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Parasite Control Recommendations for Horses During the First Year of Life

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Take Home Message

Parasite control during the first year of life should attempt to limit but not preclude infection so juveniles can develop acquired immunity without accumulating pathogenic worm burdens. Anthelmintic resistance greatly complicates nematode management during year one, and control recommendations must be based on known anthelmintic susceptibility patterns of each herd.

Introduction

More dramatic changes occur during the first year of a horse’s life than at any other time in their long existence. Juveniles experience dramatically different social situations and management conditions ranging from isolated confinement with their dams to extensive grazing with numerous members of their age cohort. In addition, they must adjust from a diet of relatively sterile milk to one based on forage that may be contaminated with numerous pathogens. Each of these changes presents new opportunities for infection by internal parasites. Although the same parasites may be encountered every day for the rest of a horse’s life, the initial exposure, resulting in disease and/or immunity, occurs within the first 12 months.

Intensive and frequently excessive parasite control for juvenile horses is regarded as essential. It is seen as an investment in the future because the popular assumption is that horses which don’t experience parasitic disease as foals will be larger, prettier, faster, and more fertile than their less fortunate stable mates.

The objectives of this presentation are to review the essential biology and epidemiology of the various parasitisms that a foal will experience from 0 to 12 months of age, discuss the scope of potential host/parasite interactions, list the anthelmintic drugs that are effective against the respective target parasites, and propose practical recommendations for preventing disease and minimizing transmission.

Management Schemes

Because the transmission of most parasites is controlled by environmental factors, it is logical to classify the various parasitisms of foals on the basis of the management venues in which they are encountered.

Coprophagy of fresh feces by young foals is a normal feature of their development, and it occurs in all types of management. Coprophagy does not represent a potential source of infection with any equine parasites. Nevertheless, one possible complication of this behavior is that eggs of
parasites harbored by the mare may appear in the feces of the foal. These eggs can be detected by routine fecal analysis, but the age and previous management of the foal should reveal the artifactual nature of the diagnostic finding.

Confinement

Chronologically, foaling stalls are the first habitats occupied by the majority of well-managed foals. Depending on farm management practices and the season of foaling, a foal and its dam might remain in a confinement setting for several weeks after parturition. Foals can be infected by three nematode parasites within the stall habitat. These parasitisms are facilitated either by unique modes of transmission, or by infective stages that are resistant to environmental conditions.

*Strongyloides westeri* (threadworms) – *Strongyloides* is the only nematode parasite of horses that can be transmitted vertically, i.e. directly from the dam to the foal without first cycling through the environment. Infective larvae of *S. westeri* appear in mare’s milk during the first week after parturition, and the foal is infected while suckling.¹

Infective *S. westeri* larvae can also exist in the environment, and are capable of invading a host by penetration of intact skin. In mature horses, including brood mares, exposure to infective *Strongyloides* larvae does not result in patent infection. Rather, larvae are distributed to somatic tissues where they encyst and become dormant. This is a dead-end cycle in most horses. The sole exception is pregnant mares, in which the hormonal changes associated with late pregnancy and lactation apparently stimulate reactivation of the larvae. They enter the circulation, travel to the mammary glands, and first appear in the milk a few days after parturition.

Neonatal foals are primarily infected by ingestion of larvae while suckling. The prepatent period of *S. westeri* is only 5 to 7 days, and foals as young as 5 days of age may begin to pass *Strongyloides* eggs in their feces.² Eggs can be demonstrated by qualitative and quantitative fecal flotation procedures, and high egg counts have been correlated with specific clinical signs. *Strongyloides* infections are usually asymptomatic, but persistent diarrhea and ill-thrift are reported occasionally. The contemporaneous occurrence of foal heat diarrhea is a confounding issue, but this universal syndrome is not caused by *Strongyloides* infection. A parasitic syndrome known as “frenzy” has been described, in which foals react frantically to putative, massive percutaneous infection by *S. westeri* larvae.³

Foals that are not infected vertically from their dams frequently acquire *Strongyloides* from the environment once they are turned out to paddocks or pastures. Foals ultimately develop absolute immunity to patent *S. westeri* infection by 4 to 5 months of age.

Drugs that are effective against *S. westeri* are presented in Table 1. Moxidectin (0.4 mg/kg) is also likely effective, but this product cannot be used in the United States in foals less than 6 months of age.
Table 1. Equine anthelmintics with labeled efficacy against *Strongyloides westeri*

<table>
<thead>
<tr>
<th>Anthelmintic</th>
<th>Dosage</th>
<th>Route</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ivermectin</td>
<td>0.2 mg/kg</td>
<td>Oral</td>
<td>Intestinal stages in foals; somatic stages in mares&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td>Oxibendazole</td>
<td>15 mg/kg</td>
<td>Oral</td>
<td>Intestinal stages in foals</td>
</tr>
</tbody>
</table>

*Parascaris equorum* (roundworms or ascarids) – Foals are infected with ascarids via ingestion of larvated eggs from the environment. *Parascaris* eggs can be found anywhere on a horse farm. They have a sticky, protein coat which allows them to adhere to the udder and hair coat of a mare, to surfaces (including vertical walls) in confinement housing, and to objects and soil in paddocks and pastures. Exposure is ubiquitous, and experience has demonstrated that even heroic efforts of sanitation and disinfection are not 100% effective at preventing infection.

Following ingestion of an infective egg, a larva emerges, burrows through the wall of the small intestine, and enters the local lymphatics. Ascarid larvae are carried to the liver, where they migrate through the parenchyma for the first 7 to 10 days following infection. Thereafter, they enter the circulation and are carried to the lungs. Eventually, they leave the pulmonary vasculature, enter the airways, and are carried proximally to the pharynx and swallowed. The majority of ascarid larvae return to the lumen of the small intestine within 28 days after infection. Here, they grow and develop to maturity, and begin to lay eggs approximately 72 – 80 days after infection. Ascarid eggs can be detected by qualitative or quantitative fecal flotation techniques.

Because *Parascaris* is a potential serious pathogen, it is the main target of parasite control in juvenile horses. Symptoms of infection include diarrhea, pot-bellied appearance, weight loss or poor growth, rough hair coat, and possibly increased susceptibility to viral and bacterial pathogens. A large mass of live or dead ascarids can cause intestinal impaction, most frequently as a sequelum to anthelmintic treatment.<sup>5</sup>

Drugs that are effective against *P. equorum* are presented in Table 2.

Since 2002, there have been several reports of failures of ivermectin or moxidectin treatment to achieve >90% fecal egg count reduction (FECR) against various isolates of *P. equorum*.<sup>7-11</sup> Anecdotal reports (Dr. Gene Lyons, personal communication) have noted a similar failure of pyrantel pamoate (6.6 mg/kg) to demonstrate efficacy against certain isolates, as documented by FECR testing.

*Oxyuris equi* (pinworms) – Adult pinworms are found in the dorsal and descending colon. Pinworms exhibit reproductive behavior that is unique among the nematodes. Adult females protrude from the anus and glue their eggs as a proteinaceous film on the perianal region. These egg masses desiccate and flake off, thereby introducing eggs into the environment. In favorable environmental conditions of moderate temperatures and moisture, the ova become infective within a few weeks, and probably remain viable for months. Horses are infected by ingesting...
Table 2. Equine anthelmintics with efficacy against *Parascaris equorum*

<table>
<thead>
<tr>
<th>Anthelmintic</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenbendazole</td>
<td>10 mg/kg</td>
<td>Adults in intestine</td>
</tr>
<tr>
<td>Fenbendazole</td>
<td>10 mg/kg q24h</td>
<td>Highly effective against migrating ascarid larvae, but not currently labeled for these stages⁶</td>
</tr>
<tr>
<td></td>
<td>for 5 days</td>
<td></td>
</tr>
<tr>
<td>Ivermectin</td>
<td>0.2 mg/kg</td>
<td>Adults in intestine, and migrating larvae in liver and lungs</td>
</tr>
<tr>
<td>Moxidectin</td>
<td>0.4 mg/kg</td>
<td>Adults in intestine; not labeled for migrating larvae but probably effective</td>
</tr>
<tr>
<td>Oxibendazole</td>
<td>10 mg/kg</td>
<td>Adults in intestine</td>
</tr>
<tr>
<td>Piperazine</td>
<td>44 mg/kg</td>
<td>Adults in intestine; rarely used</td>
</tr>
<tr>
<td>Pyrantel pamoate</td>
<td>6.6 mg/kg</td>
<td>Adults in intestine</td>
</tr>
<tr>
<td>Pyrantel tartrate</td>
<td>~2.64 mg/kg q24h</td>
<td>When administered daily, prevents invasion of somatic tissues by ascarid larvae</td>
</tr>
</tbody>
</table>

⁶ may not be used in the United States in foals <6 months of age

larvated eggs from the environment. Larval pinworms develop in the cecum and colon, in close association with the mucosa. As they approach maturity, the adults relocate to the distal alimentary tract and begin to lay eggs approximately 3.5 to 5 months after infection.

Diagnosis is rarely accomplished by fecal flotation techniques, because female *Oxyuris* do not routinely deposit eggs directly into the feces. Infection is usually confirmed by observation of adult pinworms in the feces, protruding from the anus, or adhering to a palpation sleeve during a rectal examination. *Oxyuris* eggs are more likely to be found by fecal flotation if the fecal sample was collected *per rectum* with a lubricated, gloved hand. Pinworm eggs can be demonstrated by sampling perianal debris with scotch tape or with a tongue depressor coated with lubricant, and examining the detritus microscopically.

The deposition of eggs by female worms is apparently quite irritating, which results in the primary clinical sign associated with pinworm infection – tail rubbing. Although many conditions cause tail-rubbing in horses, observation of this behavior frequently triggers empirical treatment for pinworms. If the tail-rubbing fails to abate, anthelmintic failure is often assumed, even though the initial treatment decision may not have been supported by diagnostic evidence.

It has long been thought that horses develop immunity to pinworm infection in early adulthood.¹² However, the author has recently observed sizeable infections in geriatric and middle-aged horses, suggesting either that this generalization is inaccurate, or that the biology of pinworms has changed.

Anthelmintics that are effective against pinworm infection are listed in Table 3.
### Table 3. Equine anthelmintics with labeled efficacy against *Oxyuris equi*

<table>
<thead>
<tr>
<th>Anthelmintic</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenbendazole</td>
<td>5 mg/kg</td>
<td>Used at 10 mg/kg if ascarids are a co-target</td>
</tr>
<tr>
<td>Ivermectin</td>
<td>0.2 mg/kg</td>
<td>Anecdotal reports of treatment failure</td>
</tr>
<tr>
<td>Moxidectin(^a)</td>
<td>0.4 mg/kg</td>
<td>Anecdotal reports of treatment failure</td>
</tr>
<tr>
<td>Oxibendazole</td>
<td>10 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Pyrantel pamoate</td>
<td>6.6 mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

\(^a\) may not be used in the United States in foals <6 months of age

In recent years, several sources have anecdotally reported the failure of macrocyclic lactone treatments to remove pinworms.\(^{13}\) These infections purportedly survived frequent treatment with ivermectin or moxidectin, often at elevated dosages. Although many of these reports had not been substantiated diagnostically (i.e., they were based on persistent tail rubbing), others have been confirmed by demonstration of adult worms or by recovery of *Oxyuris* eggs from the perianal region.

### Paddocks

For purposes of the present discussion, a paddock habitat will be differentiated from that of a pasture by the absence of herbage/forage in the former. A paddock (or dry lot) is considered any open area with a surface consisting of bare soil, sand, gravel, wood chips, etc. Any herbage present in a paddock should be insufficient to serve as a source of forage.

The major parasites of horses that can be transmitted in a paddock habitat (so-defined) are *Strongyloides*, ascarids, and pinworms. The likelihood of infection with ascarids and pinworms is probably greater in paddocks than in stalls, simply because stall bedding is changed with some frequency, whereas paddock footing may be unaltered for long periods. Because *Strongyloides* can be transmitted by free-living stages in soil or sawdust bedding, foals that were not infected vertically from the dam often experience their primary *Strongyloides* exposure after initial turnout.

Specific parasite control recommendations for foals held in paddocks are essentially the same as for those kept in confinement with their dams.

### Pastures

All parasites that can be acquired in paddocks are also transmitted in pasture habitats. The ecologic requirements of some parasites, however, restrict their transmission to open grass.

### Strongyles

Contrary to common belief, minimal transmission of strongyles occurs in confinement or on paddocks. Stalls are generally too dry to support larval development, and moist stalls often have...
excessive accumulations of urine. Urea in urine breaks down into ammonia, which is toxic to developing strongylid larvae. Paddocks generally lack a vegetation thatch layer which provides a humid and aerobic microclimate that is essential for larval development and survival.

Strongylid nematodes (large strongyles and small strongyles or cyathostomes) are uniquely dependent on pasture (forage/herbage) habitats for transmission. Strongylid eggs pass in the feces, and under appropriate environmental conditions (~45°F to 85°F), the eggs hatch and the emerging larvae develop sequentially through first (L1), second (L2), and third larval stages (L3). The L3 is the only stage that is capable of infecting a new host. Transmission is accomplished when grazing horses inadvertently ingest third-stage larvae with forage.

**Large Strongyles** - Large strongyles exhibit protracted migration through systemic tissues such as arteries, liver, and pancreas. Upon completion of the migratory phase, larvae return to the lumen of the gut, where they reproduce and lay eggs. Large strongyles have very long prepatent periods, with a minimum of 6 months required between initial infection and the first appearance of eggs in the feces.

The larval stages of large strongyles are highly pathogenic and damage the organs through which they migrate. Clinical signs of large strongyle infection are vague and non-specific, but may include weight loss, poor growth, recurrent colic, rough hair coat, and compromised performance. Although large strongyles are significant, historical equine pathogens, they have been all but eradicated from most well-managed farms.

**Cyathostomes** - Shortly after infection, cyathostome (small strongyle) larvae invade the lining of the cecum and ventral colon, where they molt and grow within fibrous cysts in the mucosa or submucosa. Larvae may reside in the cysts for just a few weeks, or perhaps for as long as 2.5 years. Cyathostome larvae invoke minimal inflammatory response by the host as long as they remain encysted. When the cyst wall is ruptured by an emerging larva, however, the accumulated excretory and secretory products are released, creating foci of intense mucosal inflammation. The most significant damage caused by cyathostomes is associated with synchronous emergence of large numbers of encysted larvae, resulting in diffuse inflammation (hemorrhage and edema) of the cecum and ventral colon. Adult cyathostomes live adjacent to the mucosa, and primarily feed on organic material within the ingesta. Thus, mature cyathostomes do not have a significant role in the pathophysiology of clinical strongylosis.

Anthelmintics that are effective against large and small strongyle infections are presented in Table 4.

**Tapeworms**

*Anoplocephala perfoliata* is the most prevalent cestode (tapeworm) of horses worldwide. Horses become infected by ingesting intermediate hosts (free-living, oribatid mites) while grazing. Adult *A. perfoliata* attach around the ileocecal junction and cause severe, transmural inflammation of the cecal wall. Tapeworm infection is associated with colic, ileal impaction, and ileocecal intussusceptions. Anthelmintics that are effective against equine tapeworm infections are presented in Table 5.
Table 4. Equine anthelmintics with labeled efficacy against large strongyles and cyathostomes.

<table>
<thead>
<tr>
<th>Anthelmintic</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenbendazole 5 mg/kg</td>
<td>Widespread resistance by cyathostomes</td>
<td></td>
</tr>
<tr>
<td>Fenbendazole 10 mg/kg q24h for 5 days</td>
<td>Larvicidal for migrating large strongyle larvae and encysted cyathostomes</td>
<td></td>
</tr>
<tr>
<td>Ivermectin 0.2 mg/kg</td>
<td>Larvicidal for migrating large strongyle larvae; no resistance by cyathostomes</td>
<td></td>
</tr>
<tr>
<td>Moxidectin 0.4 mg/kg</td>
<td>Larvicidal for migrating large strongyle larvae and encysted cyathostomes; no resistance by cyathostomes</td>
<td></td>
</tr>
<tr>
<td>Oxibendazole 10 mg/kg</td>
<td>Some cyathostome populations are resistant</td>
<td></td>
</tr>
<tr>
<td>Piperazine 44 mg/kg</td>
<td>Rarely used; inconvenient to administer; no efficacy against large strongyles</td>
<td></td>
</tr>
<tr>
<td>Pyrantel pamoate 6.6 mg/kg</td>
<td>Effective against adult stages only; some cyathostome populations are resistant</td>
<td></td>
</tr>
<tr>
<td>Pyrantel tartrate 2.64 mg/kg q24h</td>
<td>Top-dressed on feed daily as preventive against strongylid infection; some cyathostome populations are resistant</td>
<td></td>
</tr>
</tbody>
</table>

Table 5. Equine anthelmintics with labeled efficacy against Anoplocephala perfoliata.

<table>
<thead>
<tr>
<th>Anthelmintic</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Praziquantel 1 mg/kg to 2.5 mg/kg</td>
<td>Dosages vary with the combination product in which PRZ is included.</td>
<td></td>
</tr>
<tr>
<td>Pyrantel Pamoate 13.2 mg/kg</td>
<td>Only one pyrantel product is labeled for efficacy against cestodes</td>
<td></td>
</tr>
<tr>
<td>Ivermectin 0.2 mg/kg</td>
<td>Larvicidal for migrating large strongyle larvae; no resistance by cyathostomes</td>
<td></td>
</tr>
<tr>
<td>Moxidectin 0.4 mg/kg</td>
<td>Larvicidal for migrating large strongyle larvae and encysted cyathostomes; no resistance by cyathostomes</td>
<td></td>
</tr>
</tbody>
</table>

a Zimecterin Gold (1 mg/kg); Equimax (1.5 mg/kg); Quest Plus (2.5 mg/kg)
b Pyrantel Pamoate Paste (IVX Animal Health)

Control Programs

The objective of nematode control is not removal of worms (in fact, “deworming” is a misleading and unfortunate term.) Rather, the objective of parasite control is prevention or diminution of environmental contamination with reproductive stages. Decreasing the number of infective stages to which susceptible horses are exposed ultimately reduces transmission.
Many practitioners prefer to base treatment recommendations on diagnostic evidence (i.e., a positive fecal examination). Some equine parasites, such as large and small strongyles for instance, cause their greatest damage as larval stages, long before they are capable of reproduction. So in many cases, a positive fecal examination is not only proof of a patent infection, it is evidence of prior disease that could have been prevented.

Parasite control efforts during the past 40 years have relied almost exclusively on chemical removal of adult parasites. But, as pharmaceutical alternatives become fewer due to anthelmintic resistance, holistic approaches must be developed to achieve sustainable control. Future strategies will require exploitation of biological vulnerabilities and greater use of management interventions.

**Threadworms**

*Strongyloides westeri* infection is usually an inconsequential parasitism, so routine measures for control are not considered essential. Treating mares in late gestation with macrocyclic lactones (i.e., ivermectin, 0.2 mg/kg or moxidectin, 0.4 mg/kg) reputedly blocks vertical transmission by killing *Strongyloides* larvae in their somatic reservoirs prior to migration and lactogenic transmission. Treatment of individual foals with clinical strongyloidosis, or of herds with a history of problems can be accomplished with ivermectin or oxibendazole. Absolute immunity obviates any need for treatment after foals are 5 months of age.

**Ascarids**

Because anthelmintic resistance by ascarid populations is an emerging problem, each herd should attempt to document the susceptibility profiles of its resident *Parascaris* isolate. This can be determined by fecal egg count reduction testing (FECRT) of confined foals with patent infections. Once the spectrum of effective chemical classes is known, it is recommended that rotation among all viable options be practiced meticulously. The continued efficacy of any anthelmintics used against ascarids should be confirmed on an annual basis by routine fecal monitoring.

Treatment for *Parascaris equorum* infection is not recommended in foals prior to 60 days of age. Earlier treatments with benzimidazoles or pyrimidine anthelmintics are not likely to be effective, and administration of larvicides (i.e., ivermectin, moxidectin, or fenbendazole at 10 mg/kg q 24 h for 5 days) exerts selection pressure for the development of resistance. Treatment between 60 to 70 days after infection effectively removes adults and older larvae from the gut lumen because anthelmintic susceptibility generally increases with worm age and size. It is critical to remove ascarids before they are able to begin reproduction and contaminate the environment with their highly persistent eggs.

After the initial treatment, it may be very difficult to achieve absolute suppression of ascarid egg contamination. Because exposure to infective stages occurs on multiple occasions, if not daily, the worms resulting from each of those exposures will mature at different times. The timing of ascarid treatments should be based on the anthelmintic properties of the product used for the
previous treatment. Following administration of an effective larvicide, a subsequent treatment should not be required sooner than 60 days thereafter. Non-larvicidal anthelmintic treatments (e.g., pyrantel pamoate, oxibendazole, or fenbendazole 10 mg/kg), however, should be followed 30 days later with the next scheduled treatment. Regular treatments with known-effective products should be continued at the recommended intervals until the weanling develops acquired immunity, usually by 8 months of age.

Frequent removal of feces (twice weekly) can greatly reduce the environmental burden of ascarid eggs, but absolute control is impractical. Environmental sanitation and disinfection are beneficial, but will never remove 100% of the eggs present.

**Pinworms**

Control recommendations for *Parascaris* should prove adequate for controlling pinworms as well. The prepatent period of *Oxyurus* is about 50% longer than that of ascarids, and there is evidence that some pinworm isolates have developed resistance to macrocyclic lactone anthelmintics. Equids possibly remain susceptible to pinworm infection for life, whereas immunity to ascarids is virtually absolute in yearlings and older horses. As with any nematode parasitism, the continued susceptibility of indigenous isolates should be monitored regularly, if possible.

**Strongyles**

*Strongylus* spp. – Because a single larvicidal treatment kills nearly 100% of adult and migrating large strongyles within the host, an entire prepatent period (PPP) must expire before a treated horse is capable of re-contaminating its environment with eggs. The minimum prepatent period for any of the large strongyles is 6 months for *S. vulgaris*, so repetition of larvicidal treatments at intervals no greater than 6 months will eliminate the host as a source of infection. And, because infective strongylid larvae can only persist in the environment for a maximum of 1 year, scheduling larvicidal treatments every 6 months over a period of 18 months will eradicate large strongyle infections from a premise.\(^{18}\)

During the first year of life, a strategic treatment with a strongyle larvicide at 6 months of age is an excellent first step in establishing a comprehensive program of large strongyle eradication on a horse farm. Eradication status can be maintained by larvicidal treatments at 6-month intervals and by maintenance of a closed herd. Any incoming horses should receive a mandatory larvicidal treatment, followed by confinement for 4 days before turning them out to pasture.

**Cyathostomes** - Control of cyathostomes is becoming increasingly more difficult. Nearly all populations of cyathostomes are resistant to anthelmintics of the benzimidazole class (fenbendazole and oxibendazole), and increasingly more are resistant to pyrimidine drugs (e.g., pyrantel pamoate and pyrantel tartrate).\(^{19}\) Anthelmintic susceptibility profiles should be determined for the cyathostome populations on every farm. These can be demonstrated by fecal egg count reduction testing (FECRT) with mature members of the herd.
Macrocyclic lactones remain consistently effective against cyathostomes, but the specter of potential resistance in this class has fostered recommendations to decrease selection pressure by reducing the number and frequency of deworming treatments within a herd. A detailed discussion is beyond the scope of this presentation, but future control programs for cyathostomes will concentrate chemical intervention only during seasons of peak transmission, and will exploit differences among horses in their genetically-determined, strongylid contaminative potential.

The frequency of treatments during times of peak transmission should be based on the egg reappearance period (ERP), which is defined as the interval between treatment and resumption of fecal egg counts in excess of 100 eggs per gram (EPG). The duration of ERPs is stereotypic for various anthelmintics, and is quite predictable in mature horses. Relevant to the current discussion, however, ERP’s are much shorter in immature horses, probably as a reflection of their level of immunity to cyathostome infection. Table 6 presents general guidelines for the duration of ERPs.

### Table 6. Duration of Cyathostome Egg Reappearance Periods (ERPs) after treatment with various anthelmintics.

<table>
<thead>
<tr>
<th>Anthelmintic Class</th>
<th>Drug</th>
<th>ERP in Mature Horses*, **</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzimidazoles</td>
<td>Fenbendazole</td>
<td>4 weeks</td>
</tr>
<tr>
<td></td>
<td>Oxibendazole</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Pyrimidines</td>
<td>Pyrantel pamoate</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Pyrantel tartrate</td>
<td>Administered daily, so ERPs do not apply; counts &gt;100EPG during program suggest inefficacy</td>
<td></td>
</tr>
<tr>
<td>Macrocyclic Lactones</td>
<td>Ivermectin</td>
<td>6 – 8 weeks</td>
</tr>
<tr>
<td></td>
<td>Moxidectin</td>
<td>12 weeks</td>
</tr>
</tbody>
</table>

* assumes a cyathostome population that is susceptible to the respective drug  
** egg reappearance periods in juvenile horses are 25-40% shorter

### Tapeworms

Tapeworms are rarely found in foals less than 7 months of age, so there is no need for specific tapeworm treatment prior to weaning. Inclusion of praziquantel or Pyrantel Pamoate Paste (IVX Animal Health; 13.2 mg/kg) with the 6-and 12-month larvicidal treatments for large strongyle eradication should provide adequate control of tapeworms during the first year of life.

### Conclusion

Adequate, comprehensive management of internal parasites during the first year of life can be achieved by implementing an effective program for ascarid control, combined with biannual larvicidal treatments to achieve eradication of large strongyles. This program should provide adequate cyathostome control unless anthelmintic resistance is paired with poor management
(overstocking on highly infective pastures, poor nutrition, stress, concurrent diseases) during ages 6 to 12 months.

The goal of parasite control during the first year of life is neither eradication of parasitism nor complete prevention of infection. Exposure to the more prevalent parasites is not only unavoidable, it is desirable. Antigenic exposure to *S. westeri* and *P. equorum* are necessary for development of acquired immunity. The resulting immunity is virtually absolute, which ultimately is a more effective control strategy than a lifetime of chemicals. Horses that have developed some acquired immunity to other parasites are better able to withstand parasitism whenever existing management standards are compromised through non-compliance, or disrupted by moving a horse to a new farm or training facility.

Parasitic disease is a numbers game. The likelihood of disease and production losses is directly correlated to numbers of parasites acquired, which is in turn a reflection of the degree of transmission to which the animal was exposed. The recommendations presented herein are meant to limit, but not preclude exposure, while reducing the number of treatments and therefore selection pressure for the development of anthelmintic resistance.

**References**

3. Dewes HF, Townsend KG. Further observations on *Strongyloides westeri* dermatitis: recovery of larvae from soil and bedding, and survival in treated sites. *NZ Vet J* 1990; 38:34.