ESCCAP
EUROPEAN SCIENTIFIC COUNSEL COMPANION ANIMAL PARASITES®

8 **A guide to the treatment and control of equine gastrointestinal parasite infections**

ESCCAP Guideline 08 Second Edition – March 2019

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1. Background

The European Scientific Counsel for Companion Animal Parasites (ESCCAP) was founded in the UK in 2005 and has since then seen the creation of 11 affiliated ESCCAP national associations representing 16 European countries. The key objective of ESCCAP is to provide veterinary professionals with practical, independent and research-based advice on how best to protect companion animals from parasitic infection and disease, at the same time providing guidance on how to limit the potential for zoonotic parasitic infections. To this end, several specific guidelines addressing ecto- and endoparasitic infections in dogs and cats have already been published. This is the first equine guideline on this subject and it follows the format of previous ESCCAP guidelines.

2. Introduction

As grazing animals, horses can be infected by a broad range of gastrointestinal parasites. It must be accepted that every horse with access to pasture will be repeatedly exposed to infection with several species of gastrointestinal parasites during its lifetime. This may also apply to horses which are always or mostly kept indoors or in non-grass enclosures; these animals may be exposed to infection with gastrointestinal worms such as roundworms or pinworms. Consequently, the prevention, treatment and control of parasitic infections in horses is an ongoing task for equine veterinarians, horse farm managers and horse owners.

Thanks to the ready availability and frequent use of effective and well-tolerated antiparasitic drugs for most of the major gastrointestinal parasites, cases of clinical disease have now become much less prevalent. However, since no parasite species has been eradicated and no protective vaccine is available for any equine parasite species, routine control and surveillance are required to maintain equine health.

It is beyond the scope of this guideline to cover all equine gastrointestinal parasites, therefore those most prevalent in Europe and only those with the highest clinical relevance will be discussed. These are listed in Table 1.

The aim of this guideline is to provide equine practitioners with **concise information** and **practical advice** concerning the most important gastrointestinal parasites in horses. An **updated overview of these parasites** under prevailing epidemiological conditions in Europe is provided. The focus of this guideline is to offer recommendations which will greatly assist in the prevention or minimisation of parasite infections and thus avoid clinical disease in horses. This includes **diagnostic and preventive management measures** (i.e. **prophylactic and metaphylactic measures**) in the context of the specific needs of the different horse age groups, forms of husbandry and types of horse use.

Table 1: List of selected equine endoparasite species, their localisation and the drug classes of which effective compounds are registered for treatment in European countries.

Parasite species	Localisation	Morphological characters	Available[1] (selection)
Anoplocephala perfoliata and others	Small intestine/caecum	4–8 cm long, flat, segmented	PZQ[ISO], (PYR[PY], only partially effective at 2–3 x increased dosage)
Cyathostomins (small strongyles)	Large intestine	0.5–2 cm long, thin, small buccal capsule	IVM[ML], MOX[ML], FBZ[BZ], PYR[PY], PIP[VO]
	Mucosal/encysted stages		MOX[ML], (FBZ[BZ])
Dictyocaulus arnfieldi	Lung	2.5–8.5 cm long, round	IVM[ML], MOX[ML], FBZ[BZ]
Fasciola hepatica	Liver	Up to 5 x 1 cm, flat, leaf-like	None registered (reclassification of TCBZ[BZ])
Gasterophilus spp., bot fly larvae	Mouth, oesophagus, stomach, intestines	L3 1.5–2 cm long, barrel-shaped, two mouth hooks	IVM[ML], MOX[ML]
Habronema spp., *Draschia megastoma*	Stomach	1.0–2.5 cm, thin, hair-like	IVM[ML], MOX[ML]
Oxyuris equi (pinworms)	Large intestine/rectum	♀ 4–15 cm and with tapering tail, ♂ 0.9–1.2 cm	IVM[ML], MOX[ML], FBZ[BZ], PYR[PY]
Parascaris equorum, *P. univalens* (roundworms)	Small intestine	♀ 16–50 cm, ♂ 15–28 cm, round, mouth opening with three lips	IVM[ML], MOX[ML], FBZ[BZ], PYR[PY], PIP[VO]
	Lung stages		IVM[ML]
Strongyloides westeri	Small intestine	0.8 cm, very thin	IVM[ML], MOX[ML], FBZ[BZ]
Trichostrongylus axei	Stomach	0.4 cm, as fine as hair	IVM[ML], MOX[ML]
Strongylus vulgaris, *Strongylus equinus*, *Strongylus edentatus* (large strongyles)	Large intestine	1–5 cm long, thin, large buccal capsule	IVM[ML], MOX[ML], FBZ[BZ], PYR[PY], PIP[VO]
	Migrating/somatic stages		IVM[ML], MOX[ML], (FBZ[BZ] only partial efficacy against *S. vulgaris* and *S. edentatus*)

[1] Drugs and drug classes: benzimidazoles (BZ), fenbendazole (FBZ), isoquinoline (ISO), ivermectin (IVM), macrocyclic lactones (ML), moxidectin (MOX, cave: only use moxidectin in horses >4 months old), piperazine (PIP), pyrimidines (PY), pyrantel (PYR), triclabendazole (TCBZ), praziquantel (PZQ) and various other (VO). Those marked in red indicate when cases of anthelmintic resistance have been published for the respective drug class and parasite species in Europe.

3. General factors: age, husbandry, use, weather and climate

For effective and sustainable parasite control in horses, it is important to apply all the available knowledge on preventive management measures designed and adapted according to the specific needs of the type of horse and the conditions under which the animal is being kept.

Some parasite infections, like roundworms, lead to a partially protective immune response and older horses do not normally require intensive metaphylactic treatment or specific husbandry measures to protect them from disease. In situations where horses have no access to pasture, they are not usually exposed to strongyle infections.

The parasites addressed in this guideline essentially occur in all European countries and thus under varying existing climatic conditions. The effect of climate and weather on parasite bionomics and the epidemiology of some parasitic diseases such as strongylosis, caused by heavy infections with the small and large strongyles, should also be considered during the assessment of control measures required.

4. Specific information and recommendations for control measures against selected equine gastrointestinal parasites (key biological factors, life cycle, epidemiology/prevalence, clinical signs, diagnosis, drug treatment/resistance)

4.a. Non-migratory strongylids (commonly named "small strongyles")

These include cyathostomins and non-migratory strongyline species (*Triodontophorus, Craterostomum* and *Oesophagodontus*). Infections with "small strongyles" occur in all European countries and practically all horse farms. Horses mainly become infected on pasture through the uptake of infective third stage larvae (L3), which subsequently undergo larval development in the intestinal mucosa before they re-enter the intestinal lumen (Figure 1 and Figure 2).

Figure 1: Small strongyle/cyathostomin life cycle

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

Moulting to L4; histotropic phase of 1–2 months (in winter hypobiosis of L3 possible)
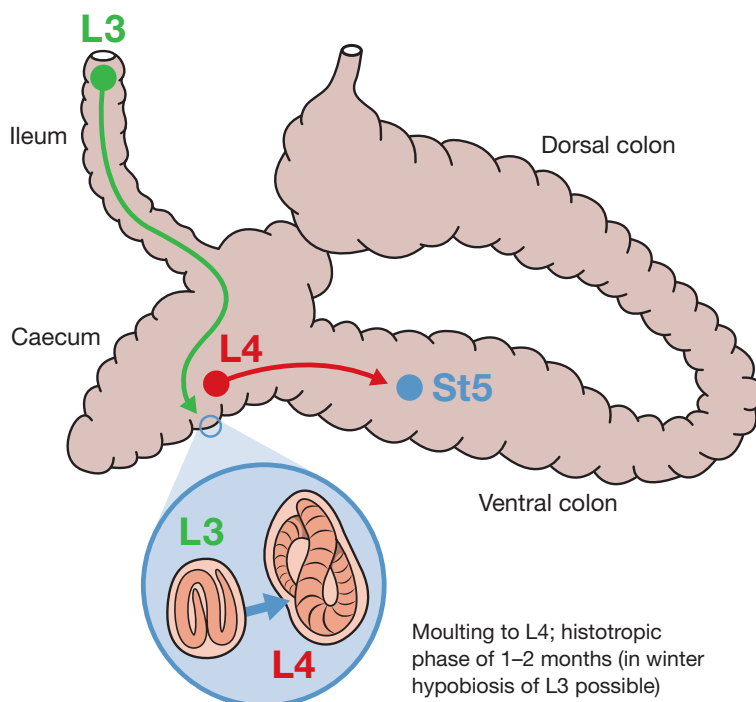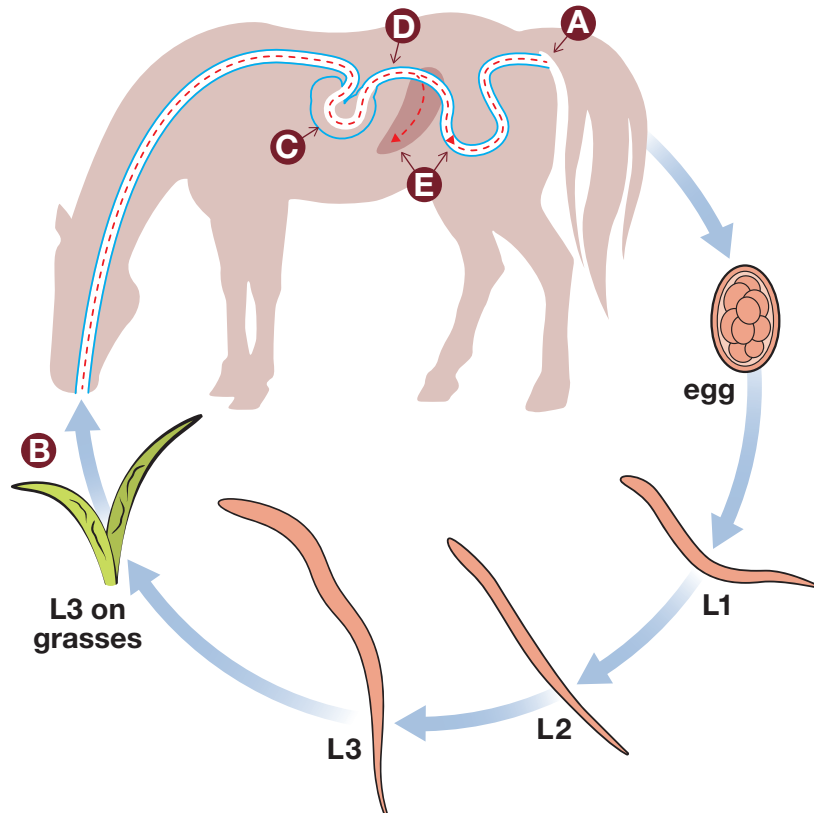
Figure 2: Development of cyathostomins in the intestine

Modified after Deplazes *et al*., 2016, Parasitology in Veterinary Medicine, Wageningen Academic Publishers, pp 268

Infections acquired indoors are considered to be rare and of minor importance. Non-migratory strongylids are considered much less pathogenic than the migratory strongylids like *Strongylus* spp., however a large number of *Triodontophorus* spp. (the most common being *T. serratus* and *T. brevicauda*) may damage the intestinal mucosa and result in emaciation and diarrhoea because of their tendency to feed all together like "worm herds". More than 40 cyathostomin species are recognised in horses and individual horses may be simultaneously infected with several, often more than ten, cyathostomin species. Cyathostomins can cause larval cyathostominosis, a syndrome which is a result of the synchronic restart of the development of numerous inhibited/encysted L3 (Figure 3) and simultaneous migration of mucosa dwelling larval stages into the lumen with massive tissue destruction.
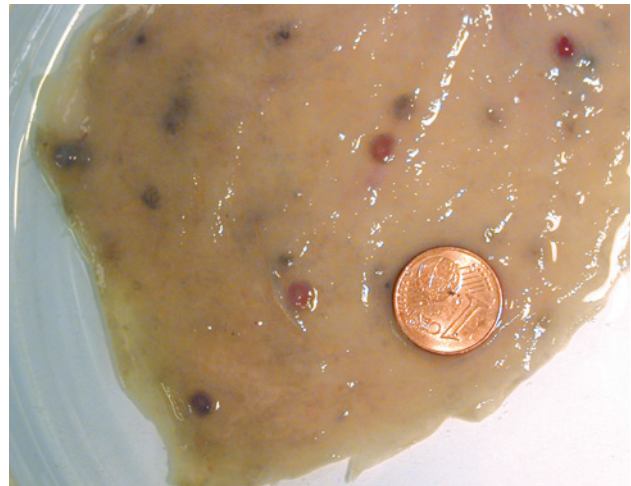
Figure 3: Large intestine of a horse with multiple encysted small strongyle larvae in the mucosa

This disease is mostly seen in animals up to six years of age and results in acute and persistent diarrhoea (sometimes accompanied with colic, weight loss or fever) and in a considerable number of cases a fatal outcome. Normally, lumen-dwelling larval and adult cyathostomins are considered to be of low pathogenicity and most infected animals do not appear to be clinically affected, even if fairly high worm burdens are present. Nevertheless, some studies have suggested a possible correlation between cyathostomin infection and recurrent diarrhoea and intermittent colic.

Figure 4: Gastrointestinal strongyle eggs in horse faeces, egg marked with L1 contains first stage larva

Diagnosis of a patent small strongyle infection is performed by faecal examination and identification of the thin-shelled, ovoid, strongyle-type eggs which measure approximately 80–100 µm in length (Figure 4).

Often larval/pre-adult and adult stages are found in large numbers on the faeces of treated horses (Figure 5).

Various methods can be employed allowing either the qualitative or quantitative analysis of strongyle eggs. There are no scientific data available concerning the correlation between the number of strongyle eggs per gram of faeces and the intestinal adult worm burden in adult horses. One study, in which horses younger than three years were examined, showed that low or even negative faecal egg counts can be found in horses with thousands of intestinal worms.

Figure 5: Horse faeces with typically red-coloured cyathostomin stages

Overall, it may be assumed that the correlation between faecal strongyle egg counts and worm burden is weak in all age groups. It is noteworthy that the eggs of small and large strongyles (e.g. *Strongylus vulgaris*) are not reliably distinguishable based on morphological criteria. However, following *in vitro* culture, the third stage larvae (L3) can be differentiated based on the number of their midgut/gut cells. This differentiation is significant due to the considerably higher pathogenicity of large strongyles, which, owing to the widespread use of effective anthelmintics, are now only considered to occur on a low percentage of farms. However, recent data show that *S. vulgaris* is still present in the European horse population (see 4.b.).

Horses first become infected with small strongyles as soon as they start grazing and start to shed strongyle eggs 6–14 weeks after infection. Accordingly, treatment and control measures should be applied to foals beginning at approximately two months of age. As a result of the widespread occurrence of anthelmintic resistance (AR), it is important to reduce the frequency of treatment to the minimum possible without risking the establishment of clinically relevant worm burdens. Under currently prevailing epidemiological conditions in most European countries, where the intensity of small strongyle infection is only low to moderate, effective three-monthly treatment of foals and yearlings can be considered appropriate. In adult horses it may be feasible to treat only twice yearly. In the absence of large (migrating) strongyles one annual treatment is sufficient, when faecal monitoring does not indicate further treatment and provided that strict quarantine procedures are being employed on the respective farm.

Horses suffering from larval cyathostominosis should be treated palliatively i.e. reducing diarrhoea (for example using codeine phosphate), reducing mucosal inflammation and administering fluid therapy if needed. Irrespective of clinical status, all horses of the same group should receive anthelmintic treatment against the mucosal worm burden either using moxidectin (once orally 0.4 mg/kg bodyweight only in horses >4 months old) or fenbendazole (7.5 mg/kg bodyweight orally once daily over five days and only when the respective cyathostomin population is susceptible). It is recommended that such treatments against mucosal cyathostomin larvae are employed once a year for foals and young horses up to and including four years of age (e.g. at the end of the grazing season).

With regard to AR, recent studies in France, Germany, Italy and the UK, showed that the small strongyle populations present on more than 80% of the farms studied had reduced susceptibility to the benzimidazole group of anthelmintics (BZs). In the case of pyrantel, this was only found on approximately 20–30% of farms. In contrast, the macrocyclic lactones (MLs) ivermectin and moxidectin were found to be fully effective with 95–100% faecal egg count reduction at 14 days post treatment on nearly all of the farms tested. Nevertheless, a reduced egg reappearance period (ERP) post ML treatment has been reported occasionally and this is considered to be a sign of reduced efficacy. It is therefore advisable to regularly confirm/test the efficacy of any anthelmintic drug class used by, for example, conducting an annual faecal egg count reduction test (FECRT).

4.b. Migratory strongylids (commonly named "large strongyles")

This group of parasitic worms occurring in the large intestine consists of migratory species of some strongylines (*S. vulgaris, S. edentatus* and *S. equinus*, Figure 6).

Strongylus edentatus   *Strongylus equinus*   *Strongylus vulgaris*
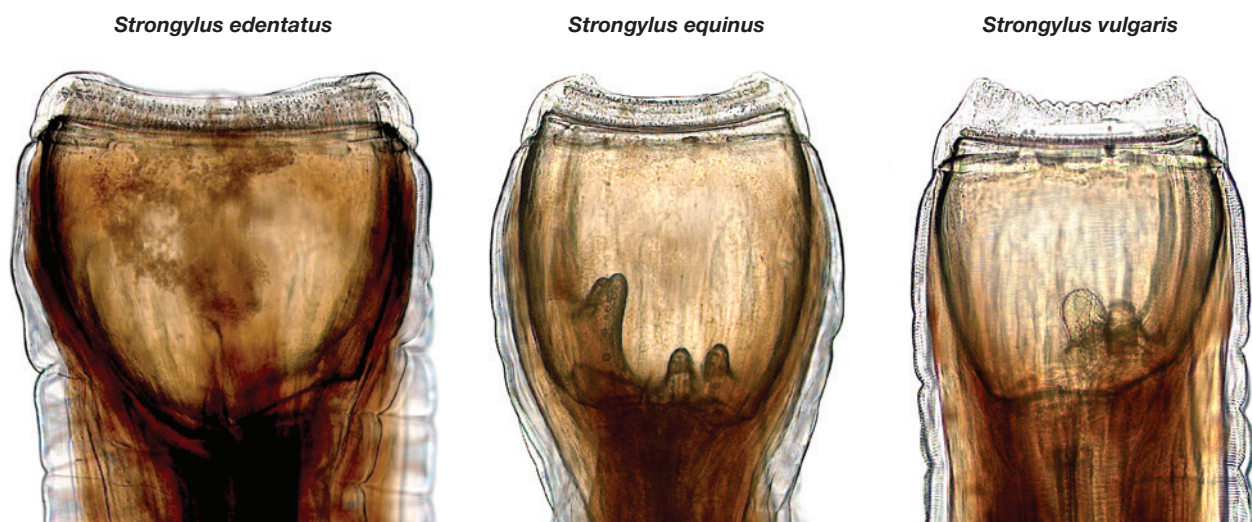
Figure 6: Anterior end of large strongyles depicting the buccal capsule, leaf crown and tooth-like structures at base of the buccal capsule

Clinically, these are the most important of the equine parasites with *S. vulgaris* considered a major threat to equine health. Their larvae migrate extensively before developing to maturity in the large intestine: in the anterior mesenteric and nearby arteries (*S. vulgaris*, Figure 7 and Figure 8), through the liver to the subperitoneal connective tissues (*S. edentatus*) and to the liver and the pancreatic and renal region (*S. equinus*). These larval migrations result in long prepatent periods, which are 6–7 months for *S. vulgaris*, 9 months for *S. equinus* and 11–12 months for *S. edentatus*. The damage caused by the migrating larvae leads to severe pathological consequences and clinical signs which differ depending on the respective *Strongylus* species.

Figure 7: *Strongylus vulgaris* life cycle

Parasitic phase: 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.

Free-living phase: Thin-shelled eggs expelled with faeces, development to first stage larva (L1) within the egg, moulting to second stage larva (L2) and infective third stage larva (L3).
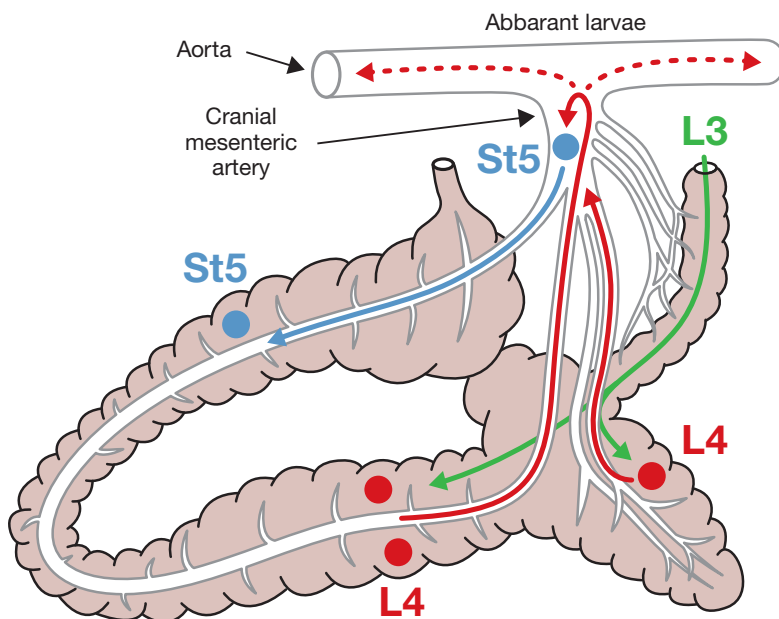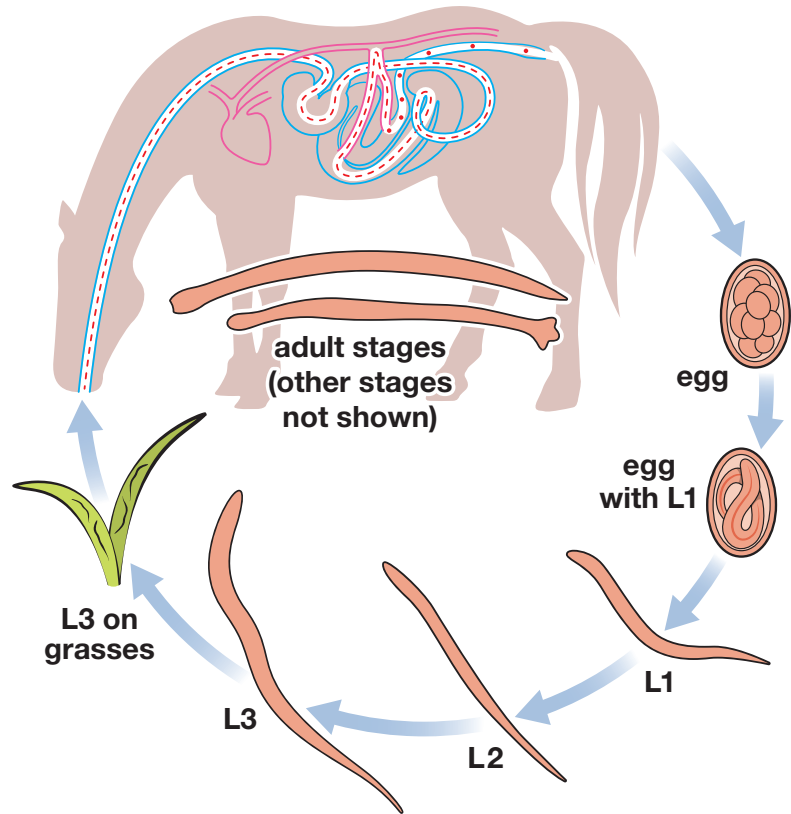
Figure 8: Development and migration of *Strongylus vulgaris* larvae

Development: L3 penetrate the intestinal wall and moult to L4, migration of L4 into the cranial mesenteric artery, moulting to St5 from 90th day p.i., backwards migration from arteries to the gut.

Modified after Deplazes *et al*., 2016, Parasitology in Veterinary Medicine, Wageningen Academic Publishers, pp 269

In the past, *S. vulgaris*, the "horse killer", has received most attention due to the clinical syndrome of thromboembolic colic caused by larvae migrating to the cranial mesenteric artery (Figure 9). Adult strongyles feed on plugs of intestinal mucosa, the resulting damage causing diarrhoea, weakness, emaciation and sometimes anaemia.

The migrating larvae and the thromboses they cause can lead to non-strangulating intestinal infarctions most often seen in the large intestine. Depending on the infection intensity, initial clinical signs of non-strangulating intestinal infarctions may be mild, often recurrent abdominal pain (colic), fever and peritonitis. If the infarcted intestine is not recognised and surgically resected, the intestine will necrotise and rupture leading to the death of the horse.
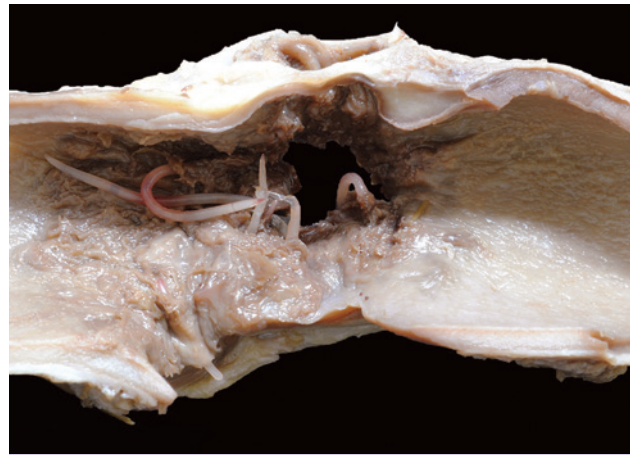
Figure 9: Aorta at cranial mesenteric artery junction showing several L4/pre-adult stages of *Strongylus vulgaris*

It is noteworthy that sometimes even horses with severe intestinal necrosis caused by thrombosis, do not show signs of serious pain. Peritonitis is therefore often the only sign advocating surgical intervention.

Detection of patent large strongyle infections is based on *in vitro* culture of the third stage larvae (L3) which can be differentiated from those of other strongyles based on the number of midgut cells (see also 8.1. Diagnosis of worm infections).

Previously, routine treatments at regular intervals have been recommended for all horses to minimise the level of pasture contamination and thus reduce the risks associated with migrating *S. vulgaris* larvae. Due to years of this intensive metaphylactic chemotherapy, infection with *S. vulgaris* has become uncommon. However, during the recent past, a selective therapy approach has been increasingly recommended in an attempt to reduce the development of anthelmintic resistance in the cyathostomins by reducing treatment intensity i.e. to leave horses with low strongyle egg counts untreated. Consequently, specific diagnosis of patent *S. vulgaris* infections is important.

For sustainable control of strongyle infections in horses, metaphylactic therapy programmes should therefore be designed to avoid anthelmintic resistance (e.g. in cyathostomins and ascarids) and to simultaneously minimise the potential for *S. vulgaris* transmission. To date there have been no convincing reports of anthelmintic resistance in large strongyles. Biannual treatment of all horses with a drug effective against *S. vulgaris* larvae (e.g. IVM or MOX) is likely to provide adequate control of this parasite.

4.c. Roundworms (*Parascaris equorum* and *Parascaris univalens*)

The equine roundworm species, *Parascaris equorum* and *P. univalens*, cannot be distinguished morphologically. Recent findings indicate that *P. univalens*, and not *P. equorum*, is the species currently prevalent on most, if not all, horse farms in Europe where equine roundworms are found. At present, there are no molecular tools available for species differentiation and since both appear to have a similar pathogenesis and biology, we will subsequently refer simply to *Parascaris* spp.

Infection with equine roundworms is mainly prevalent on stud farms and predominantly found in foals and young horses. Recent cross-sectional studies in Europe provided prevalence rates of between 20% to over 80% in foals.

Measuring up to 50 cm in length at the adult stage, these worms which reside in the small intestine represent one of the largest known parasitic nematode species. The females can shed hundreds of thousands of eggs per day, thus contributing to considerable environmental contamination. The infective stage is the third stage larva (L3) within the egg, which can survive in the environment for several years, even under harsh conditions such as prolonged periods of frost. Consequently, both stables and pastures once contaminated will remain a constant source of infection. Following ingestion of eggs, larvae are released and penetrate the small intestinal wall to begin a somatic migration via the bloodstream through the liver, heart and lungs. There, the larvae transfer to the respiratory system where they are transported with the mucosal flow to the larynx and, after being swallowed, reach the small intestine approximately three weeks post infection. It then takes at least another 7 weeks of maturation before the first shedding of eggs in the faeces (prepatent period 10–16 weeks, Figure 10).
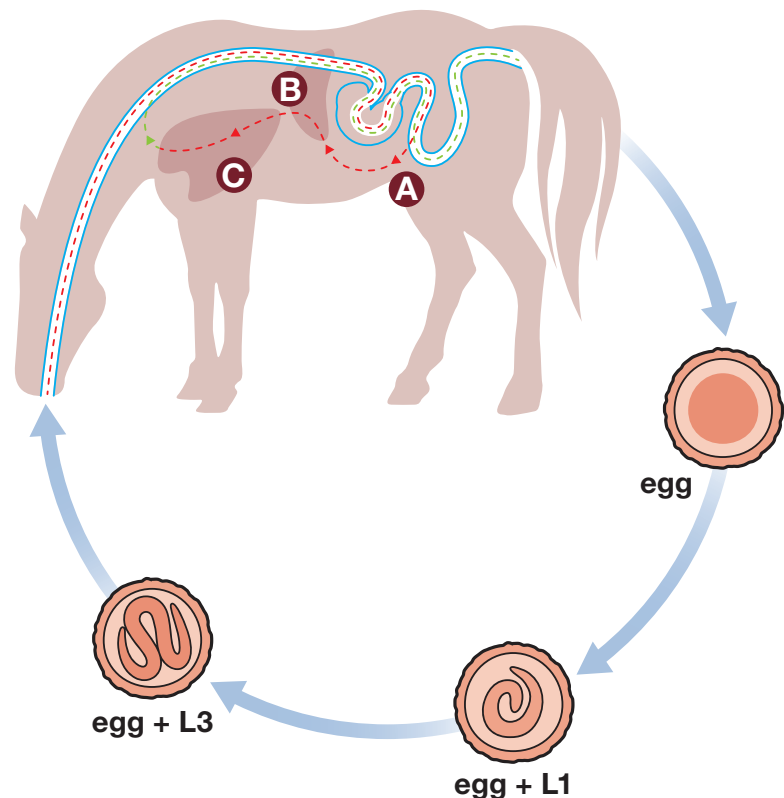
Figure 10: *Parascaris equorum/ Parascaris univalens* life cycle

A: Hatching of third stage larvae (L3) in the stomach and small intestine, penetration of 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 alveoles and migraton via trachea and pharynx to small intestine (moulting to L4 and St5 prior to development into adults).

Often, no clinical signs are observed. Sometimes during the somatic migration, clinical signs occur mostly associated with pathological changes in the lungs, whereas the migration through the liver does not appear to cause clinical signs. In the lungs, changes include haemorrhagic mucosal lesions and heavy infections can result in coughing and decreased weight gain in young stock and can also lead to secondary bacterial or viral infections. During the intestinal phase (Figure 11), *Parascaris* spp. infected animals show reduced appetite and a rough coat; intermittent colic and wasting may also occur. Occasionally, heavy infections can result in severe colic, obstruction of the small intestine, perforation, invagination followed by peritonitis. Under current epidemiological conditions in most western European countries, the infection intensity is low and the vast majority of cases in foals and young horses are subclinical. Adult mares can occasionally excrete eggs and so act as a source of infection for subsequent generations.
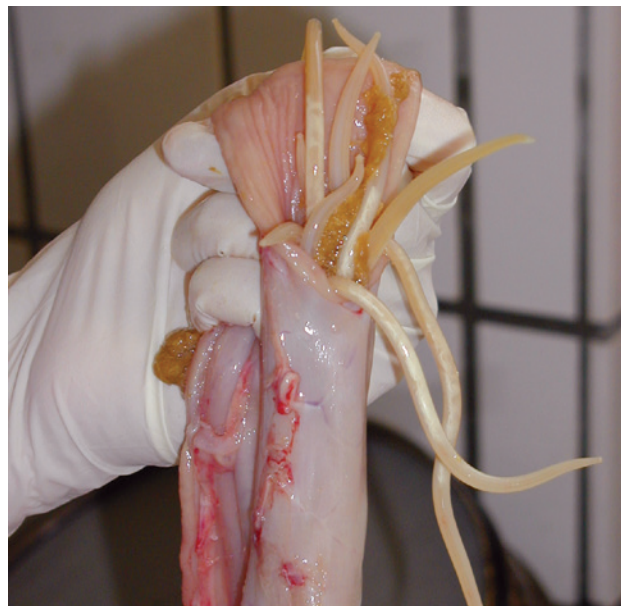
Figure 11: Small intestinal infection with *Parascaris* spp.

Diagnosis of *Parascaris* spp. infections relies on the direct detection of eggs (round, brownish, approximately 100 µm in length, thick-shelled) by faecal flotation and/or of pre-adult stages or adult worms in the faeces. Coproscopic analysis is based upon the microscopic detection of the eggs either during a qualitative or quantitative flotation protocol. As with ascarid infections in other host animals, it is impossible to reliably relate the intensity of the intestinal worm burden to the level of egg shedding in the faeces and a positive faecal examination should always be considered an indication for anthelmintic treatment. Due to the environmental contamination and long survival times of *Parascaris* eggs, it has to be assumed that horses from the same age group, sharing the same environment, which are currently not shedding eggs in the faeces, are also exposed and probably infected and that the infection may be in the prepatent phase. All horses from the same age group should be treated based upon a positive coproscopic analysis of any individual within the group. The MLs are effective against larval stages in the lungs and intestines. Thus, the previous recommendation for 6–8 weekly treatments during the first year of the animal's life is aimed at the prevention of contamination and the consequent development of intestinal worm burdens. However, highly frequent treatment is considered to be the main reason for the selection of ML-resistant *Parascaris* spp. populations.

Sustainable control approaches should include regular faecal monitoring (preferably individual samples). Stable and pasture hygiene must accompany anthelmintic treatment, which should begin at two months of age and repeated every three months during the first year of life, employing different drug classes. The above-mentioned AR situation requires that each farm assesses the efficacy of the drug classes used, most importantly the MLs, by performing a faecal egg count reduction test (FECRT) or at least a faecal examination for *Parascaris* eggs, 14 days post treatment. The beneficial effects of pasture "cleaning" and the chemical or physical disinfection of stables have been demonstrated in field surveys, and these have been associated with significantly reduced *Parascaris* spp. prevalence. When using disinfectants it is important to use only those which have been shown to be effective against worm eggs (i.e. containing cresol or peracetic acid, see also chapter 5). Resistance to MLs has been widely reported for *Parascaris* spp. and more recently there have been a few reports from North America and Australia suggesting that resistance to pyrantel and the BZs may be emerging. On farms where there is confirmed resistance to the MLs, then BZs, pyrantel or piperazine citrate (available in some EU countries only) can be used. However, the latter has to be given in comparatively high doses and large volumes often requiring nasogastric intubation. Due to a potential risk of colic caused by worm convolutes following the immediate killing/paralysis of neurotoxic drugs, MLs, pyrantel and piperazine should not be used on foals with heavy infections.

4.d. Tapeworms (*Anoplocephala perfoliata*, *Anoplocephala magna* and *Paranoplocephala mamillana*)

Two species of equine tapeworms are of significance in Europe: *Anoplocephala perfoliata* and *A. magna*. Most cases of tapeworm infections in horses are caused by *A. perfoliata* which is endemic in many European countries. *Anoplocephala magna* infections are rarely recognised, however there is evidence that this is prevalent in Spain. *Paranoplocephala mamillana* has also been found occasionally, for example in Germany.

Infection with tapeworms occurs mainly during the second half of the grazing season and essentially only on pasture by ingesting infected intermediate hosts which are oribatid mites or "box mites" (Figure 12). The prepatent period is from six weeks to four months. Adult *A. perfoliata* (Figure 13) are 4–8 cm in length and inhabit the caecum close to the ileocaecal junction while *A. magna* specimens (up to 80 cm in length) occupy the small intestine. Higher *A. perfoliata* infections can be associated with clinical signs of colic in the horse, due to bowel irritation, ileal impactions, intussusceptions and intestinal obstruction, which may lead to recurrent episodes of spasmodic colic. The risk of gastrointestinal problems increases in horses with chronic and heavy infections. *Anoplocephala magna* pathogenicity is limited to catarrhal inflammation and infections generally pass unnoticed, with higher prevalence in young horses under 2 years old.

Figure 12: *Anoplocephala perfoliata* life cycle

Gravid proglotids filled with eggs are expelled with the faeces (A), eggs (B) are released and taken up by box mites as intermediate hosts within which the infective cysticercoids develop (C), following oral uptake of the infected mite with grass (D), the cystercercoids are released during digestion of the mite, larvae attach to intestinal mucosa and develop into adults (E).
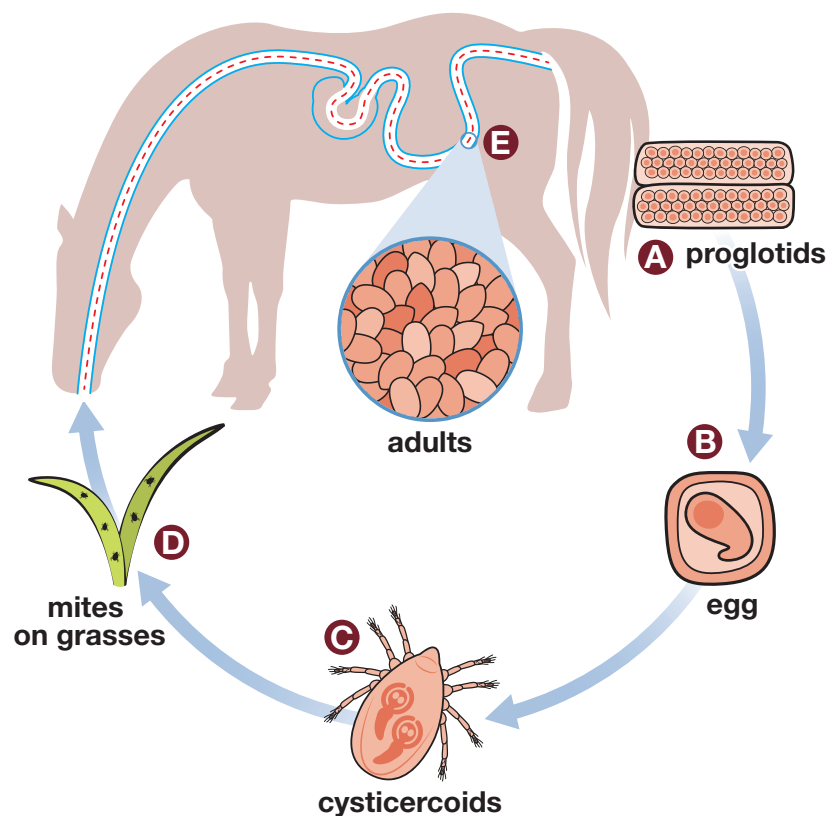
Figure 13: Head section of adult *Anoplocephala perfoliata*

Diagnosis of tapeworm infections in horses by faecal examination is limited in sensitivity as eggs are passed intermittently and this is not associated with the number of worms present. To improve the detection of *Anoplocephala* eggs in faeces, combined centrifugal sedimentation-flotation techniques have been developed which process large faecal samples (15–50 g). To compensate for the limited sensitivity of the coproscopic diagnosis, it is recommended that a group/farm diagnosis is performed and all animals treated if tapeworm eggs are found in any of the examined samples. Commercial diagnostic assays, capable of detecting *A. perfoliata* antibodies by either a serum ELISA (Diagnosteq, University of Liverpool, UK) or a saliva ELISA (EquiSal, Austin Davis Biologics, Great Addington, UK) are available. Both tests can potentially generate false-positive results in some horses due to the persistence of antibodies for up to four months e.g. in previously-infected horses that have already been treated with anthelmintics. However, if allowances are made for this, these tests can prove very useful, particularly for group/farm diagnosis using the serum test or targeting of treatments to individuals using the saliva test.

Treatment of tapeworms is based on the use of cestocidal anthelmintics and the drug of choice is praziquantel. Praziquantel is often only available in combination with MLs (e.g. ivermectin or moxidectin). In a situation where only drugs effective exclusively against nematodes are used, undiagnosed tapeworm infections can persist for several years in groups of horses. Cestocidal drugs appear to have remained fully effective, but it is difficult to evaluate the efficacy of tapeworm anthelmintics using current diagnostic methods, due to a lack of sensitivity of the available tests.

Sustainable tapeworm control strategies should be related to regional climatic conditions and management systems should be put into place to ascertain the significance of tapeworm infection at farm level. Routine multiple treatments throughout the year, although justified for the control of cyathostomins, are not recommended for the control of tapeworms, due to their different life cycle involving an intermediate host and the marked seasonality of transmission. Generally, a single annual tapeworm treatment in late autumn or winter will be sufficient to avoid significant infection, but in cases of heavy infection pressure, an additional earlier treatment during the summer may be required. Regular (i.e. at least weekly) removal of faeces from pasture may in the long term also reduce infection pressure.

4.e. Bot flies (*Gasterophilus* spp.)

Bot flies are arthropods of the genus *Gasterophilus* (Diptera: Oestridae). *Gasterophilus intestinalis*, *G. haemorrhoidalis, G. nasalis, G. inermis* and *G. pecorum* are the most prevalent species in Europe. *Gasterophilus intestinalis, G. haemorrhoidalis* and *G. nasalis* frequently infest grazing horses; *G. inermis* and *G. pecorum* are found less often. Their larvae mainly cause gastrointestinal myiasis.

Adult flies resemble honey bees and females play the major role in the infection. In southern Europe they may already be active in spring/early summer, while in temperate regions, egg laying takes place in late summer. Females of most *Gasterophilus* species fly near horses and dart very rapidly close to the skin to attach an egg to a hair (this fly activity produces a special buzzing noise which many horses find very disturbing). Females die after laying small (1–2 mm), mostly operculate and yellowish eggs. The eggs can be seen fairly easily with the naked eye, especially on animals with a dark hair coat. Regarding the egg localisation, *G. intestinalis* places eggs on the hair of the forelimbs, shoulders and flanks while most other species deposit their eggs on the head. *G. pecorum* is an exception as females lay eggs in the environment. Humans have occasionally been infected showing conspicuous tracks in the cheeks, and even infection of the digestive tract.

The hatching of first stage larvae (L1) takes place after a mechanical stimulus (*G. intestinalis* and *G. pecorum*) or spontaneously (*G. nasalis*). The L1 reach the oral cavity through oral uptake (licking or grazing for *G. intestinalis* and *G. pecorum* respectively) or by larval migration. Second stage larvae (L2) are found in the stomach and duodenum where they moult into third stage larvae (L3). The L3 measure 16–20 mm in length, have a barrel-shaped form and bear two large mouth hooks. The segments have one or two rows of spines.

After several months, the L3 finally leave the host in the faeces and pupate in the soil, before the adult flies emerge into the environment. The parasitic phase takes 8–10 months and the pupal phase 3–8 weeks. The adults mostly emerge during June/July and are usually active until October or November, although their activity may begin earlier and last longer in southern European regions.

Gasterophilus L2/L3 are found attached to the mucosa of the stomach (*G. intestinalis*), duodenum (*G. nasalis, G. haemorrhoidalis*) or rectum (*G. haemorrhoidalis, G. inermis*), where they may cause focal, superficial mucosal ulceration, cutting and piercing of tissues to facilitate feeding. When L1 are found in the oral cavity, they migrate through the mucous membranes of the tongue, gums and palate, causing gingivitis and pain which may affect food intake. Generally, the first clinical signs of gasterophilosis are characterised by difficulties in swallowing due to the localisation of the larval stages in the throat. Remarkably, massive infections with *Gasterophilus* spp. are not always associated with clinical signs and are thus considered much less pathogenic than most nematode parasites. Nevertheless, gastric and intestinal ulcerations have been associated with this infection, as well as chronic gastritis, gut obstructions, volvulus, rectal prolapses, rupture of the gastrointestinal tract, peritonitis, anaemia and diarrhoea.

The presence of *Gasterophilus* spp. can be confirmed in the summer/autumn by inspecting the horse's coat and finding yellowish eggs attached to the hair. Gastrointestinal examination through endoscopy may allow the detection of *Gasterophilus* spp. larvae attached to the stomach and duodenum. ELISA based on excretory/secretory antigens of *G. intestinalis* L2 for antibody detection and PCR techniques has been used in Europe, however these tools are not yet considered routine laboratory techniques.

The larval stages of *Gasterophilus* are highly susceptible to MLs (particularly ivermectin) and will be eliminated during regular deworming with these drugs. As fly activity ceases with the first frosts, an appropriate treatment in late autumn, e.g. early November, should remove all larvae present within horses. The removal of the eggs by hand with a special bot comb or bot knife or thoroughly washing the hair with warm water mixed with an insecticide is recommended though it is usually not sufficient to effectively prevent gastrointestinal infection.

4.f. Threadworm (*Strongyloides westeri*)

The nematode *Strongyloides westeri* resides in the small intestine, mainly the duodenum. Patent infections are largely found in young horses, i.e. foals up to six months old. Occasionally, older horses can harbour this parasite and mares are an important source of infection for their foals. It is a unique parasite, since it only develops female and no male parasitic stages. The very slender, small (maximum length 10 mm) parasitic females reproduce through parthenogenesis. They shed small, thin-shelled, embryonated eggs (40–50 x 30–40 μm) containing the first stage larvae (L1) which hatch in the environment. These can develop directly to infective third stage larvae (L3) or give rise to free-living males and females that will reproduce and in turn produce infective L3.

Infection may occur by ingestion of L3 in the mare's milk ("lactogenic infection") and this is the primary mode of transmission of *S. westeri* to foals. Later on, transmission also occurs by ingestion of infective L3 from the pasture or environment, or through percutaneous infection. When percutaneous infection occurs in immune adult horses, *S. westeri* larvae do not become established in the alimentary tract and patent infections are rare. Instead, they are distributed through various somatic tissues where they may remain viable for long periods, probably years. In mares, the hormonal shifts during pregnancy and lactation probably stimulate these larvae to resume migration to the mammary glands so that they are transmitted to the foal. After being ingested with the milk, the larvae undergo a somatic migration starting with the penetration of the small intestinal wall. They subsequently travel through the lungs, via the trachea and pharynx where they are swallowed finally reaching the small intestine. Here they mature to adult female worms. The prepatent period can take some weeks but can be as short as 5–8 days.

During massive percutaneous infection, local dermatitis can occur. The hair coat may become dull and animals can be stressed by local skin irritation and itching, which is often a consequence of an allergic response to re-infections. The major pathogenic effect of infection occurs in the intestine, where adult females embed in the small intestinal mucosa and cause local enteritis, which can lead to diarrhoea. The role of *S. westeri* as the cause of diarrhoea in young foals is unclear since there are reports of high faecal egg counts associated with severe cases of diarrhoea while high *Strongyloides* egg shedding has also been found in animals showing no clinical signs. Clinically affected foals may become anorexic and lethargic but in situations where regular worm control is employed, it appears that most *S. westeri* infections are asymptomatic. It should be noted that there are many cases of diarrhoea in foals aged 1–2 weeks which are not associated with *S. westeri* infection.

Diagnosis of *S. westeri* infection is performed by coproscopic detection of the typical eggs in faeces.

Treatment and control of *S. westeri* infections should involve both anthelmintics and basic hygiene measures. Under the current epidemiological situation, the formerly often-employed, routine treatment of foals during the first few weeks of life does not seem justified anymore due to the low prevalence and lack of evidence for *S. westeri* associated disease in foals. On farms where *S. westeri* has previously been detected, regular deworming of mares, before or shortly after parturition (i.e. 1–2 days) is thought to reduce the number of larvae in milk and lower the incidence of diarrhoea in foals. For clinical cases, a number of drugs are available including ivermectin or fenbendazole, the latter at a dosage of 50 mg/kg body weight (significantly higher than the standard dosage of 7.5 mg/kg bodyweight). Pasture and stable hygiene, together with cleaning of the mare's udder should reduce the risk of environmental contamination and foal infection.

4.g. Pinworm (*Oxyuris equi*)

The equine pinworm *Oxyuris equi* (Figures 14a, 14b and 15) has been reported as a common horse parasite in Europe. Infections arise in stables and may also occur on pasture, but usually only a few horses appear to be clinically affected. *Oxyuris equi* is rarely considered a major threat to equine health, but heavy infections may result in fatigue, decreased performance and loss of condition. Even massive invasions of fourth stage larvae usually do not lead to clinical signs, however, in single cases, they can cause severe inflammation of the colonic mucosa with non-specific intestinal signs.

Considerable numbers of *O. equi* eggs (up to tens to hundreds of thousands) are deposited by the female worms on the skin of the perianal region. The sticky fluid surrounding these eggs causes an intense itching and an indication of *O. equi* infection is persistent anal pruritus and tail-rubbing which causes excoriations and bare patches around the tail (Figures 16a and Figures 16b).

Figure 14a: *Oxyuris equi* (pinworm) adults ♂ 0.9–1.2 cm ♀ 2.5–15 cm, ♀ pointed rear end, bevelled anterior pole, operculum, u-shaped larva

Figure 14b: Anterior end of *Oxyuris equi* adult with typical hourglass-shaped oesophagus
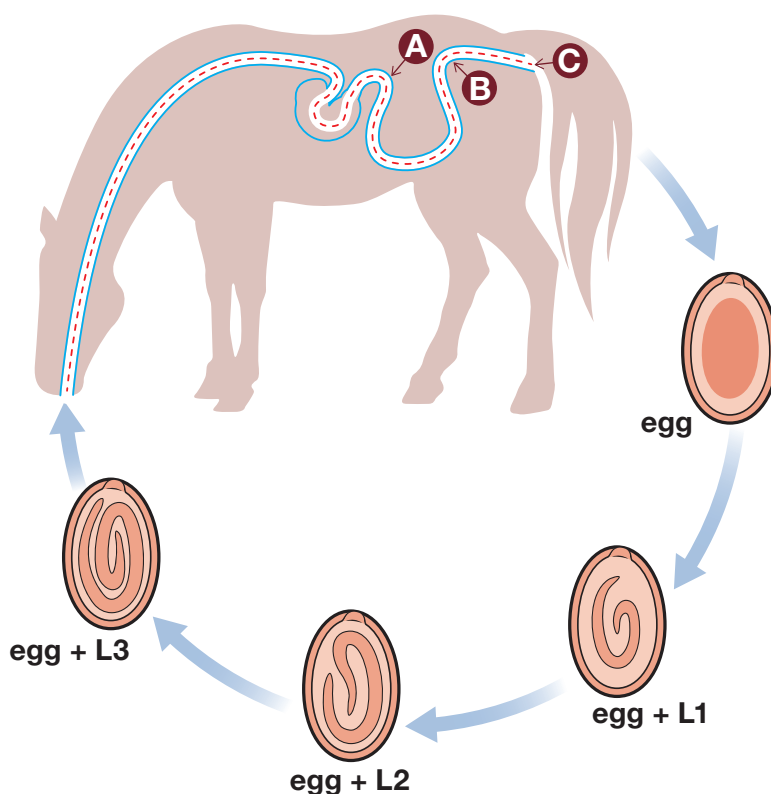
Figure 15: *Oxyuris equi* life cycle

Hatching of L3 in the small intestine (A), histotrophic phase in the caecum and colon (B), adults develop in the colon, females emerge from the anus to lay egg clusters on the perineum (C)

egg

egg + L1

egg + L2

egg + L3
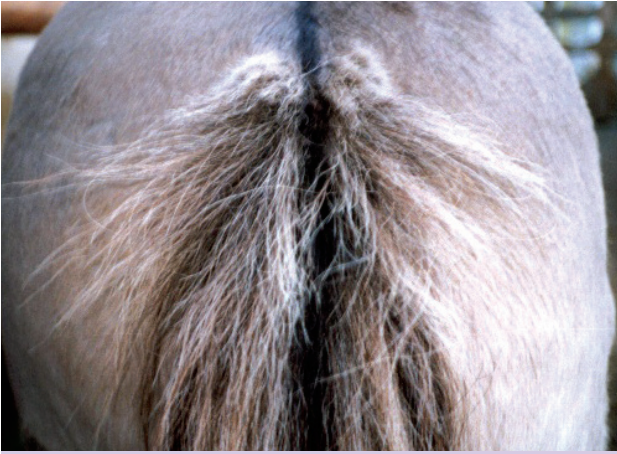
Figure 16a: *Oxyuris equi* infection with itching and dermatitis of the tail root, tail rubbing, broken/matted hair ("rat tail")

Figure 16b: Massive *Oxyuris equi* egg excretion with cream-coloured, dried egg clusters

Diagnosis of pinworm infection is made by applying a transparent adhesive tape to the skin of the perianal region, which is then removed and examined microscopically to identify characteristic oxyurid eggs, which contain an embryo and are oval and flattened on one side, with an operculum at one end.

The perianal region of infected horses should be washed with hot water containing a mild disinfectant to relieve the pruritus and to prevent the spread of pinworm eggs throughout the horse's environment.

The MLs and BZs are effective against pinworms and their larval stages. Pyrantel has variable efficacy against pinworms. Recent anecdotal reports on the reduced efficacy of MLs (ivermectin and moxidectin) against *O. equi* must be viewed as potential resistance.

5. Measures against free-living/environmental stages

The control of parasite infections in horses currently relies mainly on the use of anthelmintic treatments to eliminate intestinal worm burdens and thus reduce the contamination of the environment with eggs/infective stages. However, as explained below, this strategy alone without any other measures designed to prevent or minimise infection intensity is not sustainable, due to the development of anthelmintic resistance in several parasite species. Consequently, stable and pasture hygiene are both important components of an integrated worm control strategy and should therefore be employed. The infective stages of some equine parasites have the potential to survive in the environment for months or years and it is important to consider the following factors:

- The eggs of important nematode species need, at appropriate temperatures, at least one week (strongyles) or two weeks (*Parascaris*) to develop into infective stages. Therefore, the regular and frequent cleaning of stables and the removal of faeces from pasture will reduce the risk of heavy infections. If possible, droppings should be removed from the pasture on a daily basis. If this is impracticable, it should at least be done on a twice weekly basis. Stables should also be cleaned on a daily basis, but when this is not possible e.g. in deep litter management systems, stables should be thoroughly cleaned (mechanically and by steam) and disinfected at least once a year using a disinfectant which has been shown to be effective against ascarid eggs (e.g. as documented and listed by the committee for disinfection of the German Veterinary Society; www.desinfektion-dvg.de/index.php?id=1793).

- Using horse manure as a fertiliser will increase the risk of *Parascaris* spp. infection and thus should be avoided. However, it has been shown that by effective windrow (long row) composting, *Parascaris* eggs will be prevented from developing to the infective stage (or result in die-off), so that equine manure and soiled bedding processed accordingly can be used for pasture fertilisation without increasing the infection risk.

- All free-living stages of equine worms are vulnerable to low environmental humidity and therefore stables should be kept dry.

- To prevent importation of new parasite species and/or resistant parasite populations, each horse newly introduced to a farm should be quarantined and treated upon arrival. Subsequently, the horse should only be put out to pasture after a faecal examination conducted five days post therapy has confirmed that the horse is not shedding worm eggs and that treatment was successful.

- To date, measures aimed at the biological control of strongyle developmental stages in the environment (i.e. L1, L2 and L3) are still in an early experimental phase and although promising it seems unclear if or when these will become practical and available for routine use.

- Agricultural practices such as deep ploughing of the paddock will help to reduce not only the presence of infective nematode larval stages but also mites therefore also potentially reducing tapeworm infection, provided no new contamination occurs.

6. General treatment strategies for foals, yearlings, adults and mares (tabulated specific treatment recommendations in an annual context)

It needs to be noted that treatment-related factors, such as underdosing and frequent anthelmintic treatment, are probably the most relevant reasons for the emergence of AR. Thus, to avoid selection for AR, treatment should be administered as infrequently as possible without risking disease. This is managed by regular faecal examinations including the differentiation of small and large strongyles, so that the infection status of the individual animal or the respective age group is monitored throughout the year. Furthermore, thorough hygiene and quarantine measures both in stables and on pasture are important to reduce the infection pressure and accordingly the need for treatment.

To date, there are two alternative approaches for the control of small strongyles which are recommended by experts working in the field of equine worm control. These are the '**selective treatment**' and the '**strategic treatment**' approaches. In the following section, both approaches will be briefly described and discussed. Both strategies are considered to be effective in preventing clinical disease in adult horses when employed according to these guidelines. Their specific potential to mitigate the development of anthelmintic resistance will largely depend upon the resulting frequency of treatments per horse per year for each strategy. Comparable data are not yet available but should be generated for the future analyses of both strategies. Notwithstanding, it is essential that veterinarians and those responsible for equine health are aware of the actual resistance status of the parasites occurring on their respective farms. On farms where resistance of a certain drug class against a specific worm species has been identified (either by conducting post-treatment efficacy check-ups or faecal egg count reduction tests), consideration is needed when deciding upon the future use of this drug class. Generally, the respective drug class should not be used any more against the respective worm species.

6.1. Selective treatment approach

Repeated small strongyle infections occur in all age groups of grazing horses; however, in the majority of adult horses an immunological response leads to a suppression of small strongyle egg production. Several studies provided evidence for **consistency in** strongyle **egg shedding** after acquisition of immunity in individual horses. This phenomenon is the basis for selective treatment approaches in which only horses showing a pattern of consistently high strongyle egg shedding, exceeding a certain threshold e.g. 200 eggs per gram (EPG) of faeces, receive anthelmintic treatment. Practically, this approach involves a first year during which faecal samples from each horse are examined at least four times. All horses with strongyle EPGs above the threshold should be treated. If the responsible veterinarian regards the epidemiological situation as stable, the frequency of diagnostic examination can be reduced to three in the following years (beginning, mid and end of season, see Table 5).

The selective treatment approach is **only recommended for adult horses and exclusively designed for the control of small strongyles**. It aims at increasing the proportion of small strongyle eggs/larvae on pasture that were produced by worms that have not been exposed to anthelmintic treatment. This is known as a *refugium* of susceptibility and it has been hypothesised that a large environmental parasite *refugium* prevents or postpones the development of anthelmintic resistance. In various studies, including several from Europe, the application of the selective treatment approach has been shown to significantly reduce the number of anthelmintic treatments in horses. In these studies, horses did not develop clinical signs associated with parasite infection.

However, it is not completely certain that the intestinal worm burden of horses shedding only low numbers of strongyle eggs is in fact negligible. As mentioned above, high treatment frequency is considered one of the most relevant reasons for the emergence of AR. This is, however, much more of an issue in foals and yearlings, where previous recommendations of 4–8 weekly treatments should now be avoided. To date, it remains unproven whether the application of selective treatment in adult horses actually has a significant impact on the selection for AR in horses or if the reduction of treatment frequency in foals and young horses is more relevant. In this context, it is also worthwhile noting that it has been shown by a Danish study that the highly pathogenic large strongyle species *Strongylus vulgaris* is more prevalent on farms which had selectively treated their horses during recent years compared with those which had employed strategic whole herd treatments. It should however be noted that the selective treatment approach employed in these farms differed from that described herein, particularly concerning the monitoring of *S. vulgaris* presence and the respective treatment decisions.

Strongylus vulgaris or other large strongyle species have not, or only very rarely, been found in recent European studies using larval cultures and microscopic identification of L3. However, it has been reported in several single cases and studies, often associated with severe clinical consequences, which demonstrates that the parasite is nevertheless still present but at low levels. Consequently, monitoring the occurrence of large strongyles using faecal larval cultures has to be an integrated component of the selective treatment programme and this approach should not be recommended on farms where large strongyles have been shown to occur. Before (re)integration of stables harbouring large strongyles into a selective control programme, biannual anthelmintic treatments (i.e. late spring and autumn/winter) using drugs active against the adult AND larval stages of the large strongyles (MLs and FBZ), should be given to all grazing horses for at least two years. The large strongyle status of the farm should be documented by examination of pooled larval cultures at least once per year. All other treatment decisions remain the responsibility of the veterinarian in discussion with their horse-owning clients.

6.2. Strategic treatment approach

A horse's age and usage can determine the appropriate worm treatment. Foals particularly but also young horses need comprehensive protection with regular anthelmintic treatments, even on well-managed farms with good stable and pasture hygiene. While in the past it was often recommended to treat foals frequently (up to every 4–8 weeks during their first year), because of AR e.g. in ascarids and non-migrating strongylids, this is no longer considered appropriate. Generally, the first treatment during the grazing season will be either at turnout or 1–2 months post turnout, which is considered strategically more meaningful in order to obtain a higher epidemiological impact on the production of strongyle larvae and thus pasture contamination.

The tabulated, age-group-specific schemes for treatment plans shown in tables 2–4 provide concrete guidance as to which control measures (including monitoring of infection) should be employed at which time points throughout the year. Using this approach, generally all animals of the same age group are treated.

One disadvantage of the strategic treatment approach is that a certain proportion of horses will be dewormed even though they do not harbour any, or only very few, worms in their intestine. As mentioned above, unfortunately these are not necessarily horses showing no worm eggs in their faeces. By reducing the use of the same drug class to a maximum of twice a year, it is assumed that the selection of anthelmintic resistance will be reduced. It is however, currently unclear whether two annual treatments select for resistance in equine helminths and therefore some experts refrain from recommending this approach.

Table 2: Age-specific scheme for a treatment plan[1] of grazing foals.

Time point of treatment	Indication	Drug class[2]	Animals to be treated	Remarks
Age approx. 4 weeks (e.g. April/May)	*Strongyloides westeri*	BZ or ML	All foals	Monitoring[3] by faecal examination, **treatment only if *S. westeri* found on farm**
Age 2 months (e.g. May/June)	Cyathostomins, *Parascaris*, large strongyle larval stages	BZ or PYR[4] or ML[5]	All foals	Monitoring[3] at three months of age by faecal examination
Age 5 months (e.g. August/September)	Cyathostomins, *Parascaris*, possibly tapeworms	BZ or PYR[4], PZQ but only if tapeworms found on farm	All foals	Monitoring[3] by faecal examination
Age 8 months (November/December)	Cyathostomins, *Parascaris*, possibly *Gasterophilus*, tapeworms, large strongyles	ML[5], PZQ but only if tapeworms found on farm	All foals	Monitoring[3] by faecal examination

1   Treatment plans need to be adapted specifically to the farm and region.

2   Drug classes: benzimidazoles incl. pro-benzimidazoles (BZ), macrocyclic lactones (ML), the tetrahydropyrimidine pyrantel (PYR) and the isochinoline praziquantel (PZQ).

3   Monitoring: These dates are suitable for the qualitative monitoring of the overall herd infection status. Individual animal testing provides the most reliable data, however where this is not feasible, pooled sample testing (e.g. of up to five horses) can provide qualitative information on the spectrum of parasites present. If monitoring provides positive results, a faecal egg count reduction test could be performed to confirm drug efficacy. If performed quantitatively, the analysis of a pooled faecal sample may also provide an estimate of the strongyle egg shedding intensity in the respective group.

4   BZ-resistance in cyathostomins is widespread and PYR-resistance also common therefore these drug classes should only be used if efficacy has been confirmed on the farm using post-treatment coproscopic testing.

5   ML-resistance in *Parascaris* is widespread, particularly on stud farms therefore MLs should only be used if efficacy has been confirmed on the farm using post-treatment coproscopic testing.

Table 3: Age-specific scheme for a treatment plan[1] of grazing yearlings and young horses (up to and including four years old).

Time point of treatment	Indication	Drug class[2]	Animals to be treated	Remarks
Age 11–12 months (February/March)	Cyathostomins, *Parascaris*	BZ or PYR[4]	All yearlings/young horses however only if monitoring provided evidence of infection	Monitoring[3] by faecal examination
1–2 months post turn out (June/July)	Cyathostomins, *Parascaris*, possibly large strongyles	ML[5]	All yearlings/young horses	Monitoring[3] by faecal examination
4–5 months post turn out (August/September)	Cyathostomins, *Parascaris*, possibly tapeworms	BZ or PYR[4]	All yearlings/young horses	Monitoring[3] by faecal examination
At housing (November/December)	Cyathostomins, *Parascaris*, possibly *Gasterophilus*, tapeworms, large strongyles	ML[5], PZQ but only if tapeworms found on farm	All yearlings/young horses	Monitoring[3] by faecal examination

1   Treatment plans need to be adapted specifically to the farm and region.

2   Drug classes: benzimidazoles incl. pro-benzimidazoles (BZ), macrocyclic lactones (ML), the tetrahydropyrimidine pyrantel (PYR) and the isochinoline praziquantel (PZQ).

3   Monitoring: These dates are suitable for the qualitative monitoring of the overall herd infection status. Individual animal testing provides the most reliable data, however where this is not feasible, pooled sample testing (e.g. of up to 5 horses) can provide qualitative information on the spectrum of parasites present. If monitoring provides positive results, a faecal egg count reduction test could be performed to confirm drug efficacy. If performed quantitatively, the analysis of a pooled faecal sample may also provide an estimate of the strongyle egg shedding intensity in the respective group.

4   BZ-resistance in cyathostomins is widespread and PYR-resistance also common therefore these drug classes should only be used if efficacy has been confirmed on the farm using post-treatment coproscopic testing.

5   ML-resistance in *Parascaris* is widespread, particularly on stud farms therefore MLs should only be used if efficacy has been confirmed on the farm using post-treatment coproscopic testing.

Table 4: Age-specific scheme for a strategic treatment plan[1] of grazing adult horses.

Time point of treatment	Indication	Drug class[2]	Animals to be treated	Remarks
February/March	Cyathostomins	BZ or PYR[4]	All horses, however only if monitoring provided evidence of infection	Monitoring[3] by faecal examination
1–2 months post turn out (June/July)	Cyathostomins, possibly large strongyles	ML	All horses	Monitoring[3] by faecal examination
4–5 months post turn out (August/September)	Cyathostomins, possibly tapeworms	BZ or PYR[4], PZQ but only if tapeworms found on farm	All horses, however only if infection was proven by prior monitoring	Monitoring[3] by faecal examination
At housing (November/December)	Cyathostomins, possibly *Gasterophilus*, tapeworms, large strongyles	ML, PZQ but only if tapeworms found on farm	All horses	Monitoring[3] by faecal examination, if positive possibly conduct FECRT[5]

[1]   Treatment plans need to be adapted specifically to the farm and region.

[2]   Drug classes: benzimidazoles incl. pro-benzimidazoles (BZ), macrocyclic lactones (ML), the tetrahydropyrimidine pyrantel (PYR) and the isochinoline praziquantel (PZQ).

[3]   Monitoring: These dates are suitable for the qualitative monitoring of the overall herd infection status. Individual animal testing provides the most reliable data and is the preferred approach. However where this is not feasible, pooled sample testing (e.g. of up to 5 horses) can provide qualitative information on the spectrum of parasites present. If performed quantitatively, the analysis of a pooled faecal sample may also provide an estimate of the strongyle egg shedding intensity in the respective group. If monitoring provides positive results, a faecal egg count reduction test could be performed to confirm drug efficacy.

[4]   BZ-resistance in cyathostomins is widespread and PYR-resistance also common therefore these drug classes should only be used if efficacy has been confirmed on the farm using post-treatment coproscopic testing.

[5]   FECRT: faecal egg count reduction test.

Table 5: Schedule and key procedures for selective treatment[1] of small strongyle (cyathostomin) infections in adult horses.

Year 1	Year 2 and later
■ Four FECs[2] starting in April/May until Oct/Nov ■ Treat all horses with strongyle FEC ≥200 ■ Do post-treatment faecal analysis check-ups ■ Perform large strongyle testing (larval culture/PCR) ■ Treat all horses with evidence for other parasite infections (e.g. *Parascaris*, tapeworm, large strongyles)[3] ■ Keep one treatment at the end of the year for those horses who have not received diagnostic-based treatment during the season (employ drug with activity against migratory stages of large strongyles) ■ Perform strict quarantine procedure (see chapter 5)	■ Same procedure as in year 1 but frequency of faecal egg counts can be reduced to three if the epidemiological situation is stable

[1]   It is recommended that selective treatment is only employed in adult horses and not in stables harbouring large strongyles. Monitoring can be adapted regionally based on epidemiological evidence. The selective treatment concept is preferably employed as a 'whole stable approach' and not for single horses grazing together with unmonitored horses.

[2]   Faecal egg counts (FECs) based on methods with at least a lower detection limit of 50 eggs per gram.

[3]   Sensitivity of larval culture is lower than a combination of larval culture and PCR. These methods can only detect patent infections while disease is caused during prepatency by larval stages.

7. Training of practice team, guidance for the horse owner

Protocols and recommendations for the control of parasitic infection should be communicated clearly to the veterinary and para-veterinary staff and consistently advised. Preventive measures, routine monitoring and regular deworming practices should be made clear to the horse owners by veterinarians, veterinary nurses and other animal health professionals.

One goal of any parasite control programme in horses is to minimise the risk of parasitic diseases. This will involve strategic anthelmintic treatments or, in adult horses, selective treatments and in both cases needs to be accompanied by effective stable and pasture hygiene measures. Periodic faecal egg counts should be carried out to monitor anthelmintic efficacy and thus any signs of the development of anthelmintic resistance especially with regard to the cyathostomins and ascarids.

Parasite control programmes need to be tailored to the specific conditions existing on each individual horse farm or facility and should be discussed and developed with veterinary guidance.

8. Diagnosis of worm infections and anthelmintic resistance

8.1. Diagnosis of worm infections

As for most other hosts, faecal examination still represents the method of choice for the identification of horses infected with worms.

The basic principles of flotation and sedimentation (and the combination of both) followed by microscopic examination are employed for the detection of eggs and/or larvae from nematodes and trematodes, respectively. However, several recent developments have provided methods or protocols with improved sensitivity i.e. reduced lower detection limits. These include the FLOTAC and mini-FLOTAC methods, which exhibit a high sensitivity of 1 and 5 strongyle EPG, respectively, and a quantitative assessment of excreted worm eggs.

Faecal cultures of strongyle eggs to obtain L3 stages can be performed with plastic cups, where faecal samples should be placed in incubators with temperatures of 25–27°C, with relative humidity of 80–100%. After 14 days, L3 can be identified to genus/species level based on morphometric characters using keys available in the literature.

For the detection of tapeworm eggs, a double centrifugation/combined sedimentation-technique using sugar solutions (a flotation technique with considerably improved sensitivity when compared to standard flotation methods) has been described. Additionally, exposure to tapeworm infection (i.e. *A. perfoliata*) can also be examined by using serological and, more recently, saliva-based ELISAs. These latter tests have considerably higher sensitivity so are far better suited to the identification of horses requiring treatment and thus, may also allow the application of the selective treatment approach for the control of tapeworm infections.

Generally, repeated faecal examinations are recommended for each horse during the course of the year (see Table 2–4). However, where this is not feasible (e.g. practically or economically), repeated analysis of pooled faecal samples of up to five horses (of the same age group) should be performed to monitor the overall spectrum of worm infections present within the respective group of horses and to obtain an assessment of the infection quality (e.g. parasite species composition on the farm).

8.2. Diagnosis of anthelmintic resistance

The faecal egg count reduction test (FECRT) currently represents the only established approach for the field analysis of anthelmintic susceptibility in equine worm populations. This test can be readily employed for the evaluation of the efficacy of all nematocidal drug classes against strongyle and *Parascaris* spp. populations. In principle, this test can also be used to assess the effect of anthelmintics against other parasites like liver fluke or lungworms. However, this has not been established for horses so far. Due to the inconsistent occurrence of worm eggs, this test is less meaningful for further species like pinworms or tapeworms. Where full FECRT are not feasible (e.g. practically or economically), it is strongly recommended to regularly monitor the efficacy by pooled post-treatment sample examinations (once per drug class within three years).

9. Supplement: minor species

The common liver fluke (*Fasciola hepatica*)

The common liver fluke, *F. hepatica*, is a prevalent helminth parasite mostly found in domestic and wild ruminants and is seldom associated with infection and disease in horses. However, liver fluke infection may be of significance in areas where horses share pastures with ruminants or graze those previously occupied by ruminants. Therefore, liver fluke in horses is mostly associated with areas where fasciolosis is endemic in ruminants. Suitable environments for the development of the intermediate host, the snail *Galba truncatula*, may be found in regions with high annual rainfall and poorly-drained pastures. That is to say, climate and soil conditions play an important role in the epidemiology of the infection. Occasionally, *F. hepatica* can also infect humans.

Metacercarial stages are ingested while grazing and reach the liver after penetration of the intestinal wall via the peritoneal cavity. Following several weeks of migration through the liver tissue and penetration into the bile ducts, juvenile worms develop into adult flukes. The prepatent period is approximately two months, after which the 120–150 µm large, ovoid, operculated, yellowish eggs can be found in the faeces.

Very little is known about the pathological consequences of liver fluke infection in horses. They appear to be more resistant to the infection than cattle or sheep and *F. hepatica* infection reaches patency only in a small number of cases. Most of the pathology is produced by the juvenile flukes while migrating within the hepatic parenchyma but peritonitis and secondary bacterial infections may occur due to the transperitoneal route of migration. Within the bile ducts, liver flukes produce an inflammatory response which can be associated with reactive hyperplasia and erosion of the epithelium, chronic cholangitis, cholestasis and fibrosis. Subclinical infections are not uncommon in horses and when clinical signs do occur, the most common are those of a chronic non-specific anaemia.

However, fluke infections in horses may remain undetected for a long period of time due to the non-specific clinical signs. Faecal analyses for the presence of fluke eggs may be performed, but this is unreliable and a negative test for fluke eggs does not mean that the horse is not infected. Haematological changes include increases in liver enzyme levels such as sorbitol dehydrogenase (SDH), aspartate aminotransferase (AST), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) and -glutamyltransferase (-GT), which may be associated with increased concentrations of conjugated bilirubin. In horses with non-patent infections, a serum agglutination test can be used. Tests developed for serological detection of *F. hepatica* infections in ruminants provide unreliable results unless they are adapted to employ horse secondary antibody coupled conjugate.

Horses with suspected or clinical fluke infections can be treated with triclabendazole at a dosage of 12 mg/kg bodyweight. Although this product is not licensed for horses, several independent reports confirm its efficacy and safety in treating equine *F. hepatica* infections. Triclabendazole is the only drug with efficacy against both juvenile and adult flukes. However, in ruminants, triclabendazole resistance has been reported in flukes in several countries. Albendazole has limited activity, and mostly against adult stages. Other drugs (e.g. clorsulon or closantel) used to treat fluke infection in cattle and sheep can be toxic for horses, and veterinarians/horse owners should be aware of this. Control of the intermediate snail host populations depends upon pasture management by improved drainage, which is often impractical or prohibitively expensive.

Lungworm (*Dictyocaulus arnfieldi*)

The equine lungworm, *Dictyocaulus arnfieldi*, is a nematode which occurs most commonly in the donkey. Although less common, patent infections can also be found in mules and horses, particularly in foals and yearlings. Cross transmission may occur when these different hosts share the same pasture. This parasite can measure up to 6 cm in length and can be found in the bronchial tree, especially in the terminal bronchioles. Adult females lay eggs containing first-stage larvae (L1) in the bronchial secretions which are transported with the mucus to the pharynx, swallowed and passed in the faeces. The eggs hatch almost immediately to release L1 which moult twice to produce third stage infective larvae (L3). Infection occurs by ingestion of L3 with herbage. Following ingestion of L3, the larvae penetrate the small intestinal wall and migrate via the lymph and blood vessels to the heart and lungs. There they penetrate the alveoli and develop into adult stages in the bronchial tree. The prepatent period is about three months.

The most common finding in the majority of clinical cases of lungworms in horses is a history of previous direct or indirect contact with donkeys. Major significant lesions are those of chronic eosinophilic bronchitis and bronchopneumonia. Clinically, chronic coughing is the most frequent sign; occasionally there may be a mucopurulent bilateral nasal discharge, dyspnoea, tachypnoea and weight loss. The clinical disease is more severe in young horses (yearlings). However, pony foals may be infected and shed L1 in the faeces while showing no clinical signs. Infected donkeys rarely show any signs of infection despite the presence of adult worms in the lungs. Comparatively, mild clinical signs like hyperpnoea and harsh respiratory sounds can be associated with some infections; however, there are a few reports of severe outbreaks of disease in adult animals which have resulted in fatalities, even in donkeys.

Diagnosis is mainly based on grazing history and clinical signs, since in horses, lungworm infections rarely develop to patency. Confirmation of a patent infection can be achieved by the demonstration of embryonated eggs or free L1 of *D. arnfieldi* (420–480 µm length) isolated by flotation technique in combination with the Baermann method. In some cases, bronchoalveolar lavage has proved successful in the recovery of eggs/L1 and L4/L5 of *D. arnfieldi* from the nasal and upper respiratory tract. A positive clinical response to appropriate anthelmintic treatment in suspected cases may be an indicator that lungworm infection was indeed present.

In general, equine lungworm infections are well controlled on farms with good parasite control measures in place. However, when respiratory signs including coughing are associated with a poor response to antibiotics, a parasitic pneumonia should be considered as a possible diagnosis, especially if donkeys are or were present on the farm. This is particularly true when the anthelmintic control programme is infrequent and when there is a history of horses and donkeys grazing on the same pasture. Generally, control of equine lungworm infection will be achieved by following the general control measures for parasitic diseases in horses. The MLs and BZ anthelmintics are effective against this parasite. It is likely that control programmes for large and small strongyles which use these compounds and are applied strategically during the course of the year will also be effective in the control of *D. arnfieldi* infections.

Stomach worms (*Trichostrongylus axei, Habronema* spp. and *Draschia megastoma*)

Trichostrongylus axei is a small (5–6 mm), slender, whitish nematode that can be found mostly in the stomach and rarely in the small intestine of equids. It is a common parasite of domestic and wild ruminants, and even wild lagomorphs, occurring worldwide. Cross-infection may occur when these different host species share the same pasture. Different species of equids may show different patterns of infection; for example, *T. axei* may show a higher prevalence in donkeys than in horses and infection may result in high worm burdens. The life cycle is direct and its non-parasitic development similar to that of equine strongyles, involving egg shedding in the faeces and development of infective L3 on the pasture. After ingestion, L3 penetrate the mucosa of the stomach, mainly entering the gastric glands; in massive infections they may also parasitise the anterior small intestine. After two moults, the adult stages emerge to the lumen and females will shed strongyle-type eggs as early as 14 days post infection.

Clinical signs vary depending upon the intensity of infection. They can range from mild gastrointestinal disturbances to chronic catarrhal gastritis and in massive infections, nodular thickenings of the glandular mucosa, with erosions and ulcer formation. Severe signs may include loss of condition and progressive weight loss leading to emaciation.

The faecal flotation methods used for the detection of strongyle-type eggs are also suitable for *T. axei* eggs, which are morphologically indistinguishable. Thus, species-specific diagnosis requires faecal culture and microscopic identification of the slender *T. axei* L3. The MLs and BZ and pyrantel anthelmintics are effective against this parasite. It is likely that control programmes for large and small strongyles which use these compounds and are applied strategically during the course of the year will also be effective in the control of *T. axei* infections.

Other stomach nematodes include three species of spiruroids: *Habronema microstoma, H. muscae* and *Draschia megastoma*. These species each have an indirect life cycle and require an arthropod intermediate host for transmission to the horse. The adult parasite stages are found in the stomach, while the larval stages may be found in the stomach or in aberrant sites such as the skin or the conjunctiva of the eyes, where they are responsible for the development of "summer sores".

The adult nematodes are 10–25 mm long, *Habronema* spp. being the longest (22–25 mm) and *D. megastoma* the shortest (13 mm).

All three species are commonly found in the glandular portion of the stomach both as L4/pre-adults and as adults. Females lay thin-shelled embryonated eggs and both eggs and first stage larvae (L1) can be passed in the faeces. These nematodes use muscid flies as intermediate hosts and vectors, *Musca domestica* for *H. muscae* and *D. megastoma* and the stable fly *Stomoxys calcitrans* for *H. microstoma*. Fly larvae (maggots) ingest L1 from the faeces and these develop to third stage larvae (L3) in one week. The L3 concentrate in the mouth parts of the adult fly and they can then be deposited around the horse's mouth and muzzle and subsequently ingested. Alternatively, horses can become infected through the ingestion of dead flies. Swallowed L3 complete their life cycle after two moults into adult worms in the stomach of equines. During their development in the stomach, there are marked differences in terms of their pathogenic effects. For example, *D. megastoma* induces the formation of nodules, which are globular granulomas filled with purulent material in which the worms live; these may extend into the lumen of the glandular stomach, and become large, sometimes over 10 cm in diameter. *Habronema* species induce a catarrhal gastritis with occasional haemorrhage and ulceration.

The L3 of these spiruroid nematodes are occasionally deposited in skin wounds or at mucocutaneous interfaces such as the eye conjunctiva and anal or vulvar mucosae, being responsible for proliferative lesions that grow and ulcerate throughout the fly season as "summer sores". These skin and mucocutaneous lesions tend to regress during the cold months of autumn and winter. However, new lesions may occur as temperatures increase in spring and summer, since fly development increases and these deposit new L3, stimulating more granulomatous lesions.

The skin and mucocutaneous lesions are fibro-granulomatous, rich in connective tissue and eosinophils, frequently with secondary bacterial infection and ulceration. They may be extremely painful if located at the medial canthus of the eyes or in skin areas under the saddle or harness or on the prepuce. This makes the skin infection by larvae much more serious and irritating compared to adult worm infections in the stomach. However, controlling the stomach population is necessary to reduce the risk and incidence of "summer sores".

The MLs are the drugs of choice for the treatment of infections with adult *Habronema* and *Draschia* species. Cutaneous lesions can be treated with systemic MLs, although treatment failure of MLs is reported from the field.

10. Glossary

Anthelmintic
A drug used to control worm (helminth) infections.

Anthelmintic resistance
The ability of a worm population to survive the standard recommended and effective dose of an anthelmintic; this is a heritable trait.

Definitive (or final) host
This is the host in which a parasite completes its development into the sexually mature/adult stages producing eggs or larvae.

Efficacy
This is the ability of a drug to produce the desired therapeutic effect at the recommended dosage. In the field, faecal egg count reduction tests are used to indicate efficacy.

Eggs per gram (EPG)
The number of helminth (usually nematode) eggs per gram of faeces from an animal.

Egg re-appearance period (ERP)
The time interval between the last effective anthelmintic treatment and the reappearance of egg shedding.

Faecal egg count reduction test (FECRT)
This test provides an assessment of treatment efficacy based on pre- and post-treatment faecal egg counts. FECRT is recommended for detecting anthelmintic resistance in grazing animals.

Helminth
A parasitic worm such as a roundworm (ascarid, strongyle, pinworm), tapeworm or fluke.

Hypobiosis
Arrested development normally in worm larval stages in the intestinal mucosa of the definitive host.

Intermediate host
This is a host harbouring immature stages of a parasite species which develop into infective stages for the definitive host.

L1 – L2 – L3 – L4 – Pre-adult
This is the normal larval development sequence of nematodes, beginning with the first larval stage (L1) which moults four times to the pre-adult stage. Generally, the development of equine nematodes from first stage larvae (L1) to third stage larvae (L3) occurs in the environment or in an intermediate host and the fourth stage larvae (L4), pre-adult and adult within the horse.

Metaphylactic measures
Measures given to infected, but not yet sick or damaged, host animals taken to prevent or minimise an expected disease.

Myiasis
Infection of vertebrates e.g. a horse, by developing fly larvae.

Patent period
Time period during which the parasites are sexually mature and produce offspring (e.g. eggs or larval stages), ending when they cease to reproduce or with their death.

Prepatent period
This is the time interval between infection of a definitive host with a parasite species and the first detection of eggs or larval stages in the faeces.

Prevalence
A term describing the proportion (usually given as a percentage) of infected hosts within any group of animals.

Prophylactic measures
Measures taken to prevent or reduce the risk of infection.

Refugium
The parasite population that is not exposed to the drug at the time of treatment e.g. parasites in non-dewormed horses, larval stages on the pasture or encysted worms not affected by the anthelmintic used.

8 A guide to the treatment and control of equine gastrointestinal parasite infections

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