys and other birds. “Cecal worm”.

- A. Morphology: These are smaller than *Ascaridia*; 5 to 15 mm long. The esophagus has a posterior bulb and lateral alae are present along length of body. Males have a very prominent pre-anal sucker.
- B. Life cycle: Infection is by ingestion of infective larvated eggs or an earthworm (paratenic host containing infective larva). Prepatent time is 30 days.
- C. Pathogenesis: *Heterakis* worms cause **minimal pathology in chickens**. HOWEVER, the *Heterakis* eggs and larvae are carriers of the protozoa *Histomonas meleagridis*, allowing transmission from one bird to another. *Histomonas* causes a severe disease in turkeys (“blackhead disease”) but is not pathogenic in chickens. In turkeys, the protozoa invade the gut wall causing inflammation and necrosis of the cecum and liver. The protozoa are protected within the nematode eggs and larvae.
- D. Treatment and Control: Many effective anthelmintics. Also, control by cleaning up the environment; **don't mix turkeys with chickens or house them on areas previously occupied by chickens** since *Heterakis* infective stages can remain in the environment for years using the earthworm paratenic host. The threat to turkeys is from possible infection with *Histomonas* which is not of concern in chickens!