Kentucky Equine Research®

World Leaders In Equine Nutrition

Advances in Equine Nutrition

Volume IV

Edited by

J.D. Pagan

RATIONAL APPROACHES TO EQUINE PARASITE CONTROL

CRAIG R. REINEMEYER
East Tennessee Clinical Research, Inc., Knoxville, Tennessee

Introduction

Since the 1950s when phenothiazine was introduced as the first broad-spectrum anthelmintic, horse owners and veterinarians have applied dewormers in a systematic fashion to limit the transmission of equine parasites. The best-known and most widely practiced control program has been to deworm horses at bimonthly intervals throughout the year. Although this recommendation was evidence-based and highly effective when introduced in the mid-1960s (Drudge and Lyons, 1966), the reproductive behavior of target parasites has changed during the past 40 years, and resistance to certain classes of anthelmintics has further diminished the efficacy of this program.

This historical example confirms the concept that all control measures have a finite life span (Michel, 1976). Parasites are plastic organisms with the ability to adapt to, and ultimately triumph over, virtually all man-made selection pressures. Because most of these adaptations have a genetic basis, future generations of worms may not be susceptible to the same interventions that would have killed their grandparents.

Our knowledge of the biology and ecology of equine/parasite relationships has expanded greatly through research, but this information has effected few changes in control practices in the United States. Unfortunately, many horse owners and most veterinarians still expect parasite control recommendations to be packaged as a simple recipe. The intent of this presentation is not to swap recipes, but rather to examine the essential components of parasite control recommendations, and to present examples of rational programs that can be customized for specific herds in various geoclimatic regions of the United States. Recipes have finite life spans, but understanding the basic elements of parasite control gives us the power to adapt… just like the worms.

Considerations for Control Programs

TARGET ORGANISMS

Equine parasite control programs vary with the age of the host, and strongyles are the only significant parasitic pathogens of mature horses in North America. This diverse group of parasites is usually subclassified as large and small strongyles. Large strongyles were eradicated from most well-managed farms during the past decade, so small strongyles (cyathostomes) are now considered the major targets of parasite control programs for mature horses. Cyathostomes are ubiquitous, and virtually all grazing horses are infected. Horses never develop total immunity to small strongyles, and positive fecal examinations are the rule in untreated animals.

Adult cyathostomes reside in the large intestine, and the females lay eggs, which pass into the environment in the horse's feces. The eggs hatch in favorable environmental conditions, and small, worm-like larvae emerge. After two molts, a third stage larva (L3) results, which is the only phase capable of infecting another horse. Once ingested by grazing horses, infective larvae burrow into the lining of the large intestine, where they are surrounded by a fibrous capsule. After an interval ranging from a few weeks to more than two years, the capsule ruptures, larvae emerge into the lumen of the gut, and the worms mature into adults. Adult females lay eggs, and the cycle is repeated for another generation.

The current prominence of cyathostomes was achieved partially by default, but nevertheless they are valid pathogens that cause colic, weight loss, poor growth, anemia, hypoproteinemia, loss of condition, and rough hair coats (Love et al., 1999). Small strongyles also can cause larval cyathostominosis, which is a severe and potentially fatal diarrheal syndrome associated with the synchronous emergence of large numbers of immature worms from the gut wall. Even in well-managed horses, cyathostomes probably cause subclinical production losses, such as compromised feed efficiency and suboptimal performance. However, these effects remain largely uninvestigated, perhaps because Western cultures refuse to view the horse as a production animal.

Patterns of Transmission

Where? Strongyle eggs pass into the environment anywhere that a horse defecates, but translation (i.e., development into infective, third stage larvae) occurs only in pasture habitats. Stalls are usually too dry to support translation, and a large component of the moisture in wet stalls often comes from urine. The urea in urine breaks down into ammonia, which is highly toxic to developing strongyle larvae. Thus, strongyle infection is an unavoidable risk for grazing horses, but exposure for stabled animals is nil.

When? Development to the Infective Stage. Translation from eggs to infective larvae is regulated entirely by environmental conditions. Moisture and oxygen are essential, but concentrations of both within a fecal pile are usually adequate. Over a wide temperature range (45° F to 85° F), the rate of larval development is directly proportional to environmental temperature. At lower temperatures, hatching and development may require several weeks or months, whereas eggs can hatch and develop into third stage larvae in three to five days when ambient temperatures are in the high 70s (F). Beyond both temperature extremes, eggs either cannot hatch if it is too cold, or larvae develop rapidly but soon die when it is too hot.

When? Persistence of the Infective Stage. Infective L3s cannot ingest nutrients, so they survive by consuming limited, intracellular energy reserves. The duration of their survival is inversely proportional to temperature. Because very little catabolism of energy occurs at low temperatures, cyathostome larvae readily survive through northern winters (Ogbourne, 1973; Duncan, 1974). Conversely, larvae are short-lived during southern summers because energy reserves are consumed more quickly at higher temperatures (English, 1979).

The rigorous environmental limitations on the strongyle life cycle result in predictable, seasonal patterns of transmission. Given the regulatory influences of climate, it should be no surprise that the patterns of strongyle transmission differ among geographic regions. Table 1 presents the seasonal patterns of transmission in major geoclimatic divisions of the continental U.S.

Table 1. Climatic suitability for larval translation and survival, by location and season.

Season	*Development/Persistence Northern Temperate Climate*[A]	*Development/Persistence Southern Temperate Climate*[B]
Spring	++/++	++/++
Summer	++/+	--/--
Autumn	++/++	++/++
Winter	--/++	+/++

[A]Roughly above the latitude of the Ohio River
[B]Below the latitude of the Ohio River

Objectives of Parasite Control

Most owners and practitioners would agree that the ultimate goal of equine parasite control is to optimize the health and performance of horses. The responses differ, however, if one asks, "What are you trying to do when you give a dewormer?" The most frequent answer is, "Kill worms." However, killing worms per se is **not** the objective of a parasite control program. This is especially true for cyathostomes, which exert the majority of their damaging effects before they are susceptible to many dewormers.

The direct source of cyathostome infection is larvae on pasture, and those larvae developed from eggs that were deposited by grazing horses. Once strongyle eggs turn into infective larvae, the only factors that can diminish the risk of future infections are hot weather, time, and exclusion of horses from pasture. The only practical way to decrease future infection is by limiting the passage of worm eggs, and this can be accomplished by killing female worms before they reproduce. Therefore, the objective of parasite control is preventing contamination of the environment with reproductive stages (eggs) of the target parasites.

Appropriate strategies for equine parasite control must be prophylactic. A control program should not be envisioned as a regularly implemented, therapeutic procedure (like dipping a dog to remove fleas), but rather as a series of scheduled interventions that prevent parasite populations from reproducing (like preemergent herbicides). Simply reiterated, cyathostome control recommendations should attempt to limit the passage of large numbers of strongyle eggs onto pasture.

Tools of Parasite Control

Although other management techniques can be useful as adjuncts, anthelmintics (dewomers) are the mainstays of equine parasite control programs. It is essential to understand the relationships and properties of available equine anthelmintics so their characteristics can be exploited.

One finds a bewildering array of equine dewormers on the shelf at the local farmers' co-op, but the available choices for strongyles belong to only four major chemical classes (Table 2).

Table 2. Currently marketed equine dewormers by chemical class, generic name, and trade names(s).

Chemical Class	*Generic Name*	*Trade name*
Benzimidazoles	Fenbendazole	Panacur; Panacur PowerPak
	Oxfendazole	Benzelmin
	Oxibendazole	Anthelcide E.Q.
Tetrahydropyrimidines	Pyrantel pamoate	Anthelban; Exodus; Strongid Paste; Strongid-T; Pyrantel Pamoate Paste
	Pyrantel tartrate	Continuex; Strongid-C, Strongid-C 2X
Macrocyclic lactones	Ivermectin	EquiMax; Equimectrin; Equell; Eqvalan; IverCare; Ivercide; Phoenectin; Rotation 1; Zimecterin; Zimecterin Gold, etc.
	Moxidectin	Quest; Quest Plus; ComboCare
Heterocyclic compounds	Piperazine	Piperazine, various

All of the listed compounds have good efficacy against adult and immature cyathostomes in the lumen of the gut. Only two, however, are known to demonstrate activity against cyathostome larvae that are encysted within fibrous capsules in the wall of the gut. Those are moxidectin, which is effective at 0.4 mg/kg administered once, and fenbendazole (Panacur PowerPak), which is effective when given at 10 mg/kg daily for 5 consecutive days.

When cyathostomes are killed by effective anthelmintics, the fecal egg counts of treated horses should decrease by 90% or more. None of the dewomers, including the larvicides, are 100% effective, however, and strongyle egg production eventually resumes when the larvae that were encysted at the time of treatment mature and begin to reproduce. The interval between treatment and resumption of significant strongyle egg production is termed the "Egg Reappearance Period" (E.R.P.), and its duration varies with the anthelmintic used (Table 3).

Table 3. Duration of egg reappearance periods following use of therapeutic dewormers.

Class or Compound	*Egg Reappearance Period (E.R.P.)*
Piperazine	4 weeks
Benzimidazole	4 weeks
Tetrahydropyrimidine	4 weeks
Ivermectin	6 to 8 weeks
Moxidectin	~12 weeks

The E.R.P. is an extremely important tool to be exploited in parasite control programs. Because the primary objective is prevention of environmental contamination with worm eggs, the E.R.P. tells us how long that condition can be sustained after each treatment with a specific compound.

Anthelmintic Resistance

Resistance is defined as a measurable decrease in the efficacy of a compound against a population of worms that were previously susceptible. Resistance is not due to any change in the drug, but rather to genetic adaptations by the target parasites. How does resistance develop? Genes for resistance traits occur naturally at extremely low levels, but frequent treatments and exclusive use of one drug class provide certain advantages. Whenever resistant worms survive treatment, they are able to continue reproducing in the absence of competition from susceptible worms, and the resistant genotype becomes more frequent in the population. Continued and frequent use of the same class of drug ultimately results in a predominance of resistant genotypes in the population.

Traditionally, many horse owners "rotate" dewormers, meaning they alternate among the available chemical classes (see Table 2). Rotation was originally implemented to cover deficient spectra of the available anthelmintics, not to thwart resistance. Rotation per se is no longer as important as ensuring that all anthelmintics used are still effective.

Resistance to certain drug classes (e.g., benzimidazoles) is alarmingly prevalent, but by no means universal (Kaplan, 2002). Therefore, it behooves practitioners and/or horse owners to determine which drug classes are still effective in a herd, and which should be avoided in the future.

Monitoring Infection Status

Useful information about an animal's parasite status can be gleaned from quantitative fecal examination. This procedure counts the numbers of worm eggs per unit weight of feces, and differs from a standard fecal examination, which can only determine the presence or absence of parasite eggs (Reinemeyer and Barakat, 2004). Although the numerical results are not necessarily correlated to worm numbers or to the severity of disease, fecal egg counts are the essential tool of rational parasite control.

The most important use of quantitative egg counts is determining the spectrum of effective anthelmintics on a farm (Table 4). Subsequent follow-up can confirm the duration of the E.R.P. of various anthelmintics against resident worms (Table 4). And finally, fecal egg counts of untreated horses can determine the relative contaminative potential of individual horses within a herd (Table 4).

Quantitative fecal examination is absolutely essential if one intends to approach parasite control in a rational fashion. However, most equine practices probably don't offer this procedure for their clients at the present time. Diagnostic testing may appear to be just an additional expense, but management decisions based on the results may decrease the total cost of a farm's parasite control program due to savings on unnecessary or ineffective anthelmintic treatments.

Host Factors

Individual horses vary widely in their individual susceptibility to cyathostome infection, and those differences are reflected in the magnitude of their respective fecal egg counts (Duncan and Love, 1991). The majority of the parasites in any group of animals are concentrated in a minority of the animals. Despite this fact, all horses in a herd have been treated exactly the same when it came to parasite control. It should be obvious that rote deworming is wasted on those members of the group that apparently can handle strongyles on their own. It is also likely that the same programs could be suboptimal for the highly susceptible members of the herd.

Fortunately, it is possible to categorize the strongyle contaminative potential of each horse (Table 4). Quantitative fecal egg counts can identify the troublemakers as well as the easy keepers in a herd.

Developing Rational, Customized Control Programs

All of the factors discussed previously should be considered when designing and implementing equine parasite control programs.

Table 4. Various applications of quantitative fecal egg counting techniques.

Application	*Steps*
Determining anthelmintic efficacy or resistance	1. Determine egg counts in fecal samples collected from 6 or more horses prior to deworming. 2. Treat horses with label dosage of anthelmintic(s) to be evaluated. 3. Collect fecal samples from the **same** horses 10 to 14 days after deworming. 4. Perform quantitive fecal examination and calculate efficacy (fecal egg count reduction; FECR) by the formula: ([Pre-count minus post-count] / pre-count X 100). 5. Interpretation: >90% FECR = effective, <80% FECR = resistant, 80% to 90% FECR = equivocal, repeat in future.
Determining duration of Egg Reappearance Period (E.R.P.)	1. Determine egg counts in fecal samples collected from 6 or more horses prior the deworming. 2. Treat horses with label dosage of anthelmintic(s) to be evaluated. 3. Collect fecal samples from the **same** horses at 2-week intervals after deworming. 4. The E.R.P. has expired when egg counts average 50% or greater of pretreatment levels.
Determining strongyle contaminative potential	1. Collect fecal samples from all horses in a herd at least 4 weeks after the expiration of the E.R.P. for the previous anthelmintic treatment. 2. Horses with counts <100 eggs per gram (EPG) are low contaminators; those >500 EPG are high contaminators; those with 100 to 500 EPG are moderate contaminators.

The first step is to determine the spectrum of anthelmintics that are effective in a given herd. This can be accomplished by performing Fecal Egg Count Reduction (FECR) testing with all desired classes of anthelmintics (Table 4). FECR testing will identify the drugs that are viable candidates for inclusion in a program, and also reveals the classes of drugs that should never be used on the farm again.

The second step is to determine the relative strongyle contaminative potential of animals within a herd (Table 4). This procedure identifies those animals that require the greatest deworming attention, and also those that require the least.

The start of the annual strongyle transmission season will differ depending on whether the premise in question is located within the northern temperate or southern temperate region. The transmission season begins when the risk of reinfection for grazing horses changes from minimal to inevitable unless control measures are implemented. In the northern temperate U.S., this shift occurs when horses that were stabled during winter are turned out to pasture in the spring. Spring pastures still harbor residual larvae that developed during the preceding grazing season but survived on pasture through winter. Larval numbers decline during spring, thanks to the warmer weather, and ultimately will reach annual lows by about the first of June. The numbers of pasture larvae will remain low if horses are not allowed to recontaminate the environment with new worm eggs. And how does one stop egg-shedding? By killing adult parasites with anthelmintics.

In the southern temperate U.S., the shift in risk of infection occurs at the end of summer. Just prior to this time, climatic conditions are too hot and often too dry to support survival of infective larvae, even if the horses are dropping lots of worm eggs on pasture at that time. The risk of infection increases during autumn as a consequence of eggs shed recently on pasture.

Our hypothetical herds are now on pastures that are relatively clean (south) or are in the processing of being cleaned up (north), and these grazing venues will remain safe if the horses don't contaminate them with new worm eggs. A single, effective anthelmintic treatment can accomplish this, but we also know that the horses eventually will resume egg-shedding when larvae that survived treatment mature and begin to reproduce. So, the question is, "How long before we need to retreat the horses to maintain zero or at least very low egg counts?" The answer is found in the duration of the E.R.P. following the use of various drugs (Table 3).

Suppressive deworming is the practice of repeating treatment within the E.R.P. of the last compound administered. Thus, repeating treatments nose-to-tail should render fecal egg counts consistently zero or at least very low for as long as effective dewormers are used. However, we must remember that not every horse in the herd requires such an intensive program. We suggest that the low contaminators in a herd receive a single anthelmintic treatment at the beginning of the annual cycle, and another perhaps six months later. Moderate contaminators should receive two treatments administered at suppressive intervals, and the high contaminators should be treated throughout the entire transmission season (i.e., until autumn in the north or late winter in the south).

Table 5. A rational control program for horses pastured in southern temperate climates.

Contaminator Catagory	*Begin Annual Program*	*Additional Winter Dewormings*	*Terminate Annual Program*
Low	October	None	March
Moderate	October	One*	March
High	October	Through entire winter*	March

*Additional treatments are best administered suppressively, i.e., to coincide with expiration of the egg reappearance period of the previously used dewormer.

Table 6. A rational control program for horses pastured in northern temperate climates.*

Contaminator Catagory	*Begin Annual Program*	*Additional Winter Dewormings*	*Terminate Annual Program*
Low	April	None	October
Moderate	April	One**	October
High	April	Through entire summer**	October

*Northern programs are more effective if horses are stabled or held off pasture through the winter months, and first turned out in April or May.
**Additional treatments are best administered suppressively, i.e., to coincide with expiration of the egg reappearance period of the dewormer used most recently.

It is advisable to begin and end each seasonal program with a drug that is effective against migrating large strongyle larvae (ivermectin, moxidectin, or fenbendazole 10 mg/kg for 5 days) to facilitate or maintain eradication of *Strongylus* species from the premises.

Conclusion

The changing patterns of resistance among target nematodes lend an element of urgency to implementing major changes in parasite control strategies for horses in the U.S. The most critical change will be in the attitudes of horse owners and equine veterinarians because the notion of limiting treatment to certain seasons of the year seems radical, and the prospect of leaving certain animals untreated is tantamount to heresy. However, the recommendations and justifications presented in this paper will provide effective control, will decrease selection pressure for the development of anthelmintic resistance, and may accomplish both at a lower cost than current, inefficient practices.

References

Drudge, J.H., and Lyons, E.T. 1966. Control of internal parasites of the horse. J. Am. Vet. Med. Assoc. 148:378-383.
Duncan, J.L. 1974. Field studies on the epidemiology of mixed strongyle infection in the horse. Vet. Rec. 94:337-345.
Duncan, J.L., and Love, S. 1991. Preliminary observations on an alternative strategy for the control of horse strongyles. Equine Vet. J. 23:226-228.
English, A.W. 1979. The epidemiology of equine strongylosis in northern Queensland. 2. The survival and migration of infective larvae on herbage. Aust. Vet. J. 55:306-309.

Kaplan, R.M. 2002. Anthelmintic resistance in nematodes of horses. Vet. Res. 33:491-507.

Love, S., Murphy, D., and Mellor, D. 1999. Pathogenicity of cyathostome infection. Vet. Parasitol. 85:113-122.

Michel, J.F. 1976. The epidemiology and control of some nematode infections in grazing animals. Adv. Parasitol 14:399-422.

Ogbourne, C.P. 1973. Survival on herbage plots of infective larvae of strongylid nematodes of the horse. J. Helminthol. 47:9-16.

Reinemeyer, C.R., and Barakat, C. 2004. Parasite control check. Equus 319:69-78.