SUPERFAMILY ANCYLOSTOMATOIDEA (HOOKWORMS)

Introduction:

I. Morphology: Buccal cavity - teeth or cutting plates at the anterior opening of a large buccal cavity; dorsal flexion of the anterior end i.e. ‘hooked’. Eggs are typically strongyle-like, and are designated as ‘hookworm eggs’ if seen on a small animal fecal floatation.

II. Life cycle: Infection is by ingestion of or skin penetration by free-living infective L3 larvae. Additionally, *Ancylostoma caninum* can infect by the transmammary (lactogenic) route (puppies not kittens); i.e. L3 are acquired by ingestion of milk while nursing. Larvae migrate through the tissues of the host to establish as adult worms in the lumen of the small intestine where they attach and feed on blood. One adult female may shed as many as 16,000 eggs per day! Go to CAPC site www.capcvet.org for really good video of adult worm.

III. *Ancylostoma caninum*: adults are found in the small intestine of dogs only. (3 teeth, pair) *A. tubaeforme*: cats only. (3 teeth, pair) *A. braziliense*: dogs and cats, especially in tropical areas. (1 tooth, pair) *A. duodenale*: man (2 teeth, pair) *Uncinaria stenocephala*: Cutting plates and much less pathogenic (see Nematode Appendix section)

*Ancylostoma caninum*

A. Morphology: Adults are 10 to 16 mm long, grey-red; the buccal cavity is dorsally bent and has three teeth on either side of the ventral aspect of the anterior opening. Eggs are 56-75 um by 34-47 um; usually at 8-celled morula stage in feces.

B. Life cycle: Eggs shed in the environment hatch to release larvae which feed on bacteria in the feces/environment, to become infective L3 larvae. Development to the L3 stage is rapid under warm, moist conditions i.e. ~5 days. Infective L3 enter the host by skin penetration or ingestion. Infection may also occur by ingestion of an infected paratenic host. L3 have 2 choices:

1. to undergo direct development into L4 which become young adults as early as 7 days post-infection. Prepatent period is about 3 weeks (15-18 days).
2. arrest as L3 in the surrounding tissue. Arrested L3 can remain in this state for months to years, and when reactivated, they resume normal development to become L4, then adults.

Reactivation of arrested L3 is triggered by factors such as stress, removing adult by deworming and near-term pregnancy. During late pregnancy, arrested L3 in the tissues of the mother reactivate and lodge in the mammary glands, ready for transmission to the puppies during lactation (in dogs not cats). Note that a previously infected animal may have no evidence of hookworm eggs in the feces but could have arrested L3 in its tissues; arrested larvae are NOT very susceptible to drug treatment due to reduced metabolic activity.

C. Pathogenesis: Is due to the blood-feeding adult worms. Classic disease -- *acute anemia* in nursing puppies with large worm burdens. (50 adult worms can remove as much as 3 ml of blood per day). L3 acquired in the milk while nursing on the infected mother mature into blood-sucking adults in the small intestine. In older dogs, small amounts of arrested larvae can also reactivate, migrate to the intestinal tract and develop into adult worms, causing a chronic blood-loss disease. In cats, a chronic condition of weight loss and poor condition could be due to *A. tubaeforme*. No peracute disease in cats because no L3 in milk.

D. Resilience: Adult dogs are frequently infected and shed hookworm eggs but are more resilient to hookworm disease than puppies due to (1) acquired immunity (from previous infections), (2) natural defense mechanisms (even if previously uninfected), (3) ability to compensate for blood loss, and (4) premunition, which is the inhibition of further infection exerted by the resident population of hookworms.
E. Clinical signs: Seasonal; especially with warm, moist conditions. Often seen in newborn and young pups or immunocompromised, older dogs exposed to heavy infection. Anemia, edema, emaciation, bloody diarrhea or tarry stool. Peracute disease (no eggs in feces) occurs in newborns 1-2 weeks of age, due to transmammary transmission of infection.

F. Treatment and Control: Treat at the first sign of anemia with an anthelmintic; iron supplementation and blood transfusions may be required in very small animals suffering extreme blood loss. Treatment should be repeated weekly due to reactivation of arrested larvae or continuing exposure to infection. In general, puppies should be treated at 2-3 weeks of age with a drug that controls hookworms and ascarids, and treatment should be repeated every 2 weeks till 2 months of age when the animal is put on a heartworm prevention program. The monthly heartworm preventatives such as pyrantel/ivermectin (HeartGard Plus), milbemycin (Interceptor), selamectin (Revolution), moxidectin (Advantage) as well as other drugs are very effective in controlling ADULT hookworms. Available drugs are NOT very effective in completely eliminating arrested larvae in tissues. WHY?

Multi-drug resistant *Ancylostoma caninum* has been described in dogs from a variety of locations in the USA including Raleigh, North Carolina. Standard doses of fenbendazole, ivermectin, and moxidectin have failed to clear adults as judged by fecal recheck within 14 days of treatment to exclude possibility of arrested larvae repopulating the small intestine. Emodepside in Profender is still effective against resistant *A. caninum*.

Keep run areas clean and dry; sodium borate or bleach will kill larvae. Remove feces. To control the infection in the pregnant female, Standard monthly doses to mother of selamectin during the periparturient period and at whelping has also been shown to be effective in preventing infection in newborn puppies.

G. Zoonotic disease potential:
1. Cutaneous larva migrans: an itchy dermal lesion caused by the skin penetrating larvae.
2. Eosinophilic gastroenteritis: Rare. *A. caninum* can penetrate human skin, migrate to the intestine, mature to adulthood (not a patent infection i.e. no egg production) and only 1-2 worms seem to cause a painful, eosinophilic enteritis.
ORDER OXYURIDA:

Pinworms are parasites of the large intestine; adult worms have a long, tapering tail and are host specific. *Enterobius* is a common human pinworm which children do NOT get from their pets! Includes the relatively large *Oxyuris equi* in horses and the smaller pinworms in other species.

*Oxyuris equi*:
A. Morphology: whitish, thick-bodied; adult males are only 9 to 12 mm long while females are 40 to 150 mm long. Eggs have a single operculum.

B. Life cycle: Adult females migrate to the anus, protrude their anterior end (location of vulva) outside and deposit a sticky, itchy fluid containing eggs, on the perianal area. Eggs mature within 4 to 5 days, retaining the infective L₃ within the shell. Sticky fluid containing the developing eggs dries and flakes off the horse to eventually be ingested from the environment by the same or another horse. Ingested eggs hatch in the small intestine and release the L₃ which enter the mucosal crypts of the large intestine. The L₄ develop here and emerge into the bowel lumen to begin feeding. Prepatent period is ~ 5 months.

C. Pathogenesis: Large numbers of adults and larvae can cause colitis. More commonly, horses display perianal itching due to the fluid and eggs deposited in this area.

D. Clinical signs and diagnosis: The tailhead of infected horses is rubbed bare and appears scruffy. To diagnose, press 'scotch-tape' on the perianal region and examine for eggs with a single operculum. (May need to clean the perianal area of debris, and tape-test the next day.)

E. Treatment and control: Many anthelmintics such as fenbendazole are effective, but there are an increasing number of reports of *Oxyuris equi* resistance to ivermectin and moxidectin. Keep stall area clean to prevent environmental contamination with eggs.