AGRICULTURAL EXPERIMENT STATION
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SR-109

Strongyles in Horses

Update 2015

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Introduction

Parasites live in a host from which they obtain food and protection. They may harm but usually do not benefit the host. The word "parasite" is derived from the Latin and Greek languages meaning, in general, "one who eats at the table of another." It is said that a "good" parasite does not overtly harm or kill its host. It is theoretically possible that a more benign parasite (e.g. *Gasterophilus* spp.) is much "older in eons of time" and it and its host have adjusted better to each other than a conceivably "newer" parasite (e.g. *Strongylus* spp.) which may be more harmful to its host.

Taxonomy

Horses can harbor over 100 species of internal parasites. About one half of these species are nematodes in the strongyle group (family Strongylidae Baird, 1853). They are separated taxonomically into two categories—large strongyles (subfamily Strongylinae Railliet, 1893) and small strongyles (cyathostomes) (subfamily Cyathostominae Nicoll, 1927). Historically, large strongyles included only those strongyles in the genus *Strongylus* Müller, 1780. In this discourse, the latter designation will be used for large strongyles i.e. it includes only *Strongylus* spp. More recently in revision of the taxonomy of the strongyles, based on morphology, large strongyles include, besides, *Strongylus* spp., the genera *Bidentostomum* Tshoijo in Popova, 1958, *Craterostomum* Boulenger, 1920, *Oesophagodontus* Railliet et Henry, 1902, and *Triodontophorus* Looss, 1902. The latter four genera are much less important because, unlike *Strongylus* spp., they do not migrate outside the intestinal tract.

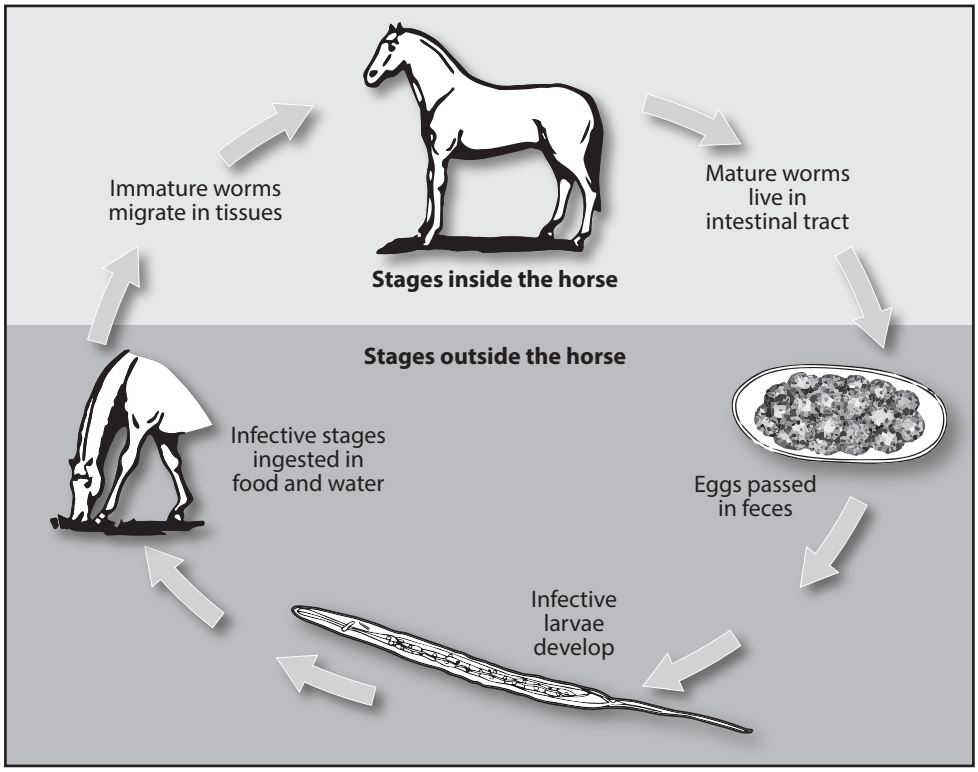

Figure 1. Strongyle life cycle.

Life Cycle

Outside the Horse

Strongyles live as adults in the large intestine (cecum, ventral colon, and dorsal colon) of the horse and lay eggs that are voided in the feces (Figures 1 and 2). In the environment, an egg embryonates to the first stage larva (L_1) which hatches and then develops to the second stage larva (L_2), and finally to the third stage larva (L_3) which is the infective stage (Figure 3). The L_1 and L_2 feed on organic matter, but the L_3 does not feed but subsists on internal nutrients. The L_3 is the infective stage which typically crawls up on pasture vegetation, especially under moist conditions which make movement easier than dry situations, and is then accidentally ingested by the grazing horse (Figure 4). Inside the horse the L_3 develops to the fourth (L_4) and then fifth (L_5) (adult) stage.

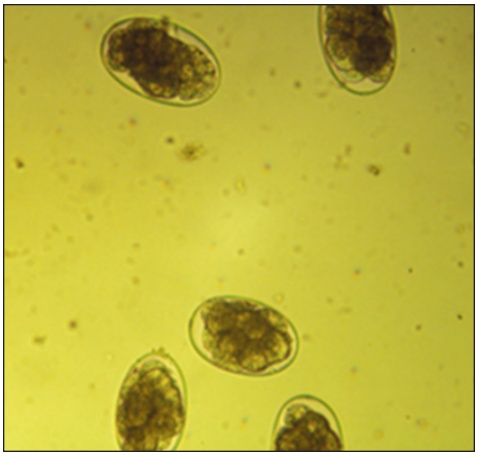

Figure 2. Strongyle eggs (small and large strongyle similar).

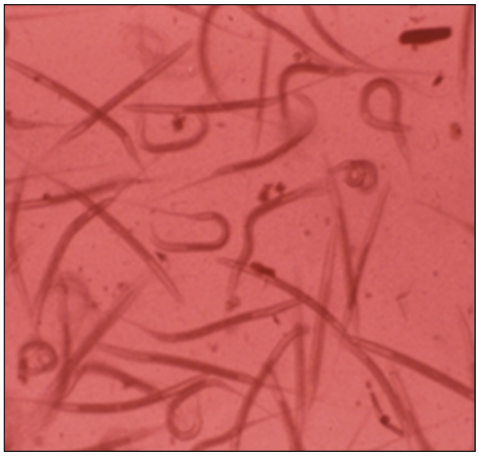

Figure 3. Free-living third stage (L_3) infective strongyle larvae.

Figure 4. Free-living L_3 in moisture droplet on grass.

Inside the Horse

Within the genus *Strongylus* are four species—*S. asini, S. edentatus, S. equinus, and S. vulgaris.* These species are the most pathogenic of the strongyles because they can cause colic and even death of horses. *Strongylus vulgaris* is the most damaging of the four species. Detrimental effects of these parasites usually are most evident during migration of immature stages in organs outside the gastrointestinal tract.

Strongylus vulgaris will be highlighted because it is the most pathogenic parasitic nematode species in horses. When L_3 are ingested they penetrate the intestinal mucosa, mainly of the posterior part of the small intestine, cecum, and ventral colon, and enter arterioles in the walls of these organs. About two weeks later they begin arriving and accumulating primarily in the cranial mesenteric artery (CMA) (Figures 5 and 6). There they undergo development to the L_4 and L_5. Their presence stimulates the immune system

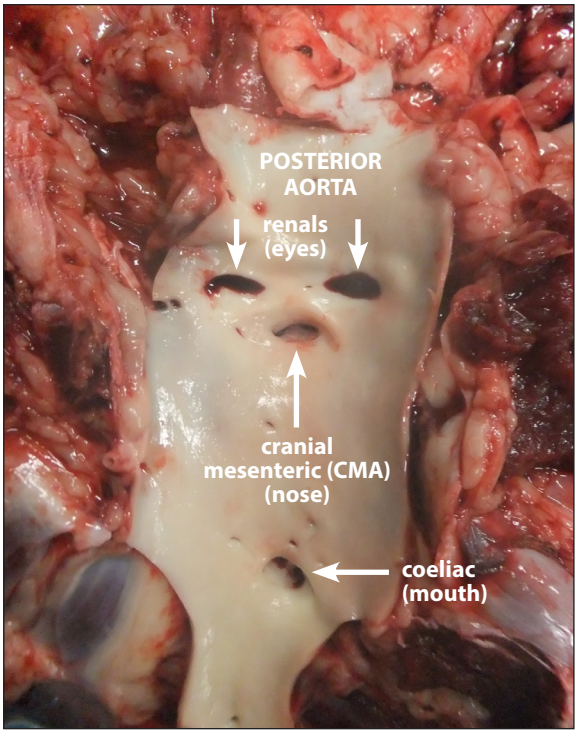

Figure 5. Posterior aorta showing orifices of some branching arteries with their equine anatomic names (= some features of a human face): renals (eyes), cranial mesenteric (nose), coeliac (mouth). Note: The cranial mesenteric artery is the major location of migrating *Strongylus vulgaris* larvae causing development of an aneurysm.

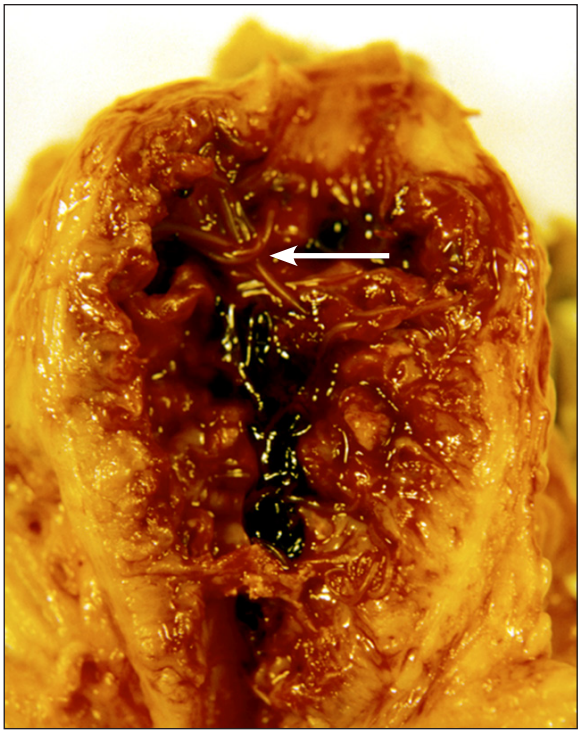

Figure 6. Verminous aneurysm in cranial mesenteric artery with celluar debris and migrating *Strongylus vulgaris* larvae (see arrow) evident.

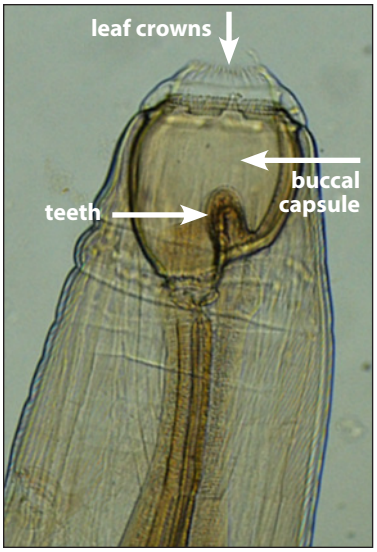

Figure 7. *Strongylus vulgaris* adult male—anterior end.

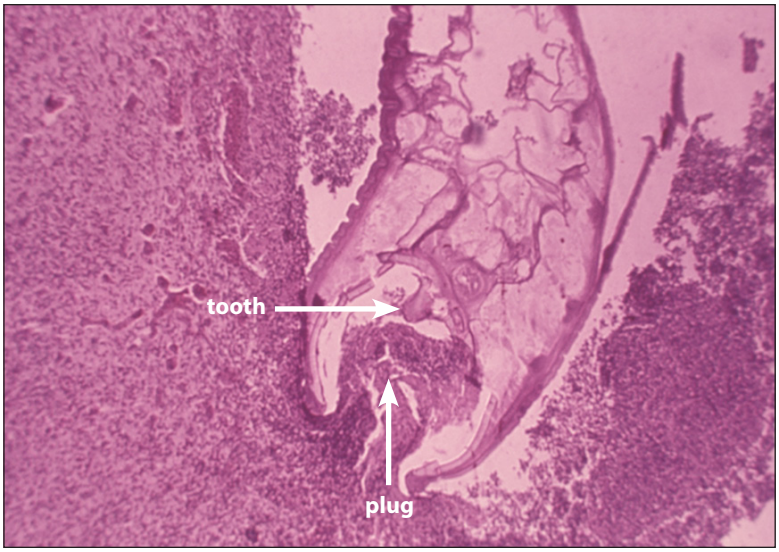

Figure 8. Cross-section of head/buccal capsule of *Strongylus vulgaris* adult attached to plug of intestinal mucosa; plugs are bitten off/sucked in, dissolved and eaten.

to "repel" these invaders. Upon initial infection, this action is a negative feature because the host reaction produces cellular debris which may result in thrombi and emboli that can block blood flow. Thus resulting in chronic or acute colic and possible death of the horse because of lessened or lack of blood supply to the intestine. In addition, there is a condition called a "verminous aneurysm" which is enlargement and thickening of the CMA and adjoining arteries. Acquired immunity to *S. vulgaris* is typical in varying degrees. The prepondence of literature states that the L_4 and L_5 come back to the large intestine via arteries. Some researchers wonder how such large larvae can get back through the small arterioles. They hypothesize that maybe the L_5 that actually come back to the intestine are ones that do not migrate to the CMA. Possibly, L_3 penetrate through the large intestinal wall and undergo development to L_4 and L_5 there on the serosa surface before returning. In any event, L_5 coming back (assuming they left) can be found encysted in the mucosa of the cecum, ventral colon and dorsal colon. After L_5 excyst they locate primarily in the cecum, but also in the ventral colon, which are the usual locations for mature adults that attach to and feed on the mucosa of these organs (Figures 7, 8, and 9). The prepatent period, the time since acquisition of L_3 until females begin laying eggs, is about six months. Future improved methodology may allow more exact determination of the route of migrating larval *S. vulgaris.*

Small Strongyle (Cyathostome) Group

The small strongyle group includes more than 50 species worldwide. Only 10 or 12 species are the most common. Virtually, 100 percent of horses are infected with at least some species of small strongyles. Numbers of these worms are usually lower in older horses that have had time to develop some immunity to them. They are much less harmful than *Strongylus* spp. because the infective third stage (L_3) penetrates only into the lining of the large intestine where it encysts (Figure 10). Here they develop to the fourth (L_4) and sometimes young

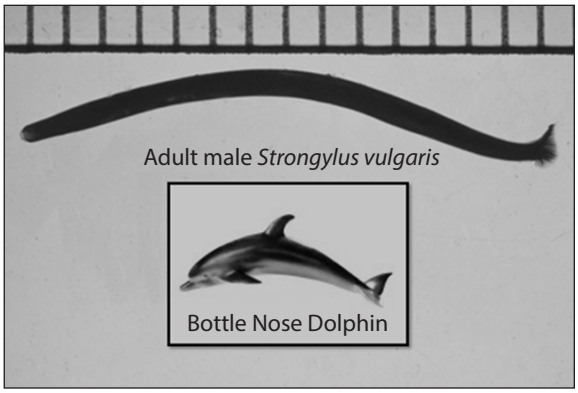

Figure 9. Adult male *Strongylus vulgaris (S.v.)* showing typical "dolphin-type" posture in gross lateral view. Two similarities of *S.v.* and the dolphin are body curvature and tails—bursa of *S.v.* and fluke of dolphin. One difference is that the base of the *S.v.* tail is slightly up curved (each unit on scale = one millimeter representing length of *S.v.*) (size of dolphin not related to scale).

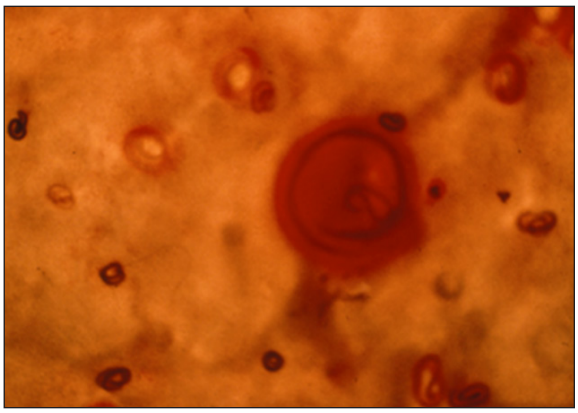

Figure 10. Small strongyle larvae encysted in mucosa—large intestine.

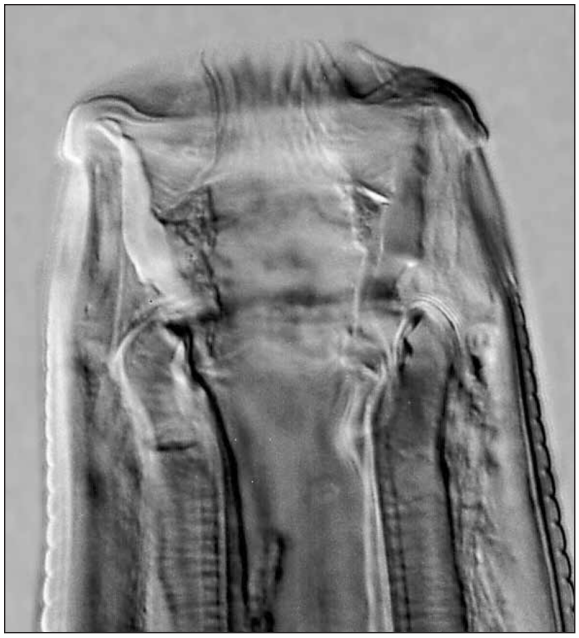

Figure 11. Small strongyle (cyathostome)—head *(Cyathostomum catinatum)*.

fifth (L_5, adult) stages which excyst usually by trickling out into the intestinal lumen and maturing (Figure 11). Also they may remain encysted for long periods. In a recent study, the earliest prepatent period for a small strongyle species *(Cylicostephanus longiburatus)* was about two months. There is great variation in the fecundity of species of strongyles. Under poorly understood circumstances, massive numbers of larval stages can excyst and emerge in a short time, causing severe damage to the intestinal lining, resulting in extensive fluid and protein loss. The condition is called "larval cyathostomiasis," and has been reported more commonly in Europe than the United States. Death can occur from this disease situation. This can be related seasonally, especially in late winter or early spring, and also after deworming. Overall the small strongyles are not considered very pathogenic, except under certain conditions, but they should not be overlooked as disease entities.

Treatment/Drug Resistance

Over several centuries various methods have been advocated for control of internal parasites of horses. It was not until the early 1900s that scientific methods were begun to detect actual efficacy of antiparasitic compounds against horse parasites. The first compound so-tested was carbon disulfide which was proven to be efficacious against horse bots (*Gasterophilus* spp.) and ascarids (*Parascaris equorum*). Since the early 1900s until presently (2015), more than 25 products have become commercially available for control of endoparasites in horses. These compounds are in only a few chemical classes including benzimidazoles (BZs) (e.g. thiabendazole), phenylguanidines (Pro-BZ) (e.g. febantel); imidothiazoles (e.g. levamisole + piperazine), macrocyclic lactones (e.g. ivermectin and moxidectin), organophosphates (e.g. dichlorvos), pyrimidines (e.g. pyrantel) and others (carbon disfulfide, phenothiazine, and piperazine). No new classes of equine parasiticides have been marketed in the past 25 years except praziquantel, which only has activity on tapeworms in horses. Currently (2015), only fenbendazole, oxibendazole, ivermectin, moxidectin, and pyrantel are commercially available for treatment for internal parasites in horses in the USA. Preparations of praziquantel combined with ivermectin and with moxidectin are marketed also.

Phenothiazine was marketed in the early 1940s for control of strongyles in horses. In the late 1950s and early 1960s, there were reports in England and Kentucky of small strongyles resistance to phenothiazine. These were the first indications of any horse parasites resistant to a chemical compound. It should be mentioned that in the early 1950s the barber pole nematode in sheep was documented in Kentucky to be resistant to phenothiazine. This was the first report on drug resistance of any internal parasite species in any animal. Finding of small strongyles resistant to phenothiazine was an indicator (in retrospect) that these parasites would potentially become resistant to other parasiticides used frequently. Thiabendazole, one of numerous benzimidazoles, was commercially available for horses in the early 1960s, but very soon small strongyles were observed to be resistant to this drug. Currently, both of the commercially available benzimidazoles (fenbendazole and oxibendazole) and pyrantel pamoate are ineffective on small strongyles (cyathostomes). The macrocyclic lactones (ivermectin and moxidectin) are now less effective against small strongyles then initially. It was established that these compounds now have lower activity on luminal stages (especially fourth stages) of small strongyles so the life cycle is shortened.

At the present time, in Kentucky and world-wide, *S. vulgaris* is uncommon any more in horses on farms with frequent deworming programs. In other words, drug resistance for these parasites has not been demonstrated like it has with the small strongyles. Constant monitoring needs to be done to determine if parasiticides remain efficacious against this nematode species.

Treatment of all horses in a herd with chemical compounds every six to eight weeks for parasite control has been done for several decades. In later research, it has been found that as horses age, only a few in a herd are shedding

"high" numbers of strongyle eggs. Current opinion is to establish a profile of strongyle eggs per gram (EPG) for older horses and then only treat those with "high" EPG values. Recent research at the University of Kentucky has shown that under most conditions one strongyle EPG count is sufficient to establish this profile for a horse. Thus, only treatment of "necessary" select horses saves money, and also may help prolong drug effectiveness. Even though small strongyles are resistant to the benzimidazoles and pyrantel pamoate, it may still be beneficial to use them, because of their activity on some of the other parasites. Use of ivermectin and moxidectin twice a year (in the spring and fall) is recommended for treatment of older horses. There is no absolute strongyle EPG value that should be used to determine whether it seems necessary to treat a horse. Some recommend treatment for horses with strongyle EPGs of 200, 500, or higher, but this can be decided on an individual basis. In general, there is not a direct relationship between strongyle EPG counts and worm counts. With *S. vulgaris* being rare, there is much more leeway in chemotherapy for parasite control. It is difficult to prove that small strongyle larvae, while they are encysted, and larvae and adults in the lumen of the large intestine cause clinical problems. However, in the future there may be insidious negative effects found that are not detected by current methods. At this time, it is believed that the major benefit of reduction of small strongyle numbers in a horse is lowering of egg deposition on pastures; thus lessening development of infective stages to enter horses. It is obvious that much more research on management practices needs to be done to reduce or supplement use of parasiticides. These might include, for example, removal of feces from pasture, chain harrowing, rotation on pasture with ruminants, fungicidal activity on parasite eggs/larvae and other innovative ideas.

Acknowledgements

This investigation (No. 15-14-019) was made in connection with a project of the University of Kentucky Agricultural Experiment Station and is published with the approval of the director. Research in the animals used in the study was approved by the University of Kentucky Institutional Animal Care and Use Committee (IACUC) under Protocol 2008–0257.

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