

# Down a worm hole: first steps in designing a deworming plan

**In this article, Siobhan McAuliffe MVB DACVIM, Fethard Equine Hospital, Co. Tipperary looks at designing anthelmintic or deworming plans for 'major' parasites in horses**

Parasites of horses can be divided, in a very simplistic fashion, into two groups: the 'major' players or those that are capable of causing significant disease; and the 'minor' players, which rarely, if ever, cause disease and which, in general, are susceptible to most anthelmintics. The major players are: small strongyles (cyathostomes), large strongyles (red worms), ascarids (roundworms) and tapeworms. The minor players are: oxyuris, gastrophilus (bots) and strongyloides. This article looks only at designing anthelmintic or deworming plans for the 'major' players.

When designing a deworming programme, there are several important things to consider. Firstly, the parasites we are aiming to treat. This will vary depending on the population of horses, eg. grazing versus stabled, and will also be dependent on the time of the year. We also need to consider the prepatent period of the parasites we are treating to allow timing our treatments for when they are most effective.

Secondly, the age population we are treating. In general, horses develop immunity to and self-eliminate ascarids by 18 months of age and, therefore, treatment targeting this parasite is only necessary in younger stock.

Thirdly, the other control mechanisms and farm factors that are coming into play. A strategy for a farm that is historically and currently overstocked will be different to one designed for a well-managed farm with rotational grazing and separation of age groups, which, in turn, will be different to that designed for a racing yard or a single horse.

## LIFE CYCLES AND DISEASE

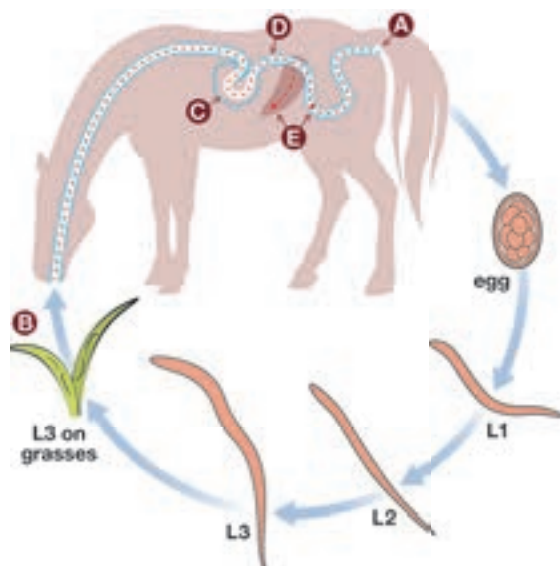
### SMALL STRONGYLES

Infection occurs following ingestion of third-stage larvae on pasture (Figure 1 – lifecycle diagram from European Scientific Counsel Companion Animal Parasites [ESCCAP]). Exsheathment of this stage occurs through contact with gastric fluids and the exsheathed larvae pass through the small intestine. Invasion of the mucosa and submucosa of colon and caecum then occurs. The larvae moult to fourth stage and return to the lumen where a final moult to L5 occurs in adulthood.

Infections acquired indoors are considered rare as the third-stage larvae are found attached to vegetation. Severe cold slows down development of eggs or stops it altogether. Extreme heat (40°C) will kill eggs and L3 in days. In temperate climate, such as that of Ireland, eggs and L3 can survive for six to nine months on pasture. Infection is acquired as soon as

animals start grazing and strongyle eggs are shed from six to 14 weeks after infection.

The most significant disease caused by small strongyles is larval cyathostomiasis (Figures 2-4). This occurs when there is mass emergence of the third-stage larvae from the wall of the caecum and colon leading to diarrhoea and a severe protein-losing enteropathy, which can result in death in many cases. Larval cyathostomiasis is most commonly seen in younger horses (up to six years of age). Severe infestations with mass feeding by adults can also cause poor thrift and weight loss.



**Figure 1: Life cycle of small strongyle. Source: ESCCAP.**

- A: Egg shedding.
- B: Oral uptake of third stage larvae (L3) with grass.
- C: Exsheathment through gastric fluids.
- D: Passage of exsheathed L3 through small intestine.
- E: Invasion of mucosa/submucosa of colon and caecum, moult to fourth stage, return to intestinal lumen and final moult before development to adult stage.



**Figure 2: Adult small strongyles seen on faeces.**



**Figure 3: Severe and rapid weight loss is often seen in cases of larval cyathostomiasis.**



**Figure 4: Preputial oedema as a result of hypoproteinaemia is also frequently seen in cases of larval cyathostomiasis.**

The stimuli for the mass exodus of L3 includes seasonal factors (disease is seen in late autumn and winter in the northern hemisphere but late spring or early summer in the southern hemisphere). Another important stimulus is the synchronous death of the lumen dwelling adults. A frequent scenario is where diarrhoea and a rapid weight loss follow the recent administration of an adulticidal anthelmintic.

### Diagnosis

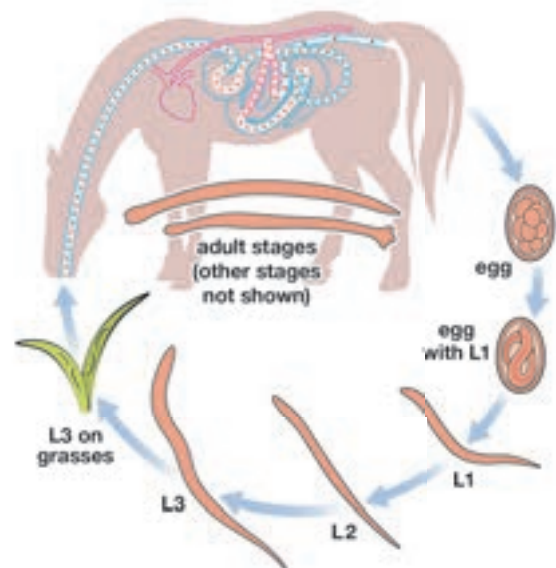
Only patent or adult infections can be diagnosed with the use of faecal egg counts. This is especially important to remember if larval cyathostomiasis is suspected. The emerging larvae will not yet be capable of producing eggs and therefore no eggs will be detected in faeces despite what is a severe parasite related disease. Another important factor in diagnosis is that it is for practical purposes impossible to differentiate small strongyle eggs from those of large strongyles and to complicate things further it has been shown that there is no correlation between egg counts and the actual parasite burden. Larval cyathostomiasis is frequently diagnosed based on history, clinical signs and ultrasound examination of the abdomen where marked oedema of the wall of the right dorsal colon and caecum can be detected.

### Treatment

Given a prepatent period of six to 14 weeks, treatment regimens should start at two months of age. Macrocyclic lactones (ivermectin, moxidectin) are efficacious against intraluminal adults. Moxidectin and Fenbendazole 7.5-10mg/kg are effective against encysted L3. Ivermectin has no efficacy against encysted L3. However, neither moxidectin nor fenbendazole are 100% efficacious against encysted L3 and are much more likely based on the latest available data to have an efficacy of 50-60%. Therefore, disease may still occur following the administration of these products if the number of encysted larvae is large. In simplistic terms all anthelmintic programmes should include the administration of a product effective against small strongyle larvae in the autumn before disease would be likely to be seen.

### LARGE STRONGYLES

These were historically regarded as the most significant parasite of horses. They are also known as migratory strongyles. Infection occurs following ingestion of L3 on grass. Exsheathment occurs in the small intestine with penetration into the wall of the large intestine and moulting to L4, migration on or in intima of arteries of large intestine followed by migration to the cranial mesenteric artery and moulting to pre-adult stages. These then migrate to the large intestine and penetrate the wall to enter the lumen where development to adults is completed (Figure 5). Depending on the species (*S. edentatus*, *S. equinus*, *S. vulgaris*), larval migration can involve the mesenteric arteries, liver and sub-peritoneal tissue, pancreas and renal tissue. Because of such lengthy migration, prepatent periods are also long, varying from six to 12 months.



**Figure 5: Lifecycle of *Strongylus vulgaris*. Oral uptake of L3 with grass, exsheathment in small intestine. Penetration into wall of large intestine and moulting to L4, migration on or in intima of arteries of large intestine, migration to cranial mesenteric artery and moulting to pre-adult stage, migration to intestine and penetration of intestinal wall to enter lumen where development to adults is completed. Source: ESCCAP**

## Diagnosis

Based on the detection of eggs in faeces but similar to small strongyles this means that only adult or patent infections can be diagnosed while it is the larval or migrating parasite that is most responsible for disease.

On a positive note, there is no known resistance of large strongyles to commonly used anthelmintic and, therefore, infestation levels which are sufficient to cause disease are now rarely seen.

## Treatment

Treatment plans are largely aimed at reducing pasture contamination. Historically treatment at regular intervals was used and often with rotational use of anthelmintics. However, both of these mechanisms have been found to increase resistance of small strongyles and ascarids. Nowadays selective programmes are preferred, which are based on an individual's egg-shedding capacity.

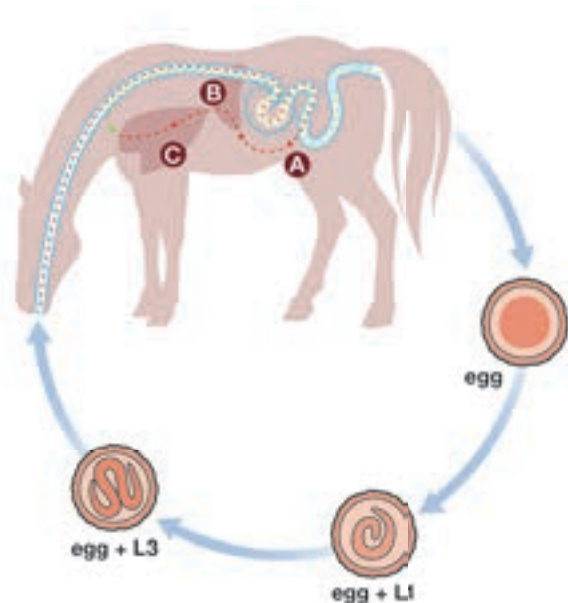
## ASCARIDS

Also known as roundworms or a common lay term is 'spaghetti' worms (Figure 6). The infective stage is the third stage larvae within the egg, which can survive in the environment for several years and is unaffected by weather conditions. Unlike strongyles contact with vegetation is not required and infections can be acquired within stables. This fact combined with the very resistant nature of the eggs means that large numbers of infective eggs can build up in the environment.



**Figure 6: Adult ascarids. Their long white bodies mean they are often referred to as 'spaghetti' worms.**

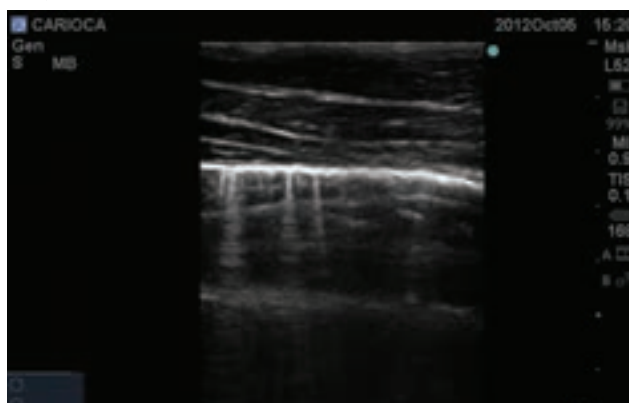
Following ingestion of eggs, release of L3 occurs in the stomach and small intestine with subsequent penetration of intestinal veins (Figure 7).



**Figure 7: Lifecycle of *Parascaris equorum*. Source: ESCCAP.**

- A:** Hatching of third stage larvae (L3) in the stomach and small intestine with penetration of the intestinal veins.
- B:** Larvae reach liver via portal vein, migration through liver tissue and penetration of liver veins.
- C:** Larvae reach lung via vena cava and right heart, penetration into lung alveoli and migration via trachea and pharynx to small intestine (Moulting to L4 and St% prior to development to adults).

Somatic migration through the liver, heart and lungs then occurs. Larvae are then transferred to the respiratory system where they are transported by mucosal flow to the larynx and are swallowed and return to small intestine. This process takes three weeks and another seven weeks of maturation is required before the first shedding of eggs. Adult females can shed hundreds of thousands of eggs but egg shedding is intermittent making diagnosis through faecal egg counts difficult. If eggs are detected in the faeces of any animal in a group, then the whole group should be treated. Infestations are frequently sub-clinical. Clinical signs during migration are often related to lung lesions and include coughing and nasal discharge (Figure 8-9).



**Figure 8: Typical comet tail lesions seen in young foals with ascarid migration. Coughing is also frequently present.**



**Figure 9:** Nasal discharge from a young foal with ascarid migration. Nasal discharge is not as consistent in these cases as coughing. Diagnosis is retrospective based on response to therapy.

Bearing in mind that the time from ingestion to completion of migration is as short as three weeks and that infection can be acquired in the stall, it is not uncommon for respiratory signs associated with ascarid migration to be seen in very young foals. Heavy infections can lead to coughing, decreased weight gain and predispose to secondary infections. Ascarid impaction and intestinal rupture can also occur (Figure 10). Larval stages cannot be diagnosed definitively and therefore disease based on larval migration is largely diagnosed based on clinical signs.



**Figure 10:** Ascarid impaction of the small intestine. Large ascarids can clearly be seen through the intestinal wall.

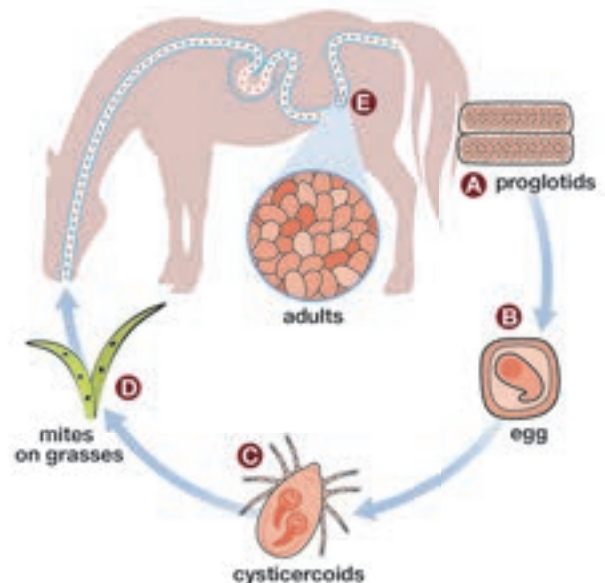
## Treatment

There are extensive reports of resistance to macrocyclic lactones and pyrantel. Benzimidazoles (panacur) and piperazine can be used.

Immunity is generally acquired at approximately 18 months but there is increasing evidence of infestation in older animals.

## TAPEWORMS

Two species of equine tapeworm are of significance in Europe: *Anoplocephala perfoliata* and *Anoplocephala magna* although the latter is predominantly recognised in Spain. Infection occurs mainly during the second half of the grazing season and essentially only on pasture by ingesting infected intermediate hosts (oribatid mites) [Figure 11].



**Figure 11:** Lifecycle of *Anoplocephala perfoliata*. Source: ESCCAP.

- A:** Gravid proglotids filled with eggs are expelled with the faeces.
- B:** Eggs are released and taken up by oribatid mites as intermediate hosts
- C:** The infective cysticercoids develop within the mites.
- D:** Oral uptake of the infected mite with grass.
- E:** The cysticercoids are released during digestion of the mite; larvae attach to intestinal mucosa and develop into adults.

The prepatent period is six weeks to four months. *A. perfoliata* adults inhabit caecum near ileocaecal junction and *A. magna* adults inhabit the small intestine (Figure 12). Higher infections can cause signs of colic associated with bowel irritation, ileal impactions, intussusceptions and intestinal obstruction. Higher prevalence of disease is seen in young horses that are less than two years of age.

## Diagnosis

Diagnosis is difficult as egg shedding is intermittent and there is no correlation between faecal egg counts and the number of parasites present. To compensate for the limited sensitivity of coproscopic diagnosis group sampling can

be considered and all animals in the group treated if eggs are identified. Commercial diagnostic tests (ELISA) which detect antibodies in serum or saliva are available but can generate false positives due to persistence of antibodies for up to four months.

Cestocidal drugs are required for treatment and the drug of choice is praziquantel. Often only available combined with ML. Cestocidal drugs appear to have remained completely effective but true evaluation of efficacy is difficult given the limitations of currently available diagnostic tests.

Treatment plans for tapeworms need to take into climatic conditions and the presence of the intermediate host but generally a single annual treatment in the autumn is sufficient though in cases of high infection pressure an earlier treatment in the summer may be required.

Pasture removal of faeces is also highly efficient.



Figure 12: *A. perfoliata* at the ileocecal junction.

## CONTROL MECHANISMS FOR FREE LIVING/ ENVIRONMENTAL STAGES

The control of parasitic infections in horses is currently reliant on the use of anthelmintics to eliminate intestinal worm burdens and therefore reduce egg contamination of the environment. However, the use of anthelmintics alone is becoming unsustainable due to emerging resistance in several parasitic species. Pasture and stable hygiene are important components that should not be overlooked.

- Strongyle eggs following shedding in faeces take at least one week to develop into infective stages and Ascarid eggs take at least two weeks. Therefore, regular and frequent cleaning of stables and pastures where practical can dramatically reduce the numbers of infective parasites. Pasture cleaning should be especially be considered in small establishments where perhaps a number of horses share a small turnout area.
- Using horse manure as a fertiliser dramatically increases the risk of *Parascaris spp* infection.
- Humidity favours survival of free living forms. Therefore keep stables dry is an important element in control.

- Agricultural practices such as follow-on grazing with other species such as sheep will eliminate large numbers of infective free living forms.
- To prevent importation of new parasite species or resistant parasite populations, any new horse should be stabled and treated upon arrival. Egg release is dramatically increased in dying populations of adults and the number of eggs shed in faeces can dramatically increase for three to five days post treatment. Horses should remain stabled for this time before being turned out on pasture.

## GENERAL TREATMENT PLANS

Treatments plans can be selective or strategic.

Selective treatment approaches can be used only for adult horses and are exclusively for the control of strongyles. The majority of horses develop an immunological response that results in suppression of small strongyle egg production.

Studies have shown that in adults there is a consistency in egg shedding patterns and adult horses can be classified as high, low or medium shedders.

This approach involves an initial year where FEC would be performed at least four times and animals classified according to their shedding tendency. Frequency of treatments for small strongyles are then based on the shedding category. Decreasing treatment frequency is thought to be an important element in reducing the development of anthelmintic resistance.

Strategic treatment approaches use a combination of the horses age, time of year and climatic factors to develop an age-related plan. One disadvantage to this approach is that a certain proportion of horses will be dewormed even though they may actually, at that time, have few, if any, worms present. An example of a strategic plan for mares is as follows:

Table 1: Strategic plan for mares.

Month	Treatment	Targeting/comments
January	Moxidectin	Large and small strongyles
The day of foaling	Ivermectin	Strongyloides These treatments can be combined if the January treatment is within six weeks of foaling date.
April	Fecal sample from all. Treat those with >500EPG with Ivermectin	Adult large and small strongyles If mares have foaled or are expected to foal within 14 days of the scheduled April treatment, then only treat with the Ivermectin at foaling.
July	Treatment with Ivermectin of those mares exceeding 500 strongyle EPG at the April sample.	
September	Moxidectin and praziquantel	Large and small strongyles, tapeworms, bots.

The above strategic plan has been developed by the author for specific farms based on identified clinical disease and faecal egg counts. It is important to remember that these are guidelines only and plans should be modified based on each farm's needs and any additional control mechanisms in place as discussed earlier in this article.