Parasitology

17

17.1 Internal parasites

Gastro-intestinal parasites of working equids

Studies have shown that gastro-intestinal (GI) parasites have been detected in a high proportion of working equids: 76% of horses and 71% of mules in one study of 532 samples in India (Singh et al. 2012), and 91% of animals had a positive faecal egg count (FEC) in a study of 112 donkeys, horses and mules in Mexico (Valdez-Cruz et al. 2006).

Type of parasite	Predilection sites
Large strongyles (Strongylus spp. particularly vulgaris)	Adults Large intestine Larvae Liver and arteries
Small strongyles (Cyathostomes)	Adults Large intestine Larvae Intestinal wall
Roundworms (Parascaris equorum)	Adults Small intestine Larvae Liver and lungs
Pinworms (Oxyuris equi)	Adults Large intestine Larvae Intestinal wall
Tapeworms (Anoplocephala spp.)	Adults Large and small intestine Larvae Forage mites
Thread worms (Strongyloides westeri)	Adults Small intestines Larvae Lungs and other tissues
Liver fluke (rarely causes disease in equids) (Fasciola hepatica - temperate Fasciola gigantica - tropical)	Adults Liver Larvae Snail

Table 16.1.1 Commonly found GI parasites of equids and predilection sites.

Clinical signs

Reported clinical signs for equids affected by GI parasites are varied, including poor coat health, anorexia, weight loss, diarrhoea, colic, rectal prolapse, lack of vigour, poor work performance, and productive loss (Singh et al. 2012).

The pre-patent period is the period between infection with a parasite and the demonstration of the parasite in the body, determined by the recovery of an infective form (for example by detecting eggs during a faecal egg count; FEC).

Large strongyles

Strongylus vulgaris

Adult nematodes vary between 1.5 and 5 cm and live in the large intestine.

Life cycle

Eggs are passed in the faeces and develop into infective larvae in the soil. Larvae are ingested, then penetrate intestinal mucosa, enter small arteries and migrate in the endothelium to the predilection site at the cranial mesenteric artery and its branches. After development, the nematodes return to the intestinal wall, via the artery lumen, to form nodules in the wall of the caecum and colon. The adults remain within the walls of the large intestine, and eggs are excreted in the faeces.

Clinical signs

> The most significant pathology is caused by migration of the larval stage through the cranial mesenteric arteries and adjacent branches.

Migration induces thrombus formation and thickening of arterial walls. Blockage of the blood supply to the intestine results in infarction and severe colic. If the cranial mesenteric artery is completely occluded the intestines become necrotic; this condition is fatal. A high count of large strongyles causes weight loss and anaemia.

Diagnosis

- Clinical signs
- Confirm diagnosis using an FEC.

> The faecal egg count cannot distinguish between species of strongyle (large and small).

- Speciation is possible by providing favourable conditions for the eggs to hatch; the larvae of each species can then be differentiated. Larval culture takes several days and this technique increases the cost of laboratory diagnosis.
- The FEC only reflects the number of egg-laying adult parasites present. Currently there is no test available that can accurately detect migrating larvae, which is the most pathogenic stage. Investigations are on-going to develop a pre-patent test (Anderson et al. 2013).

Treatment

All stages of the life cycle are susceptible to benzimidazoles, ivermectin and moxidectin. In equids that are regularly de-wormed, S. vulgaris has been largely controlled. However, working equids may not receive this treatment so large strongyles should still be considered a priority.

Strongylus edentatus

Life cycle

The life cycle of S. edentatus is similar to that of S. vulgaris except the larvae migrate through the liver. From here the larvae return to the large intestine sub-peritoneally.

Clinical signs

Migration of the larvae causes liver damage, and large numbers of adults in the intestine result in unthriftiness, weight loss and anaemia. However, the severe colic associated with intestinal infarction is not a feature.

Diagnosis and treatment

As for S. vulgaris

Small strongyles

Small strongyles, also referred to as Cyathostomes, have a non-migratory life cycle. There are more than 50 species in this genus infecting all equids (Eysker et al. 1989). Cyathostomes are generally less than 1.5 cm in length and range in colour from white to dark red.

Life cycle

Eggs are excreted in the faeces and develop into infective larvae in the soil. Larvae are ingested and invade the wall of the large intestine. Larvae develop into adults and emerge into the gut lumen.

Verminous enteritis: If environmental conditions are not favourable for development (e.g. cold winter, dry season) larvae hypobiose (arrest development) and encyst in the large intestinal wall. The numbers of larvae in the large intestinal wall gradually increase over several months. When environmental conditions improve larval development is triggered. Mass emergence of encysted larvae from the gut wall causes significant damage to the large intestine.

Clinical signs

- The signs of cyathostome infection are more moderate than that of large strongyles but include loss of condition, peripheral oedema, vague malaise, weight loss, poor appetite, lethargy and disrupted intestinal motility.
- Mass emergence results in a marked pathology, 'verminous enteritis', with profuse diarrhoea, colic, weight loss, dehydration, inappetance, dullness depression and protein-losing enteropathy.
- Mortality rates can be as high as 40–70%.

Diagnosis

- Clinical signs. Examine all animals sharing grazing.
- An FEC can confirm the presence of strongyles, although large and small strongyle eggs cannot be distinguished.
- The FEC reflects only the number of egg-laying adult parasites present. Eggs are not produced by the encysted hypobiosed larvae. The FEC may be low in equids presenting with signs of verminous enteritis.
- In heavy burdens, adult worms may be visible in the faeces.

Treatment

There are three drug classes effective against the cyathostomes: benzimadazoles, pyrantel and macrocyclic lactones. Cyathostomin larvae have a low susceptibility to anthelmintics when in the hypobiosed state. Treatment and control is also complicated by the fact that there is widespread resistance to benzimadazoles (Eysker et al. 1989), so this medication is frequently ineffective. Ivermectin resistance is also present. Multi-drug resistance is a major problem for treatment efficacy. Paradoxically, treatment with anthelmintics is thought to trigger mass emergence and severe verminous enteritis.

Treatment for verminous enteritis

Give supportive treatment, including fluid therapy to address dehydration, and a balanced,

palatable diet. Treat with moxidectin, in preference to fenbendazole, as the latter can cause severe inflammation of the colon as the larvae die off (Steinbach et al. 2006). Administer steroid treatment, dexamethasone or prednisolone is advised, particularly before anthelmintic treatment, to reduce the gut inflammation caused by the killed larvae. Prognosis is poor and mortality is high even with intensive treatment. If one equid has shown clinical signs of verminous enteritis then the others in the same grazing system are likely to have high burdens.

Roundworms

Parascaris equorum is one of the largest endoparasites and can grow up to 40 cm.

Life cycle

Roundworms have a direct life cycle with no intermediate host. Eggs are excreted in faeces and the larvae develop into the infective stage whilst in the egg. Following ingestion, the larvae penetrate the small intestinal wall and migrate through the liver and lungs. Larvae are coughed up from the lungs and swallowed. Development is completed in the small intestine. Adult roundworms produce high numbers of eggs, so transmission rates are high.

Clinical signs

- Mild coughing and nasal discharge in the migratory phase. Affected equids remain bright and alert.
- Light intestinal infections are tolerated well with no clinical signs.
- Heavy infections result in unthriftiness, a dull coat, poor growth in young stock, and lethargy. Younger animals can show signs of colic if a heavy roundworm burden causes a blockage of those parts of the digestive tract with a particularly narrow lumen.
- Significant clinical signs are reported predominantly in foals as adults generally develop resistance. However, Parascaris equorum does cause clinical disease in adult working equids, possibly due to reduced immunity as the result of physiological stress. In a recent large-scale study of working adult donkeys in Ethiopia, Parascaris eggs were found in 51% of the animals surveyed (Getachew et al. 2010b).

Diagnosis

- Clinical signs
- An FEC can confirm infection with roundworm. Parascaris eggs have a characteristic thick shell.
- Worm egg counts only detect adult egg-laying roundworm.

Treatment

Generally ascarids are readily controlled by routine de-worming with broad spectrum anthelmintics. However, macrocyclic lactone resistance has been reported. Adjust the treatment strategy to use only those anthelmintics known to be effective in indigenous populations (Reinemyer 2009). In areas where Parascaris infection is common, treat foals at 2 months, then every 2 months until 1 year of age.

Pinworm

Pinworms (Oxyuris equi) are a common parasite in equids but are often non-pathogenic. Female adult worms can reach up to 10 cm.

Life cycle

Adult worms live in the lumen of the colon. After fertilisation the female migrates to the anus and deposits eggs in yellowish white streaks on the perineal skin. Eggs are deposited on the pasture in the faeces, and then ingested. Larvae encyst in the large colon, develop, and the adults are released into the lumen of the colon.

Clinical signs

- Pinworms rarely cause signs of GI pathology.
- Intense pruritus around the anus can lead to self-trauma.

Diagnosis

Use sticky tape around the perineum to recover eggs for microscopic examination.

Treatment

Oxyuris equi is well controlled by all anthelmintics. Resistance to macrocyclic lactones has been reported in some populations. A small clinical trial did not find resistance when horses were treated with ivermectin and pyrantel (Reinemyer et al. 2010). If marked pruritus is evident, clean the underside of the tail and perineal area regularly with a disposable cloth.

Tapeworms

Tapeworms (Anoplocephala spp.) have an indirect lifecycle with an insect vector.

Life cycle

Eggs are passed in the faeces in a gravid segment. Following disintegration of the segment, eggs are ingested by forage mites in the soil where larval development occurs for 2–4 months. The forage mite is eaten and the adult tapeworm is released into the intestine. Anoplocephala perfoliata is found around the ileo-caecal junction and causes ulceration at the site of attachment.

Clinical signs

Mild infestations are considered non-pathogenic; however, moderate to severe tapeworm burdens have been associated with spasmodic colic, intussusception and intestinal rupture.

Diagnosis

Although an FEC can be used to demonstrate infection with tapeworm, this test has been shown to be unreliable (Meana et al. 1998). A test for circulating antibodies is available, although this has a low sensitivity (Skotarek et al. 2010).

Treatment

Praziquantel is an effective treatment for tapeworms (Slocombe et al. 2007). Control of tapeworms can difficult as there is a reservoir of infection within the forage mite population.

Liver fluke

Detection of liver fluke (Fasciola spp.) in working equids is generally low, particularly in arid climates (Haridy et al. 2002, Tavassoli et al. 2010). High burdens have been reported in some populations and should be considered as a differential diagnosis in cases of suspected hepatic pathology (Getachew et al. 2010a).

Stomach bot

The stomach bot (Gasterophilus spp.) is the larvae of the bot fly and is generally considered to be of little clinical significance.

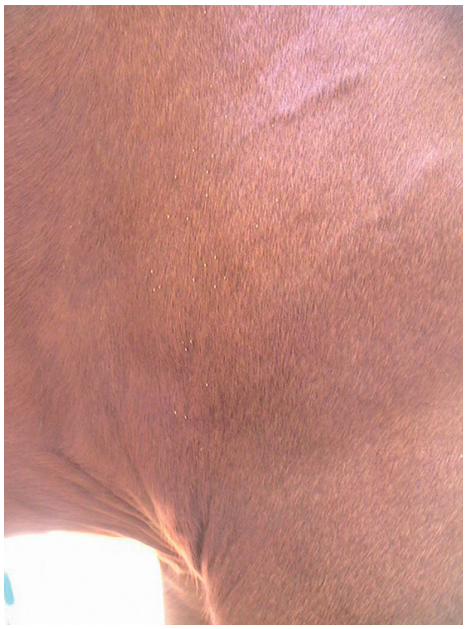


Figure 17.1.1 Bot fly eggs on the shoulder of a horse.

Life cycle

Adult flies lay eggs on the coat of equids. Eggs are easily visible, 1–2 mm long and creamy white in colour (Figure 17.1.1). Larvae either crawl into the mouth or are ingested with grooming. Larvae penetrate the tongue/buccal (cheek) mucosa, and from here migrate to the stomach. Bot larvae tend to attach in the cardiac region of the stomach and remain for up to a year. Some species of bots will re-attach to the rectal mucosa on the passage through the digestive tract. The larvae pupate in the soil and emerge as adult flies.

Clinical signs

- Presence of larvae in the buccal cavity may cause inflammation, although this is rare.
- Mild localised inflammation/ulceration can occur at the attachment site in the stomach, although the true pathogenic significance of this is unclear.
- Attachment to the rectal mucosa has been associated with rectal prolapse (Getachew et al. 2008).

Treatment

The only medication available for the treatment of Gasterophilus spp. is ivermectin and moxidectin, both of which are important for the control of nematodes. As there is little evidence for the pathogenic significance of bot fly larvae, the use of these medications for mass treatment of this condition is questionable.

> Egg removal from the coat, and fly control will minimise infection with bot fly larvae.

Lungworm (Dictyocaulus spp.)

See Section 12.9 Parasitic respiratory disease.

17.2 Anthelmintic medication

Anthelmintics are chemicals used to expel helminths (parasitic worms) from the body. Anthelmintic resistance has become increasingly important in the equine veterinary field. Resistance to individual drug classes is described in this section, and the issue of anthelmintic resistance and strategies to reduce development are addressed in Section 17.4 Principles of a strategic de-worming programme.

Macrocyclic lactones

The macrocyclic lactones (avermectins and milbemycins) selectively paralyse parasites which are then pushed out of the gut lumen by peristalsis. The macrocyclic lactones also treat external parasites. Commercially available avermectins include ivermectin, doramectin and eprinomectin. Milbemycins include moxidectin and milbemycin oxime.

Ivermectin

Indications

Used in treatment of all major helminth parasites as well as lungworm, lice, mange, cutaneous and gastric habronemiasis, thelaziasis, Gasterophilus spp. (bots). Effective against microfilaria of Onchocerca but not the adult parasites. Ivermectin does not treat tapeworm. There is questionable efficacy against encysted cyathostome larvae both at therapeutic doses (Eysker et al. 1992) and at higher doses (Klei et al. 1993). This is not as the result of drug resistance as ivermectin still shows excellent control of lumen-dwelling adults (Monahan et al. 1996).

Dose 0.2 mg/kg PO

Parenteral administration (introduction of medication into the body via a route other than the mouth) of ivermectin can cause severe life threatening reactions. The licence for the injectable form for horses was withdrawn in 1984 following severe reactions, including fatalities, after parenteral administration (French et al. 1983; Leaning 1983; Reed 1983). Studies conducted subsequently, including use in donkeys (Binev et al. 2005), have not reported adverse effects. IV use has been reported to be safe but is not licensed and cannot be recommended.

Pour-on (topical) formulations of ivermectin are available for the treatment of cattle. When applied to horses the plasma concentration and systemic availability of ivermectin was lower than the oral route (Gokbulut et al. 2010). Therefore, the use of pour-on/topical formulations is not recommended as it is likely to increase the development of resistance.

Use the ivermectin paste formulation per os if available. If parenteral administration is the only option, make the owner aware of possible adverse reactions and ensure correct dosing.

Moxidectin

Broad spectrum coverage of parasites with a wide safety margin in healthy adult equids. Moxidectin has a prolonged action compared with other anthelmintics.

Indications

Moxidectin has a similar spectrum of activity to ivermectin except for a reduced efficacy for

treatment of Gasterophilus intestinalis, and an increased efficacy for encysted small strongyles (Moahan et al. 1996). Moxidectin does not treat tapeworm.

Dose 0.4 mg/kg PO

Moxidectin has a very long half-life in body fat, which results in a long period of suppression on equine faecal worm egg counts compared to other anthelmintics (long egg reappearance period). Young and emaciated animals have insufficient adipose tissue and are at risk of moxidectin toxicity. Symptoms of toxicity include adverse neurological reactions including prolonged coma and, in some cases, death (Johnson et al. 1999). Moxidectin should not be used in foals under 4 months old.

Resistance to macrocyclic lactones

Ascarids (roundworm) Decreased efficacy and resistance of ivermectin in ascarids has been reported since 2002 in North America and Europe. Parascaris equorum isolates have been reported to be resistant to both ivermectin (Hearn et al. 2003) and moxidectin.

Cyathostomins (small strongyles) Reduced efficacy of ivermectin and moxidectin against small strongyles has been reported (Lyons et al. 2011 and Molento et al. 2012).

Benzimidazoles

This class of anthelmintic acts by interfering with the energy metabolism of the parasite; decreasing the absorption and digestion of glucose. Pharmacologically, the onset of benzimidazole action is slow compared to other anthelmintics.

Fenbendazole

Indications

Large strongyles, small strongyles, oxyuris, ascarids (Parascaris equorum), Dictyocaulus arnfieldi, strongyloides. Fenbendazole is not effective against tapeworm or bots.

Doses The licensed dose rate is 7.5 mg/kg PO

When dosed daily for 5 consecutive days, fenbendazole is effective against all stages of mucosal cyathostome larvae including early third-stage hypobiotic larvae (Duncan et al. 1998). However, a more recent study did not support this treatment protocol for encysted cyathostome larvae (Rossano et al. 2010). The efficacy of fenbendazole against cyathostomins will depend on the resistance in the population present.

Mebendazole

Indications

Treatment of strongyles, cyathostomins, mature larval Parascaris equorum, adult Oxyuris equi and Dictyocaulus arnfieldi. Mebendazole is not effective against tapeworm or bots.

Dose 8.8 mg/kg PO

15–20 mg/kg for 5 days for Dictyocaulus arnfieldi in donkeys (Clayton et al. 1979)

Mebendazole is safe for pregnant mares and foals, but may cause mild diarrhoea if overdosed. Pregnant donkeys should not receive the higher dose regime.

Oxfenbendazole and Oxibendazole

Indications Treatment of ascarids, strongyles, and Oxyuris equi

Dose 10 mg/kg PO

Triclabendazole

Triclabendazole is not licensed for the treatment of liver fluke in equids. In a case of clinical necessity, this medication can be administered at a dose rate of 12 mg/kg. Small studies have shown no adverse effects of treatment (Trawford et al.1996; Rubilar et al. 1988).

Resistance to benzimidazole

The resistance to this group of anthelmintics can be considered together, as cross resistance between medications in this class is common.

Small strongyles Resistance is documented globally and has been reported for several decades (Slocombe et al. 1977; Kuzmina et al. 2008; Slocombe et al. 2008). Fenbendazole resistance has been very well documented throughout America, Europe and Australia.

Pyrimidines (pyrantel salts)

Three pyrimidines are registered for use in equids: pamoate, emboate and tartrate. These medications work by causing spastic paralysis of parasites.

Pyrantel emboate

Indications

Treatment of large and small strongyles, Oxyuris, Parascaris equi and Anoplocephala spp. (Owen and Slocombe 2004; Marchiondo et al. 2006)

Dose 19 mg/kg PO

Resistance to pyrantel

Cyathostomin resistance to pyrantel was found in one-third of 102 horse farms in the UK, Germany and Italy (Traversa et al. 2009). Resistance of cyathostomins is even more widespread in the US where pyrantel has been available for daily feeding.

Piperazine

Indications

Treatment of Parascaris equorum and adult strongyles. Piperazine does not treat bots, tapeworm or any larval forms. Used alone this medication has a narrow spectrum of activity; however, piperazine is frequently used in combination with benzimidazoles for broad-spectrum activity against benzimidazole-resistant cyathostomins. The narrow spectrum of activity of Piperazine, only effective against adult stages of cyathostomins/ascarids, and low therapeutic margin have limited its clinical value.

Dose 110 mg/kg PO (EMEA 1999)

There is a risk of photosensitisation. Rapid death and detachment of Parascaris can cause rupture or obstruction of the small intestine.

Praziquantel

Praziquantel causes spastic paralysis of the parasite. It is available in some countries in combination with ivermectin and benzimidazoles for the treatment of tapeworm.

Indications Tapeworm only

Dose 1.5 mg/kg PO

Resistance of roundworms to praziquantel is low although, as FEC examination is unreliable in tapeworm, resistance is difficult to measure. A small study in Ethiopia demonstrated efficacy of praziquantel against tapeworm (Getachew et al. 2013).

Principles of a strategic de-worming programme 17.3

Traditional helminth control programmes rely on regular administration of anthelmintics. There are three major drug classes currently being used: benzimidazoles, pyrantel and macrocyclic lactones. Parasitic resistance to these treatments, resulting in reduced efficacy, has been documented. Several factors can influence the rate at which anthelmintic resistance develops; high frequency of treatment is the most important. To reduce anthelmintic treatment frequency significantly, it is essential to examine the efficacy of the medication routinely for each drug class and to design a targeted control strategy, with management changes if necessary.

Figure 17.3.1 The epidemiological factors to consider when designing a de-worming programme.

Local epidemiology

- Which parasites are present in working equines and how common are they?
- Of these, which parasites are causing overt disease? (These must be the main targets of the de-worming strategy.)
- Which anthelmintic medication will be effective against these parasites?

Important helminths to consider in all de-worming strategies:

- Strongyles – large (S. vulgaris), small (Cyathostomes)
- Ascarids
- Tapeworms

European and American de-worming strategies and research focus heavily on cyathostomes as, in these countries, the large strongyle species are well controlled. In working equids this is often not the case and studies have shown that the predominant disease risk is from Strongylus vulgaris. Consider this difference when reading literature based on non-working equids.

Host demographics

Young equids are more susceptible to worm infestations than adults; frequently they have higher helminth burdens and increased rates of shedding. In Europe and America certain helminths, such as Parascaris equorum and Strongyles westeri, are thought to cause clinical disease only in young animals, as adults develop resistance. In working equids adults do not seem to develop such a strong immunity. Hard work and physiological stress may result in immune-compromise. Consequently, species such as Parascaris equorum may cause disease even in adulthood.

Regional climate

Larvae on pasture become desiccated in hot, dry conditions so, in this climate, pasture contamination is rapidly reduced. In warm, humid conditions the rate of larval development increases, raising the infection level of grazing equids. Consider administering de-worming treatment at the end of the dry season/beginning of the wet season to reduce worm burdens prior to a period of increased propagation.

Cyathostomes arrest development by encysting within the gut wall when environmental conditions are unfavourable, such as cold, or dry and hot weather. Schedule de-worming, if deemed necessary by an FEC, prior to the change in season to minimise the number of cyathostomes that enter arrested development.

Animal husbandry

Keep new equids in isolation for 3 weeks before introducing them to the herd. Treat with an anthelmintic and assess efficacy with an FEC.

Remove faeces, from stable/paddocks/grazing areas, at least once a week to reduce transmission of helminths and thereby reduce the dependence on anthelmintic medication.

Grazing management

Management practices can be combined with drug intervention to minimise the spread of helminths between animals. Rest heavily grazed ground, and limit density of numbers to reduce pasture contamination.

Anthelmintic resistance

Monitor and evaluate de-worming strategies regularly using FEC, FEC Reduction and by recording clinical disease caused by helminths. If resistant parasites are detected, withdraw the use of this anthelmintic.

The proportion of a parasite population not exposed to anthelmintic treatment is described as 'refugia'.

Although many factors affect the rate at which resistance develops, levels of refugia are considered the most important as these parasites provide a pool of sensitive genes in the population.

It is the aim of any responsible worming programme to maximise the number of worms in refugia. This is done by limiting treatment to only those animals that require it, for example using FEC and treating young animals and those with counts above a pre-determined threshold.

In most host-parasite situations, the majority of animals in a group have relatively few parasites; environmental egg contamination comes from a minority of individuals with a heavy worm burden. In order to benefit the whole group and reduce the use of anthelmintics, identify and treat only these animals. Untreated animals will provide a pool of susceptible eggs (refugia). It is preferable to use an FEC to accurately identify equids with a high worm burden. However, clinical signs of 'intestinal parasitism', such as a poor coat, colic signs or adult worms seen in faeces, can also give an indication of the severely affected individuals.

Working equids in less developed countries do not generally receive regular anthelmintic treatments. This is likely to be due to the prohibitive costs, or a lack of access to veterinary medicines. There are very few studies investigating anthelmintic resistance in working equids (Kyvsgaarda et al. 2001). Resistance overall is likely to be low, and it is essential that the pool of sensitive helminths is preserved by using targeted treatments combined with improved management practices. Minimising the dependence on anthelmintic drugs will help prevent the development of resistance.

De-worming strategies

Programme	Dosing strategy	Comments
Targeted dosing	Regular FEC for entire herd De-worm all animals with an FEC above a set level (e.g. 1000 epg). FEC will not detect tapeworm. Perform a tapeworm faecal analysis or serology twice yearly. Alternatively, include a tapeworm treatment in the regime twice a year (interval dosing). Dose all tapeworm positive animals with pyrantel/praziquantel.	Appropriate if grazing is well managed, with predominantly adult horses and minimal new intake of animals (treated/isolated prior to introduction) Compliant owner to undertake frequent sampling Will detect high burden equids, those with a low burden will not require de-worming.
Strategic dosing	Dose only at times of year when de-worming will be most effective, e.g. with seasonal changes. Dose all grazing animals at the same time.	Regional variation in climate affects the time taken for the life cycle of parasites. Appropriate in countries/regions which have marked seasons
Interval dosing	Year-round synchronised dosing in all grazing animals. Dosing with principle anthelmintic at pre-determined intervals, based on ERP for each drug class.	Not recommended. Intensive use of anthelmintic predisposes development of resistance. It does not leave any refugia.

Table 17.3.1 Different de-worming dosing strategies.

17.4 Faecal egg count

The McMaster technique is a widely accepted protocol for the detection of nematode eggs in equine faeces. It is described below. An alternative technique is the FECPAK system (Presland et al. 2005). Here a larger faecal sample is taken giving greater sensitivity with low egg densities. Tapeworm egg counts are likely to be unreliable in both cases.

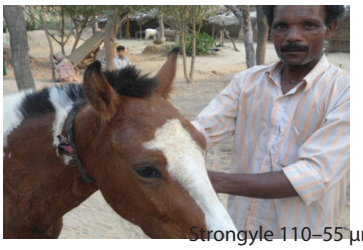
Materials

- McMaster slide (or alternative counting chamber of known volume)
- Microscope
- Clean containers with lids (such as jars)
- Wooden spatula to weigh 3 g samples
- Small sieve (such as a tea strainer or a gauze pad)
- Plastic pipettes
- Super-saturated salt (NaCl) solution:
  - Fill container with water.
  - Add table salt and shake/stir until the solution is saturated (salt settles at the bottom and will not dissolve).
  - Epsom salts, magnesium sulphate, can also be used.
- Weighing machine/scales in grams

  – or make a measuring device:
  - Draw up 42 ml water into a syringe.
  - Add the water to a jar and draw a line at the water level.
  - Add 3 ml water and draw a second line to mark 45 ml.

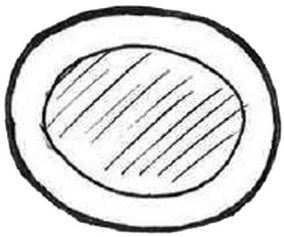
FEC procedure

1. Collect faecal samples from the target population/animal and label appropriately. Avoid collecting samples directly from the rectum.
2. Use fresh samples if possible and ensure air is taken out of the sample bag to stop development of eggs. Keep samples refrigerated and examine within 24 hours.
3. Weigh 3 g faeces from each sample and place in separate containers OR add faeces to 42 ml salt solution until the water level reaches the second line.
4. Mix the sample, by shaking the jar and breaking the faeces up with the spatula until they are evenly distributed throughout the water.
5. Using the sieve, strain the mixed faecal sample into another clean container.
6. Using the pipette, fill the McMaster counting chambers immediately.
7. Allow to sit for 1 minute, so that eggs float to the top, before reading.
8. Count the number of eggs (Figure 17.4.1) in each chamber and add together.



Strongyle 110–55 µm

Oxyuris equi 90x45 µm



Parascaris 100 µm

Figure 17.4.1 Commonly encountered helminth eggs. Note the distinctive operculum in the Oxyuris egg.

Larval culture to speciate strongyle eggs

It is possible to differentiate large and small strongyles by larval hatching from the faeces. In practice, if there are a large number of strongyle eggs, treatment without a precise diagnosis is acceptable.

To calculate the eggs per gram (epg), when using 3 g of faeces in 42 ml of salt solution read in a standard McMaster chamber (0.3 ml when both chambers counted), multiply the egg count by 50 (e.g. if there were four eggs the total count is 200 epg). Calculate the FEC for strongyles and ascarids separately. The strongyle count will include that for large and small strongyles as these eggs cannot be distinguished.

> There are four categories into which strongyle epg can be classified according to Soulsby 1982: none, low (up to 500 epg), medium (501–1,000 epg) or high (> 1,000 epg).

There is little official information on the egg counts of working equids and the level of parasite burden that causes problems for them. A study in working equids in Mexico revealed that there was no correlation between epg and body condition score (Valdez-cruz et al. 2013). The use of an epg 'cut off point' for anthelmintic treatment is likely to depend on local epidemiology and individual animal circumstances. Treat equids with an epg of > 1000 as they are likely to be high shedders and responsible for pasture contamination. If the epg is medium, clinical discretion is required.

Design field trials within an operational area to gain a broader understanding of the worm burdens in local equids, build a local databank of baseline information prior to de-worming.

Measuring anthelmintic resistance

In those animals that have been de-wormed (based on an medium to high epg) repeat the FEC 10–14 days after worming to test the efficacy of the anthelmintic. This is known as a Faecal Egg Count Reduction (FECR) test and is a technique for testing resistance. The FECR should be used in conjunction with the Egg Reappearance Period (ERP) to determine resistance levels effectively. ERP may be a more sensitive early indicator of resistance.

$$\text{FECR (\%)} = \frac{(\text{Day 0 FEC} - \text{Day 14 FEC}) \times 100}{\text{Day 0 FEC}}$$

FECR of > 95% for macrocyclic lactones and > 90% for benzimidazoles/pyrantel is expected for appropriate efficacy (Kaplan and Nielsen 2010).

Egg reappearance period

ERP is the time taken for worm eggs to reappear in the faeces after de-worming. This time period reduces as resistance starts to develop.

1. Determine egg counts in faecal samples collected from six or more equids prior de-worming.
2. Treat equids with label dosage of anthelmintic. Use a weigh tape and treat at 110% estimated weight.
3. Collect faecal samples from the same horses at 2-week intervals after de-worming, until 2 weeks after the recorded ERP for that anthelmintic (6 weeks for fenbendazole and pyrantel, 8 weeks for ivermectin and 13 weeks for moxidectin).

The ERP is usually defined as the time interval from treatment until the mean FEC exceeds a value of 20% of the pre-treatment FEC (Kyvsgaard et al. 2011).

17.5 External parasite species

Arthropod parasites

Lice

Two types of lice affect equids: the biting louse Damalinia equi and the sucking louse Haematopinus asini. Both are common where equids are kept in dirty or crowded conditions without regular grooming. Lice can be transmitted by direct contact or via saddlery or grooming kits.

Clinical signs

- Itching and hair loss varies from mild to severe.
- Adult lice are small and greyish-yellow. They can be hard to find but may be seen moving in the dust and scale on the skin surface, especially over the neck and rump/flanks.
- The eggs are pale yellow and stick to the ends of the hair, especially on the neck, upper limbs, and the base of the mane and tail.

Treatment

Wash the equid with a treatment containing a pyrethroid. Repeat treatment 3 times, once every 7–10 days. Treat all in-contact animals at the same time. Wash saddlery and grooming kits to prevent re-infestation.

Mites

Several species of mange mite affect equids: Chorioptes spp., Psoroptes spp., Demodex spp. and Sarcoptes scabei. Infestation is by direct contact with an infected equid or through saddlery and grooming kits. Mites burrow into the skin so, unlike lice, they cannot be seen with the naked eye. Skin scrapings (described in Section 15.2) or a skin biopsy is recommended for definitive diagnosis. Mites can be difficult to find, and the extent of the skin damage is not always representative of the numbers present. Itching is often severe causing the animal to rub, bite at itself or stamp its feet. Lesions are typified by hair loss and thickened, scaly, greasy skin. Debility, weakness and poor appetite may occur in severe cases.

Species	Predilection site	Pruritus	Diagnosis	Treatment
Sarcoptes	Severe lesions starting on head neck and shoulders	Intense pruritus	Skin scrape or biopsy	Pyrethroid wash or oral ivermectin. Repeat doses/ treatments at 2-week intervals for at least 3 treatments. Wash saddle and grooming equipment and treat all in-contact animals.
Psoroptes	Thickly-haired regions and ears	Pruritus	Skin scrape	Treatment as for sarcoptes
Chorioptes	Distal limbs	Pruritus, foot stamping	Skin scrape	Clip hair, treat with whole body wash with pyrethroids.
Demodex	Rare in equids, body or eyes and muzzle affected	Pruritus absent	Skin scrape	No effective treatment. Do not use amitraz as this medication can cause severe colic.

Table 17.5.1 Diagnosis and treatment of mites.

Ticks

Ticks are more common in the wet season. Predilection sites are generally protected areas such as the axilla, groin and ears (Figure 17.5.1). Tick infestations are normally mild and produce few direct clinical problems, but in many regions they are vectors for protozoal disease (see Section 17.7). Anaemia and debility only occur in very severe infestations. Remove ticks using blunt forceps (Gammons and Salam 2002).

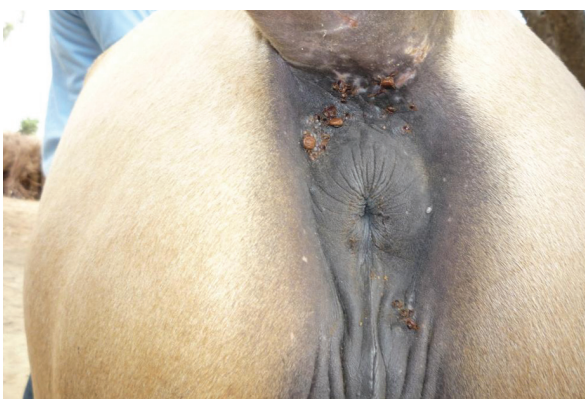


Figure 17.5.1 Ticks in a typical area under the base of the tail.

Helminth parasites

Onchocerca

Microfilariae of the nematode Onchocerca cervicalis are spread between equids by biting flies.

Life cycle

Adults live in long tendons, particularly the ligamentum nuchae in the neck. Inflammation is induced at these sites forming granulomatous, mineralised nodules. Onchocerca may be a factor in the development of fistulous withers (Doumbia 2011). Patent females produce microfilaria that migrate in the subcutaneous tissue to the ventral midline as well as other sites including the eye. At these locations the microfilariae initiate an inflammatory reaction which stimulates rubbing and self-trauma to these sites. Flies feed at the sites of inflammation and trauma and ingest the microfilaria. Microfilaria develop into infective stages in the fly. They are passed onto the next host when the fly feeds again.

Clinical signs

Although ocular and cutaneous lesions are seen, infection with Onchocerca spp. can be asymptomatic in up to 80% of cases.

Cutaneous lesions

Diffuse patchy alopecia (hair loss), erythema (redness), ulceration and scaling of the skin (Figure 17.5.2). Common sites include the ventral midline, face, base of mane, and a 'bull's-eye lesion' on the centre of the forehead.

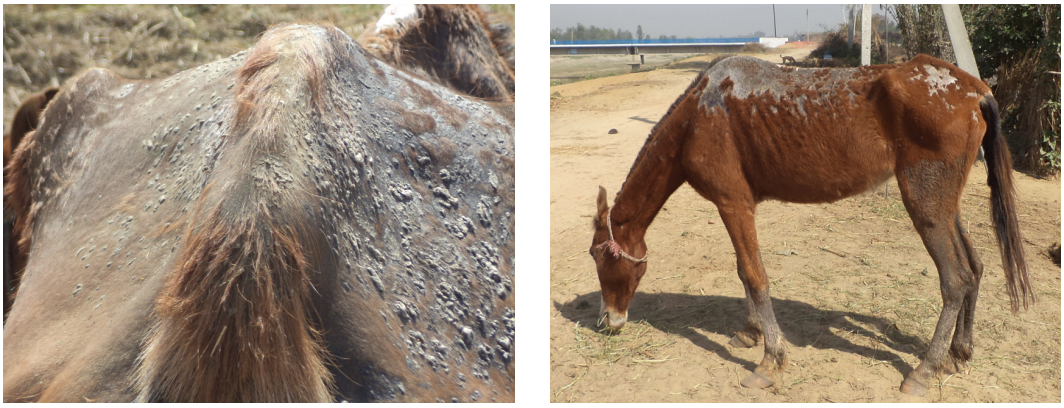


Figure 17.5.2 Clinical signs of onchocerciasis, a scaly dermatitis and patchy alopecia with an unusual presentation on the dorsal midline

Ocular lesions

Conjunctivitis and uveitis occur as an inflammatory response to dying microfilariae. Inflammation at the junction between the cornea and bulbar conjunctiva is a common clinical sign as well as de-pigmentation and inflammation at the lateral limbus of the eye. Onchocerca cervicalis has been implicated in the development of recurrent equine uveitis; however, this association is unclear (Moran and James 1987). Onchocerca volvulus causes ocular lesions known as 'river blindness' in humans. Use fluorescein dye to check that there is no corneal ulceration.

Diagnosis

The most effective method of diagnosis is by skin biopsy, preferably a full-thickness biopsy ≥ 6 mm. Mince the tissue and incubate in isotonic saline for several hours. Centrifuge the supernatant to concentrate the microfilariae. Stain with methylene blue and examine microscopically (Figure 17.5.3).

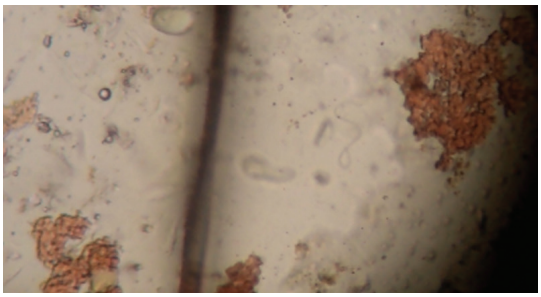


Figure 17.5.3 Microscopic view of a microfilariae. (Taken as a still from a video in which the microfilariae were very active.)

Treatment

Administer topical corticosteroids and systemic NSAIDs. Initiate this treatment 2 days prior to the ivermectin treatment and continue for a few days following. Dead microfilariae stimulate a greater immune reaction and damage than live ones. Administer systemic ivermectin (0.2 mg/kg PO). Ivermectin is not effective against the adult parasites; therefore, repeat treatment may be required after 2–4 months. Educate owners as to the importance of fly control to reduce infection rates and transmission.

Habronemiasis

Habronema spp. are small nematodes which parasitise the stomach and the skin and also cause ocular lesions (See Section 9.6 Common eye diseases of working equids). Flies, intermediate hosts of Habronema species, deposit infective larvae around the mouth. These are then swallowed by the equid. Larvae develop into adults in the stomach. Eggs are shed in the faeces and ingested by fly larvae. Habronema larvae then develop into the infective stages in the flies ready for onward transmission. The ocular and cutaneous forms develop when larvae are deposited into open wounds on the skin (summer sores) or around the eye. In these cases the larvae penetrate the dermis resulting in a hypersensitivity reaction. The Habronema larvae cannot complete development in the skin.

Clinical signs

Habronemiasis is most commonly seen in warmer climates and seasons. Lesions are nodular and ulcerated. In the ocular form, lesions are commonly seen at the medial canthus. Other ocular sites include the conjunctival sac, the lacrimal duct and the third eyelid. Yellow caseous ocular discharge contains necrotic material which encases the infective larvae. Severe ocular problems can result as sequalae, including epiphora, chemosis, and photophobia. Habronema spp. can cause a catarrhal gastritis but it is generally not considered clinically significant in this organ.

Diagnosis

Clinical signs and history can provide an accurate diagnosis. Cytology on a deep skin scraping can reveal larvae.

Treatment

Systemic treatment (PO) with ivermectin (see Section 9.6). NSAIDs and, occasionally, steroids are required to treat hypersensitivity reactions associated with the lesions. Raise awareness of the life cycle of Habronema amongst equine owners. Highlight the importance of fly control and hygiene as a preventative measure. Clean eyes regularly and cover wounds.

Myiasis (fly strike)

Flies deposit eggs around wounds or infected areas, the larvae (maggots) hatch and burrow into the tissue. Maggot infestation causes extensive inflammation, tissue damage and secondary infection.

Treatment

Remove as many maggots as possible using forceps. The animal may need to be sedated to allow deep exploration of the wound. Open any deep tracts using a scalpel blade, following administration of local anaesthesia. Apply 1–2 ml ivermectin or permethrin ectoparasiticide topically to kill any remaining maggots. Debride and clean the wound and allow to heal by secondary intention. Cover the wound to prevent further infestation and check it daily until healing is complete.

Prevention

Check all wounds daily and keep them bandaged or covered wherever possible. Keep feet clean, dry and free from infection. Use insect repellents.

17.6 Ectoparasite medication

In the treatment of ectoparasites, only when a diagnosis has been made can the optimal therapeutic protocol be chosen and a positive treatment outcome achieved. When attempting to control lice and mites, apply treatment to all members of a group rather than just to those showing reactions. Hypersensitivity to killed parasites can result in the persistence of pruritus; this may be perceived as treatment failure. Judicious use of anti-histamines, or corticosteroids, can be employed to limit pruritus.

Pyrethroids

Pyrethroids are neurotoxin insecticides. The pyrethroids – permethrin, cypermethrin, fenvalerate and deltamethrin – are available in spot-on, spray and wash formulations. These medications are effective against lice and mites, including Sarcoptes and Chorioptes. When using as a topical wash ensure that the entire coat is covered. This treatment should be repeated 3 times, 7 days apart. Powder formulations are less likely to provide effective coverage and tend to lose potency rapidly when stored.

Macrocyclic lactones

Parenteral and per os administration of ivermectin and moxidectin is generally only effective for ectoparasites that ingest blood or live within the skin, the sucking lice and skin helminths. These medications can also be used to treat Sarcoptes scabei in horses. Pour-on formulations used to treat cattle are not licensed for use in equids.

Benzyl benzoate

Benzyl benzoate is often more affordable and available than permethrin products and is used as a topical treatment for lice and Sarcoptes scabei. There are reports of variable efficacy but this medication is used frequently in the treatment of scabies in humans, particularly in less developed countries (Mounsey and McCarthy 2013). Apply to the entire coat. Rinse the coat after 24 hours as prolonged skin contact can cause irritation.

Fipronil is used to treat fleas in small animals. Fipronil is not licensed in horses but is effective against mange mites in the spray form; however, the large volume required to treat equids is prohibitively expensive.

> Organophosphates are no longer used as for ectoparasite treatment in many countries as there is a high risk of poisoning for both the animals and owners.

Synthetic pyrethroids are significantly less toxic and have been used in preference. Organophosphates may still be available in some regions; use is not recommended.

Insect growth regulators are in development for veterinary use (Pasay et al. 2012).

Protozoal infections 17.7

Equine piroplasmosis

Equine piroplasmosis (EP) is a tick-borne infection caused by the haemoprotozoa Babesia caballi and Theileria equi. This disease affects all members of the Equus genus, including donkeys and mules (Gizachew et al. 2013). B. caballi and T. equi are endemic in many tropical and subtropical countries in Mediterranean Europe, Africa, the Middle East, Asia, and Central and South America. Equine piroplasmosis is a notifiable disease in many countries and is reportable to the OIE by government veterinary services.

Twelve species of ixodid ticks of the genera Dermacentor spp. (central Asia), Hyalomma spp. (Middle East and Africa), and Rhipicephalus spp. (Africa and South America) were listed as vectors for EP by the OIE. T. equi is also transmitted by contaminated needles and syringes. Although identification of EP tick vectors is possible, it is impractical as a diagnostic tool. As the incubation period can be up to 30 days for B. caballi and 20 days for T. equi, the infected tick is likely to have dropped off the host prior to the development of clinical signs of disease. Equids presenting with a tick infestation and concurrent clinical signs of anaemia and weakness should be tested for EP to confirm the suspected diagnosis.

The causative agents of EP are intracellular parasites which invade red blood cells inducing haemolysis, thereby causing a haemolytic anaemia. The severity of clinical signs relates to the parasite burden, and disease can manifest as acute or chronic, mild to severe. Fatalities may occur in the first 48 hours of infection but chronic disease often develops. Generally B. caballi is clinically milder than T. equi; severe anaemia is very rare with a B. caballi infection. Donkeys

tend to develop the chronic form of the disease and the signs are often unspecific. Carrier equids remain sources of infection for tick vectors for up to 4 years after infection.

Clinical signs of acute EP

- Intermittent fever (> 40˚C), sudden sweating
- Anaemia, icterus
- Petechial haemorrhage of third eyelid (Figure 17.8.1)
- High heart rate and respiratory rate
- Oedema of muzzle, limbs, ventral abdomen and thorax
- Hindlimb weakness, reluctance to move, tremors
- Haemoglobinuria and dry faeces
- May lead to death within a few days (mortality 5–10% in endemic areas and up to 50% in naïve horses)

Clinical signs of chronic EP

- Inappetance, weight loss
- Poor performance
- Difficult to detect parasites in blood

Diagnosis

The number of parasites in the blood varies throughout the course of infection. In acute cases with clinical signs the haemoparasites are readily visible on a blood film, appearing as dark dots, rings or pear-shaped marks within red blood cells. It may be difficult to detect parasites in animals in the latent or chronic stages of disease, particularly with B. caballi. Laboratory techniques include PCR and antibody ELISA (Rosales et al. 2013).

Treatment

The medications used to treat EP are toxic, so administer treatment only to animals with severe clinical signs or if parasites are present in > 50% of RBCs. Provide supportive treatment such as fluid therapy, NSAIDs, and a blood transfusion (if the facilities are available and the PCV is < 12%).

Imidocarb diproprionate Efficacy of this medication is variable (Grause et al. 2012), and toxic side effects are common. Administration can cause severe colic and diarrhoea; these effects may be reduced by pre-medicating with glycopyrrolate (Kutscha et al. 2012). Administer 2.4 mg/kg as a single deep IM injection; donkeys are more susceptible to toxic side effects so use a low dose (1–2 mg/kg). A treatment protocol of 4 injections at 4 mg/kg at 72-hour intervals has been recommended for complete elimination of T. equi (Grause et al. 2012); in endemic areas complete parasite elimination will reduce endemic stability.

Diminazine aceturate It is recommended to avoid this drug in equids unless other drugs are unavailable, as it has a low therapeutic index and toxic side effects are common. Administration of 3.5 mg/kg IM reduces the clinical signs within 24 hours. (In areas of resistance, horses require even higher doses; 2 doses 24 hours apart at 5 mg/kg for B. caballi, 6–12 mg/kg for T. equi.) However, this drug is associated with marked side effects even at the lower dose rate. At higher doses, the risk and severity of such side effects would be increased and is not recommended. Toxicity can be treated with calcium salts.

Endemic stability

'Infected but not affected'

Endemic stability is an epidemiological state in which severe clinical disease is scarce despite high levels of infection in the population. In endemic areas where equids are faced with a low level of continuous challenge, immunity develops and reduces the severity of disease. Disruption of this low-level challenge, through control (of ticks or the causative agents of EP), might result in an increase in clinical disease incidence. Under conditions of epidemiological stability, when hosts are under frequent exposure to EP, foals are protected passively by maternal antibodies acquired via colostrum. This protection can last for up to 9 months. Infections of foals in this period will induce immunity without any overt signs of the disease.

Trypanosomiasis

The causative organisms of trypanosomiasis in equids are the tsetse transmitted Trypanosoma spp. (which cause the disease known as nagana in cattle), T. evansi (surra) and T. equiperdum (dourine). These flagellate haemoprotozoan live extracellularly in the blood. The different species have differing life cycles and transmission mechanisms.

Tsetse transmitted trypanosomiasis

Tsetse flies are distributed in a 'belt' across 37 countries in Africa; trypanosomiasis is endemic in these areas. For T. congolense and T. brucei, development within the tsetse is an essential stage in the life cycle. All domestic animals are at risk of infection in tsetse areas.
T. brucei gambiense and rhodesiense cause disease in humans resulting in a fatal condition known as sleeping sickness. Horses cannot be kept in tsetse areas without trypanocidal prophylaxis; however, donkeys have been kept in these areas. Although resilient, donkeys are not resistant to tsetse transmitted trypanosomiasis (Faye et al. 2001, Mukiria et al. 2010).

T. brucei Horses are particularly susceptible to this species of trypanosome and mortality usually occurs within 14–90 days if left untreated. Clinical signs include anaemia, icterus (jaundice), enlarged lymph nodes and petechial haemorrhages of the mucous membranes. Classic parasitaemic 'waves' of intermittent high fever (41°C) occur as host immunity responds to changes in the proteins on the parasite surface. T. brucei can cause a serious and acute disease in donkeys as well as horses.

T. congolense and T. vivax These are rarer with relatively milder and more chronic clinical signs compared to T. brucei, often indicated by anorexia, wasting and generalised oedema around 14 days after infection. T. vivax can also be transmitted mechanically, and therefore can spread beyond the tsetse belt as well as occurring in South America.

Surra

The causative agent of surra, Trypanosoma evansi, is transmitted mechanically by haematophagus flies. The insects which spread the disease are various types of Tabanids (horse flies) and Stomoxys (stable flies). The trypanosome parasites are mechanically transferred during feeding; there is no biological development of the trypanosome parasite within the fly. Surra is a particular problem in India, but also affects equids across Asia, North Africa, Central and South America.

The clinical signs of surra are severe weight loss (Figure 17.7.1), progressive weakness, anaemia, haemoglobinuria, intermittent fever, petechial haemorrhage of mucous membranes, oedema of limbs, lower abdomen and thorax and severe neurological signs. The mortality rate in untreated horses is almost 100%. The severity of the disease depends on the parasite strain and factors including stress and the health of the equid. Animals subjected to stress, such as malnutrition and physical labour, are more susceptible to the disease.

Chronic forms persist for several months up to 2 years, providing a reservoir of infection for other animals. There is considerable variation in host species susceptibility; severe rapidly fatal disease is common in horses, whereas donkeys and mules tend to develop chronic mild or subclinical infections.

There are rare reports of T. evansi infection in humans, these are thought to be anomalies as the individuals were immuno-compromised (Joshi et al. 2005; Powar et al. 2006).

Figure 17.7.1 A horse with severe weight loss at an equine fair in India, subsequently diagnosed with a surra infection.

Prevention of surra

Control measures include early detection and treatment of infected animals and protection of all animals from biting flies. The aim is to alter attitudes (Figure 17.7.2) so that individual animals, preferably with a laboratory diagnosis, are treated with the medication at an appropriate dose. Treatment of groups of animals for a long period at sub-optimal doses is discouraged.

- Isolate infected animals by moving them away from the rest of the herd (biting flies are then less likely to transfer the disease).
- Keep other livestock, which act as a reservoir, separate from equids.
- Ensure vector control, fly repellents, dung removal, and do not house equids close to stagnant water.

Figure 17.7.2 Developing a health plan to reduce surra within a community in India.

Prophylactic treatment is not recommended. Although in the short term this may reduce the number of clinical cases, it will ultimately lead to resistance. There are very few alternative drugs to treat surra and, if resistance develops, then there will be no effective treatment.

Dourine

Dourine is a venereal disease of equids caused by T. equiperdum and transmitted mechanically when mating. T. equiperdum is the only trypanosome that is not transmitted by an invertebrate vector. The pathogenesis of dourine differs from other trypanosomes in that it is primarily a tissue parasite which rarely invades the blood. There is no known natural reservoir of the parasite other than infected equids (Claes et al. 2005). Dourine was previously widespread; it is now limited to confined parts of Africa, Asia, Central and South America.

> T. equiperdum may persist for years in donkeys and mules without showing clinical signs; mortality can be high in untreated horses.

A mucopurulent discharge from the penis or vulva, and oedema around the area, is the most common initial presenting sign. Generalised clinical signs can include fever, oedema, anaemia and wasting. Approximately a month following inoculation, urticarial reactions erupt all over the body. Progressive paralysis is a possible sequel and these cases are frequently fatal. Abortion is also common.

Diagnosis of trypanosomiasis

A clinical diagnosis of trypanosomiasis must be confirmed by a laboratory test; however, the standard techniques for the detection of trypanosomes are not sufficiently sensitive. The infection is not always detected by microscopic examination of the blood; the trypanosomes must be at a high level to be detected by this method, and in chronic or latent infections the number of parasites will be low. The detection rate can be improved by centrifuging the blood sample to concentrate the parasites. Serologic tests (CFT and ELISA) and trypanosome antigen detecting tests (PCR) have been developed. Diagnosis is challenging as clinical signs are similar to other diseases, such as babesiosis, equine infectious anaemia and African Horse Sickness.

Collect blood samples from capillaries using an ear prick. Examine as a wet smear or as a thick or thin stained smear (Figure 17.8.2). Alternatively centrifuge the sample in a capillary tube and look at the tube under the microscope. The organisms are flagellate-protozoa free in plasma, moving with a whip-like motion just above the buffy coat. This is a relatively simple and efficient technique (75% sensitive). However, the fresh blood sample (EDTA or lithium heparin tube) must be examined within 7–12 hours of collection. Parasitaemia is intermittent, so it is good practice to take a number of samples at 4-day intervals. In chronic infections parasites in the blood are very rarely found, making diagnosis difficult. In these cases clinical signs, the presence of anaemia and acanthocytes, can be useful in diagnosis.

T. equiperdum rarely produces haemoparasitism so blood smear detection is not usually effective. Microscopy of direct smears from the fluid of infected genitalia may detect parasites; however, serology is the most reliable test for dourine. When using the CFT, cross reactions with T. brucei are common so this test is not applicable in tsetse zones.

Treatment of trypanosomiasis

As the result of overuse of medication in cases of misdiagnosis and prophylaxis, there is widespread resistance to trypanocides. In addition to concerns regarding resistance, these drugs have narrow safety margins. The availability of trypanocidal medication and resistance patterns will vary according to geographical area. Attempts to develop a vaccine have been thwarted as the parasite is able to override immune defences by rapidly changing the surface proteins. Each time an antibody response is mounted, the coat changes and the defences are useless.

Treatment is not recommended for dourine as it is never 100% effective; recovered animals can still infect others. Castration may be an option in males; however, euthanasia should be discussed with the owner. It is important to reiterate that infected animals should not be used for breeding.

Quinapyramine salts For infections of T. evansi use 3–5 mg/kg of a 5% solution split between three injection sites. Quinapyramine dimethylsulphate is recommended as a treatment whereas quinapyramine chloride is thought to have a longer duration of action (prophylaxis is not recommended). There are formulations where these drugs are delivered together (Triquin) with the aim to provide a depot at the injection site.

These medications are often poorly tolerated (particularly in horses) and now have widespread resistance. Only administer via deep IM injection using a long narrow-gauge needle, as SC injection results in sloughing and may take many months to resolve.

Phenanthridinium compounds

Homidium bromide Purple tablets (250 mg) dissolved in sterile water, prepared as 1–2.5% solution for SC or IM injection at 1 mg/kg. With both prophylactic and curative properties, this is most effective against T. vivax; however, there is widespread resistance in tsetse areas. Isometamidium is structurally similar to homidium, therefore there is the potential for cross-resistance with these drugs.

Pyrithidium bromide Red tablets (500 mg) dissolved in boiling water to make a 2.5% solution. Give via deep IM injection, 2–2.5 mg/kg will provide 4 months' protection in non-resistant areas. Severe local reactions can occur and resistance is common.

Isometamidium chloride The current recommended dose rates are T. vivax (0.5 mg/kg IM), and T. brucei, T. congolensis (0.5–1mg/kg). Administer a 1–2% solution as a deep IM injection. Resistance is common in areas where this drug is widely used for prophylaxis.

Diminazene aceturate This compound was discussed previously with reference to EP; however, in equids at therapeutic doses against trypanosomiasis, there are severe side effects which can be fatal so do not use!

Suramin This is considered the drug of choice for the early stages of Human African Trypanosomiasis. In domestic animals it has predominantly been used as a curative drug against T. evansi in camels and horses.

The standard prophylactic dose in horses is 7–10 mg/kg IV. Complexes of suramin with quinapyramine have found to be effective prophylactically but they are expensive and have severe tissue reactions. The safety margins of suramin are low and horses/donkeys are considered more susceptible than camels.

Melarsenoxide Although not widely available, this is used for treatment of T. evansi infections in camels and horses. Efficacy is also reported against T. brucei in horses. Melarsomine has no prophylactic activity. The dose rate is 0.25 mg/kg IM/SC. The introduction of this compound has been seen as a clinical breakthrough in the treatment of T. evansi in camels. Unlike other trypanocidal drugs, the therapeutic index of this drug is high. Melarsenoxide crosses the blood-brain barrier.

Resistance

The development of resistance to trypanocidal drugs is a growing concern. It has been speculated that drug resistance in trypanosomes is likely to occur under the same circumstances as for many other parasites as a result of the following factors:

- Large scale drug use as (preventive treatments)
- Treatment with inadequate dose
- Use of medication that is slowly eliminated from the body
- The phenomenon of cross-resistance has been well established. For instance, quinapyramine usage has been shown to induce resistance to isometamidium, homidium and diminazene

Equine protozoal myoencephalitis

See Chapter 16 Disorders of the neurological system.

Case study – Surra 17.8

Area India

Attending veterinarians Dr Nidhish Bhardwaj and Dr Kamlesh

History
A working horse was presented with a 4-day history of anorexia, dullness and depression.

Clinical findings
The temperature was high (38.8°C). Other vital parameters were also elevated. Petechial haemorrhages were seen on third eyelid and mucous membrane were pale (Figure 17.8.1). Oedema of the lower limbs was observed and the animal was walking with a stiff gait.

Diagnosis
A blood smear was prepared and examined in the laboratory for confirmation (Figure 17.8.2). This case was diagnosed as surra, based on the symptoms and laboratory findings.

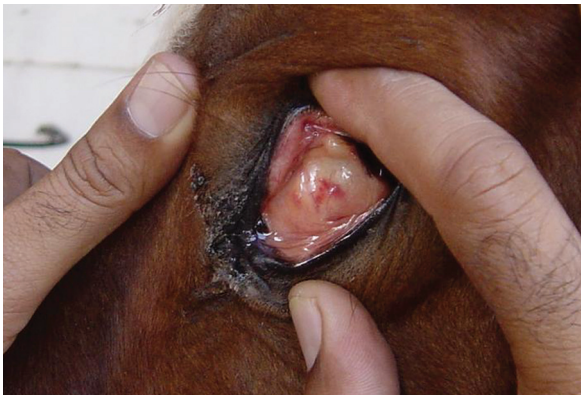


Figure 17.8.1 Petechial haemorrhage of the third eyelid.

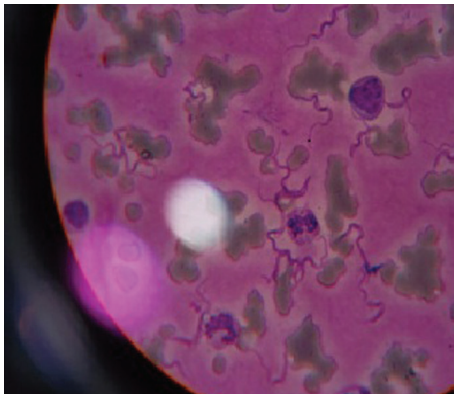


Figure 17.8.2 Trypanosomes in a blood smear.

Treatment

- Ketoprofen 2.2 mg/kg (NSAID)
- Pheniramine maleate 0.25 mg/kg (anti-histamine)
- Fluid therapy
- Quinapyramine sulphate 3 mg/kg

Outcome
The animal was showing signs of recovery after 2 days.

Discussion
Prevention is the best policy to control surra in an endemic area. Continuous owner education, generating awareness of the necessity of fly control, and identification of early signs of surra can be a key in managing this disease.

17.9 References

Andersen, U.V., Howe, D.K., Dangoudoubiyam, S., Toft, N., Reinemeyer, C.R., Lyons, E.T., Olsen, S.N., Monrad, J., Nejsum, P., Nielsen, M.K. (2013) SvSXP: a Strongylus vulgaris antigen with potential for prepatent diagnosis. Parasites Vector. 4 (6) 84.

Binev, R., Kirkova, Z., Nikolov, J., Russenov, A., Stojanchev, K., Lazarov, L., Hristov, T. (2005) Efficacy of parenteral administration of ivermectin in the control of strongylidosis in donkeys. J. S. Afr. Vet. Assoc. 76 (4) 214–216.

Claes, F., Büscher, P., Touratier, L., Goddeeris, B.M. (2005) Trypanosoma equiperdum: master of disguise or historical mistake? Trends Parasitol. 21 (7) 316–321.

Clayton, H.M., Neave, R.M.(1979) Efficacy of mebendazole against Dictyocaulus arnfieldi in the donkey. Vet. Rec. 104 (25) 571–572.

Doumbia, A. (2011) Onchocerciasis in working donkeys in Mali, Africa. 12th Congress of the World Equine Veterinary Association, Hyderabad, India.

Duncan, J.L., Bairden, K., Abbott, E.M. (1998) Elimination of mucosal cyathostome larvae by five daily treatments with fenbendazole.Vet. Rec. 142 (11) 268–271.

EMEA European Agency for the Evaluation of Medicinal Products (1999) Piperazine Summary Report. Available online at: http://www.ema.europa.eu/docs/en_GB/document_library/Maximum_Residue_Limits_-_Report/2009/11/WC500015672.pdf

Eysker, M., Boersema, J.H., Kooyman, F.N. (1992) The effect of ivermectin treatment against inhibited early third stage, late third stage and fourth stage larvae and adult stages of the cyathostomes in Shetland ponies and spontaneous expulsion of these helminths. Vet. Parasitol. 42 (3–4) 295–302.

Eysker, M., Pandey, V.S. (1989) Small Strongyle Infections in Donkeys from the Highveld in Zimbabwe. Vet. Parasitol. 30 (4) 345–349.

Faye, D., Pereira de Almeida, P.J., Goossens, B., Osaer, S., Ndao, M., Berkvens, D., Speybroeck, N., Nieberding, F., Geerts, S. (2001) Prevalence and incidence of trypanosomosis in horses and donkeys in the Gambia. Vet. Parasitol. 101 (2) 101–114.

French, D.D., Torbert, B.J., Chapman, M.R., Klei, T.R., Pierce, M.S. (1983) Comparison of anti-strongyle activity of a micellar formulation of ivermectin given parenterally or per os. Vet. Med. Sm. Anim. Clin. 78, 1778–1780.

Gammons, M., Salam, G. (2002) Tick Removal. Am. Fam. Physician. 4 (66) 643–645.

Getachew, A.M., Innocent, G., Proudman, C.J., Trawford, A., Feseha, G., Reid, S.W., Faith, B., Love, S. (2013) Field efficacy of praziquantel oral paste against naturally acquired equine cestodes in Ethiopia. Parasitol Res. 112 (1) 141–146.

Getachew, A.M., Innocent, G., Trawford, A.F., Reid, S.W.J., Love, S. (2012) Gasterophilosis: a major cause of rectal prolapse in working donkeys in Ethiopia. Trop. Anim. Health Pro. 44 (4) 757–762.

Getachew, M., Innocent, G.T., Trawford, A.F., Reid, S.W., Love, S. (2010a) Epidemiological features of fasciolosis in working donkeys in Ethiopia. Vet. Parasitol. 169 (3–4) 335–339.

Getachew, M., Trawford, A., Feseha, G., Reid, S.W. (2010b) Gastrointestinal parasites of working donkeys of Ethiopia. Trop. Anim. Health Pro. 42 (1) 27–33.

Gizachew, A., Schuster, R.K., Joseph, S., Nissy, R.W., Georgy, A., Elizabeth, S.K., Asfaw, Y., Regassam, F., Wernery, U. (2013) Piroplasmosis in Donkeys—A Hematological and Serological Study in Central Ethiopia. J. Equine Vet. Sci. 33 (1) 18–21.

Gokbulut, C., Cirak, V.Y., Senlik, B., Aksit, D., Durmaz, M., McKellar, Q.A. (2010) Comparative plasma disposition, bioavailability and efficacy of ivermectin following oral and pour-on administrations in horses. Vet. Parasitol. 170 (1–2) 120–126.

Grause, J.F., Ueti, M.W., Nelson, J.T., Knowles, D.P., Kappmeyer, L.S., Bunn, T.O. (2012) Efficacy of imidocarb dipropionate in eliminating Theileria equi from experimentally infected horses. Vet. J. 196 (3) 541–546.

Haridy, F.M., Morsy, T.A., Gawish, N.I., Antonios, T.N., Abdel Gawad, A.G. (2002) The potential reservoir role of donkeys and horses in zoonotic fascioliasis in Gharbia Governorate, Egypt. Journal of the Egyptian Society of Parasitology. 32 (2) 561–570.

Hearn, F.P., Peregrine, A.S. (2003) Identification of foals infected with Parascaris equorum apparently resistant to ivermectin. J. Am. Vet. Med. Assoc. 223 (4) 482–485.

Johnson, P.J., Mrad, D.R., Schwartz, A.J., Kellam, L. (1999) Presumed moxidectin toxicosis in three foals. J. Am. Vet. Med. Assoc. 214 (5) 678–680.

Joshi, P.P., Shegokar, V.R., Powar, R.M., Herder, S., Katti, R., Salkar, H.R., Dani. V.S., Bhargava, A., Jannin, J., Truc, P. (2006) A rare case of human trypanosomiasis caused by Trypanosoma evansi. Indian Journal of Medical Microbiology. 24 (1) 72–74.

Kaplan, R.M., Nielsen, M.K. (2010) Equine veterinary education An evidence-based approach to equine parasite control: It ain't the 60s anymore. Equine Vet. Educ. 22, 306–316.

Klei, T.R., Chapman, M., French, D.D. (1993) Evaluation of ivermectin at an elevated dose against encysted equine cyathostome larvae. Vet. Parasitol. 47, 99–106.

Kutscha, J., Sutton, D.G., Preston, T., Guthrie, A.J. (2012) Equine piroplasmosis treatment protocols: specific effect on orocaecal transit time as measured by the lactose 13C-ureide breath test. Equine Vet. J. Supplement. 44 (43) 62–67.

Kuzmina, T.A., Kharchenko, V.O. (2008) Anthelmintic resistance in cyathostomins of brood horses in Ukraine and influence of anthelmintic treatments on strongylid community structure. Vet. Parasitol. 154 (3–4) 277–288.

Kyvsgaard, N.C., Lindbom, J., Andreasen, L.L., Luna-Olivares, L.A., Nielsen, M.K., Monrad, J. (2011) Prevalence of strongyles and efficacy of fenbendazole and ivermectin in working horses in El Sauce, Nicaragua. Vet. Parasitol. 181 (2–4) 248–254.

Leaning, W.H.D. (1983) The efficacy and safety evaluation of ivermectin as a parenteral and oral antiparasitic agent in horses. P. Am. Assoc. Equine Prac. 29, 319–328.

Lyons, E.T., Tolliver, S.C., Collins, S.S. (2011) Reduced activity of moxidectin and ivermectin on small strongyles in young horses on a farm (BC) in Central Kentucky in two field tests with notes on variable counts of eggs per gram of feces (EPGs). Parasitol Res. 108 (5) 1315–1319.

Marchiondo, A.A., White, G.W., Smith, L.L., Reinemeyer, C.R., Dascanio, J.J., Johnson, E.G., Shugart, J.I. (2006) Clinical field efficacy and safety of pyrantel pamoate paste (19.13% w/w pyrantel base) against Anoplocephala spp. in naturally infected horses. Vet. Parasitol. 137 (1–2) 94–102.

Meana, A., Luzon, M., Corchero, J., Gómez-Bautista, M. (1998) Reliability of coprological diagnosis of Anoplocephalaperfoliata infection. Vet. Parasitol. 74 (1) 79–83.

Molento, M.B., Nielsen, M.K., Kaplan, R.M. (2012) Resistance to avermectin/milbemycin anthelmintics in equine cyathostomins – Current situation. Vet. Parasitol. 185 (1) 16–24.

Monahan, C.M., Chapman, M.R., Taylor, H.W., French, D.D., Klei, T.R. (1996) Comparison of moxidectin oral gel and ivermectin oral paste against a spectrum of internal parasites of ponies with special attention to encysted cyathostome larvae. Vet. Parasitol. 63 (3–4) 225–235.

Moran, C.T., James, E.R. (1987) Equine ocular pathology ascribed to Onchocerca cervicalis infection: a re-examination. Ann. Trop. Med. Parasit. 38 (4) 287–288.

Mounsey, K.E., McCarthy, J.S. (2013) Treatment and control of scabies. Curr. Opin. Infect. Dis. 26 (2) 133–139.

Mukiria, P., Mdachi, R., Thuita, J., Mutuku, J., Wanjala, K., Omolo, J., Getachew, M., Trawford, A.F., Ouma, J., Murilla, G. (2010) Semi-longitudinal study of trypanosomiasis and its vectors in donkeys (Equus africanus asinus, fitzinger) in the Lamu archipelago. Presented at 12th KARI Biennial Scientific Conference. 8–12 November. Nairobi, Kenya.

Owen, J., Slocombe, D. (2004) A modified critical test for the efficacy of pyrantel pamoate for Anoplocephalaperfoliata in equids. Can. J. Vet. Res. 68 (2) 112–117.

Pasay, C., Rothwell, J., Mounsey, K., Kelly, A., Hutchinson, B., Miezler, A., McCarthy, J. (2012) An exploratory study to assess the activity of the acarine growth inhibitor, fluazuron, against Sarcoptes scabei infestation in pigs. Parasite Vector 5:40.

Powar, R.M., Shegokar, V.R., Joshi, P.P., Dani, V.S., Tankhiwale, N.S., Truc, P., Jannin, J., Bhargava, A. (2005) Human trypanosomiasis caused by Trypanosoma evansi in India: the first case report. Am J Trop Med Hyg. 73 (3) 491–495.

Presland, S.L., Morgan, E.R., Coles, G.C. (2005) Counting Nematode eggs in equine faecal samples. Vet. Rec. 156, 208–210.

Reed, S.M. (1983) Ivermectin and CNS signs. Mod. Vet. Pract. 64, 783–784.

Reinemeyer, C.R., Prado, J.C., Nichols, E.C., Marchiondo, A.A . (2010) Efficacy of pyrantel pamoate and ivermectin paste formulations against naturally acquired Oxyuris equi infections in horses. Vet. Parasitol. 171 (1–2) 106–110.

Reinemeyer, C.R. (2009) Diagnosis and control of anthelmintic-resistant Parascaris equorum. Parasite Vector. 25 (2) Suppl 2:S8.

Rosales, R., Rangel-Rivas, A., Escalona, A., Jordan, L.S., Gonzatti, M.I., Aso, P.M., Perrone, T., Silva-Iturriza, A., Mijares, A. (2013) Detection of Theileria equi and Babesia caballi infections in Venezuelan horses using Competitive-Inhibition ELISA and PCR. Vet. Parasitol. (In press).

Rossano, M.G., Smith, A.R., Lyons, E.T. (2010) Shortened strongyle-type egg reappearance periods in naturally infected horses treated with moxidectin and failure of a larvicidal dose of fenbendazole to reduce fecal egg counts. Vet. Parasitol. 173 (3–4) 349–352.

Rubilar, L., Cabreira, A., Giacaman, L. (1988) Treatment of Fasciola hepatica infection in horses with triclabendazole. Vet. Rec. 123 (12) 320–321.

Singh, G., Soodan, J.S., Singla, L.D., Khajuria, J.K. (2012) Epidemiological studies on gastrointestinal helminths in horses and mules. Vet. Pract. 13 (1) 23–27.

Skotarek, S.L., Colwell, D.D., Goater, C.P. (2010) Evaluation of diagnostic techniques for Anoplocephala perfoliata in horses from Alberta, Canada. Vet. Parasitol. 172 (3–4) 249–255.

Slocombe, J.O., Coté, J.F., de Gannes, R.V. (2008) The persistence of benzimidazole-resistant cyathostomes on horse farms in Ontario over 10 years and the effectiveness of ivermectin and moxidectin against these resistant strains. Can. Vet. J. 49 (1) 56–60.

Slocombe, J.O., Cote, J.F. (1977) Small strongyles of horses with cross resistance to benzimidazole anthelmintics and susceptibility to unrelated compounds. Can. Vet. J. 18 (8) 212–217.

Slocombe, J.O., Heine, J., Barutzki, D., Slacek, B. (2007) Clinical trials of efficacy of praziquantel horse paste 9% against tapeworms and its safety in horses. Vet. Parasitol. 144 (3–4) 366–370.

Soulsby, E.J.L. (1982). Helminth, Arthropod and Protozoa of Domestic Animals. 7th Ed. Baillere Tindall, London, UK. 809.

Steinbach, T., Bauer, C., Sasse, H., Baumgartner, W., Rey-Moreno, C.,Hermosilla, C., Damriyasa, I., Zahner, H. (2006) Small strongyle infection: Consequences of larvicidal treatment of horses with fenbendazole and moxidectin. Vet. Parasitol. 139 (1–3) 115–131.

Tavassoli, M., Dalir-Naghadeh, B., Esmaeili-Sani, S. (2010) Prevalence of gastrointestinal parasites in working horses. Pol. J. Vet. Sci. 13 (2) 319–324.

Traversa, D., von Samson-Himmelstjerna, G., Demeler, J., Milillo, P., Schürmann, S., Barnes, H., Otranto, D., Perrucci, S., di Regalbono, A.F., Beraldo, P., Boeckh, A., Cobb, R. (2009) Anthelmintic resistance in cyathostomin populations from horse yards in Italy, United Kingdom and Germany. Parasite Vector. 2 Suppl 2:S2.

Trawford, A.F., Tremlett, J.G. (1996) Efficacy of triclabendazole against Fasciola hepatica in the donkey (Equus asinus).Vet. Rec. 139 (6) 142–143.

Valdez-Cruz, M.P., Hernandez-Gil, M., Galindo-Rodriguez, L., Alonso-Diaz, M.A. (2006) Gastrointestinal parasite burden, body condition and haematological values in equines in the humid tropical areas of Mexico. The Fifth International Colloquium on Working Equines. Addis Adaba, Ethiopia. 30 October–2 November. 62–72.

Valdéz-Cruz, M.P., Hernández-Gil, M., Galindo-Rodríguez, L., Alonso-Díaz, M.A. (2013) Gastrointestinal nematode burden in working equids from humid tropical areas of central Veracruz, Mexico, and its relationship with body condition and haematological values. Trop. Anim. Health Pro. 45 (2) 603–607.

Further Reading

Bergvall, K. (2005) Advances in Acquisition, Identification, and Treatment of Equine Ectoparasites. Clin. Tech. Equine P. 4 (4) 296–301.

Brady, H.A., Nichols, W.T. (2009) Drug Resistance in Equine Parasites: An Emerging Global Problem. J. Equine Vet. Sci. 29 (5) 285–295.

Corning, S. (2009) Equine cyathostomins: a review of biology, clinical significance and therapy. Parasite Vector. 2(Suppl 2):S1

Crane, M.A., Khallaayoune, K., Scantlebury, C., Christley, R.M. (2011) A randomized triple blind trial to assess the effect of an anthelmintic programme for working equids in Morocco. BMC Vet. Res. 7:1.

Lyons, E.T., Drudge, J.H., Tolliver, S.C. (1986) Pyrantel pamoate: evaluating its activity against equine tapeworms. Vet. Med. 81, 280–285.

Mathews, J.B. (2011) Facing the threat of equine parasitic disease. Equine Vet. J. 43 (2) 126–132.

Nielsen, M.K., Fritzen, B., Duncan, J.L., Guillot, J., Eysker, M., Dorschies, P., Laugier, C., Beugnet, F., Meana, A., Lussot-Kervern, I., Samson-Himmelstjerna, G.V. (2010) Practical aspects of equine parasite control: a review based upon a workshop discussion consensus. Equine Vet. J. 42 (5) 460–468.

OIE terrestrial manual on equine piroplasmosis. Available online at: http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.05.08_EQUINE_PIROPLASMOSIS.pdf

Proudman, C., Mattews, J. (2000) Control of intestinal parasites in horses. In Practice. 22: 90–97.

Royal Veterinary College/Food and Agriculture Organisation. Guide to Veterinary Pathology. McMaster egg counting technique. Available online at: http://www.rvc.ac.uk/review/parasitology/EggCount/Step2.htm

Trawford, A., Mulugeta, G. (2008) Parasites. In: The Professional Handbook of the Donkey. 4th Edn. Eds: Duncan, J. and Hadrill, D. Whittet, Yatesbury, UK. 82–110.

Uhlinger, C., Johnstone, C. (1984) Failure to re-establish benzimidazole susceptible populations of small strongyles after prolonged treatment with non-benzimidazole drugs. Equine Vet. Sci. 4, 7–9.

von Samson-Himmelstjerna, G. (2012) Anthelmintic resistance in equine parasites – detection, potential clinical relevance and implications for control. Vet. Parasitol. 185 (1) 2–8.