Following is a compilation of 12 articles on internal parasites of horses and control programs published in 2004 issues of The Horse: Your Guide To Equine Health Care. *They have been reproduced in their entirety, and we hope you enjoy the series!*

BAD BUG Basics

PARASITE PRIMER—PART 1

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNY FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

It seems that every year, major equine publications take on a virtually impossible task. They publish an omnibus article on equine parasitology that attempts to describe the biology of everything from pinworms to stable flies, discuss the numerous ways in which parasites can harm equine hosts, list the multitude of drugs available for treating parasitic infections, and present a variety of control strategies, all in one user-friendly package.

Like most areas of applied science, equine parasitology is simply too broad and too complicated to be covered adequately in a single article. So in an effort to really do justice to the topic in comprehensive detail, we welcome you to the first of 12 monthly articles on parasite-related topics. To implement this project, *The Horse* has enlisted the aid of three recognized experts in equine parasitology.

Denny French, DVM, MS, Dipl. ABVP, currently is jointly appointed as a

ANNE EBERHARDT

Each parasite generation must return to the environment (usually pastures) for some developmental step, a process traditionally termed a "life cycle."

professor in veterinary clinical sciences at the Louisiana State University (LSU) School of Veterinary Medicine and professor of veterinary science at the Louisiana Agricultural Experiment Station, while maintaining an active role in equine research and ambulatory practice. French's primary research interests have been in the field of equine herd health, especially parasite control programs. Collaborating with Tom Klei, PhD of LSU, French has been involved with the development of numerous anthelmintics (dewormers) and has taken the research information directly into his practice.

Ray Kaplan, DVM, PhD, has been an assistant professor at the University of Georgia College of Veterinary Medicine since 1998. He teaches and does research in large animal parasitology. Kaplan earned his PhD in veterinary parasitology in 1995 at the University of Florida. The goals of his research are to measure, understand, and solve the problems presented by drug-resistant parasites and include three major areas of concentration: 1) Measuring the prevalence of drug resistance; 2) studying the molecular basis of drug resistance; and 3) studying and developing novel and sustainable approaches to parasite control. Much of this work is focused on equine parasites.

Craig Reinemeyer, DVM, PhD, is president of East Tennessee Clinical Research in Knoxville, Tenn. He earned his PhD in veterinary parasitology from The Ohio State University in 1984, and he was a faculty member of the University of Tennessee College of Veterinary Medicine from 1984 to 1998. His major research interests are clinical development of veterinary pharmaceuticals and strategic control of internal parasites of grazing animals.

Let's start by taking a look at how parasites differ from other infectious organisms that damage horses, and go on to explore the historical perspective on equine parasite control—where we've come from, and how far we've yet to go.

What Makes A Parasite?

The world of infectious diseases is inhabited by an incredibly diverse cast of characters, and the list seems to be growing every year. Organisms such as bacteria, viruses, fungi, roundworms, and lice are familiar to most readers. However, the modern list of infectious agents also includes the prions that are responsible for "mad cow" disease, protozoa that induce malaria and equine protozoal myeloencephalitis (EPM), and ehrlichia, which are spread by ticks and other vectors and cause numerous, severe diseases of pets and humans, including Potomac horse fever.

The field of parasitology focuses on those infectious organisms that are considered to be "animals," as opposed to "non-animals" (bacteria, rickettsia, and fungi), or viruses or prions, which are too simple to be either.

The list of generally recognized parasites includes protozoa; flatworms (tapeworms and flukes); nematodes (roundworms); a few rare, worm-like organisms; and a diverse group of arthropods that includes lice, fleas, flies, ticks, and mites.

There are three important ways in which most (but not all) parasitic worms differ from bacteria and viruses:

- Parasitic worms do not amplify their numbers within the host;
- Each generation of parasites must return to the environment to undergo essential changes; and
- Immunity to parasites is rarely absolute.

What do we mean by "no amplification?" Bacteria and viruses are able to increase their numbers exponentially within a host animal. When a young horse ingests the bacterial agent of strangles (*Streptococcus equi*) from a contaminated water bucket, for example, clinical signs do not develop until after those bacteria go through numerous divisions and increase their numbers by millions. Influenza viruses infect cells in the respiratory tract and similarly induce the production of multiple copies, each of which will infect a new cell and do the same thing. In both cases, disease results when sufficient numbers of cells have been damaged to cause clinical signs.

In contrast, when a horse swallows one ascarid (roundworm) egg, one and only one adult ascarid will become established as a consequence of that exposure. The same rule applies for pinworms, strongyles, bots, and most common internal parasites of horses.

Parasitic Disease

Just as with bacteria and viruses, clinical parasitic disease results only when the numbers of infectious organisms reach critical levels. With agents that can amplify within the host, it's simply a matter of time. With non-amplifying parasites, however, numbers (and thus disease) are directly related to the level of exposure. Horses in clean environments pick up few parasites and stay healthy, while horses in contaminated environments acquire more parasites and are at greater risk of disease. (This is the magic key to parasite control and will be discussed in great detail later in the series.)

In contrast, protozoal parasites, which are tiny single-celled organisms, can amplify their numbers within the host. The protozoal agent of EPM *(Sarcocystis neurona)* is a good example, but even this critter conforms to the second general characteristic of parasites—it must return to the environment to undergo essential changes, rather than being transmitted directly from horse to horse.

ANNE EBERHARDT

Determining your horse's fecal egg counts (FEC), or number and type of parasite eggs in the feces, helps you determine what parasites infest him and how heavy his parasite load is.

The word "contagious" implies that a disease can be transmitted directly from one infected animal to another. Thus, a horse with influenza might be directly responsible for infecting others in the herd through droplet spray from a cough or nasal discharge. But not all infectious organisms are contagious. In the case of the vast majority of parasitic infections, a susceptible horse could be housed in the same stall with one harboring any number of parasites without the slightest risk of becoming infected.

Each parasite generation must return to the environment for some essential change or developmental step, a process traditionally termed a "life cycle." Female strongyles (bloodworms), for instance, lay eggs within the gut of the horse, and these eggs pass into the outside environment with the manure. Under conditions of suitable temperature and humidity, strongyle eggs hatch and go through three development stages before they are able to infect another horse. The timing of the life cycle is specific and absolutely essential for the development of the parasite.

One exception to this rule is lice, which lay eggs, hatch babies, grow old, and die, all on the same animal without returning to the environment between generations. This exclusive association is so critical to lice that most species have claws that help them to hang onto the hair shafts of the host.

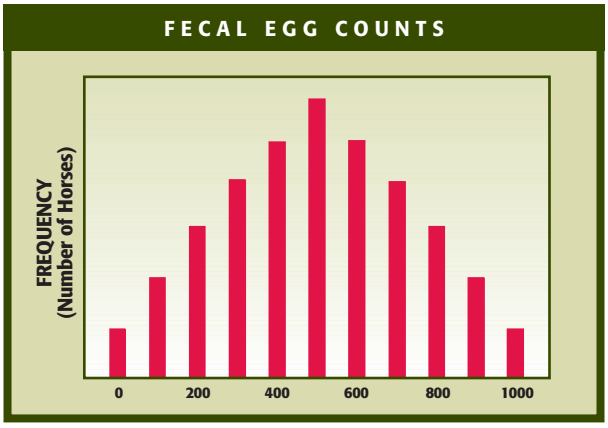
Immunity

The final unique characteristic of parasitic infections is that host immunity is rarely absolute or entirely protective, unlike many bacterial or viral diseases.

When your horse is vaccinated with tetanus toxoid, for instance, you can be quite confident that any contaminated puncture he might acquire over the next year will not result in a fatal case of lockjaw. Have you ever wondered why we don't have a vaccine for small strongyles? It's because few horses in nature ever develop effective immunity against these parasites.

That isn't to say that immunity doesn't occur, but it's probably revealed in less obvious ways. One school of thought holds that immunity against parasites is manifested as protection against disease rather than against infection. Thus, if a 10-year-old mare and a yearling filly were exposed to the same parasite challenge, both would become infected, but the older, experienced horse would be less likely to suffer weight loss, colic, and other side-effects of a parasitic infection because she was somewhat protected as a result of prior exposure.

Individual horses clearly differ in their ability to handle parasitic infections, as exemplified by strongyle fecal egg counts (FEC). If one examines the fecal egg counts of a herd of horses that has not been dewormed for several months, those egg counts will assume an overdispersed distribution (see "Fecal Egg Counts" below). This means that parasites are not evenly distributed among the herd, but rather, most parasites congregate in a small percentage of the herd. In general a few horses will have high FEC, a few will have low or zero FEC, and the majority will be grouped around the average of all counts. The few horses with high FEC will actually be producing the majority of all eggs contaminating the pastures.



Horses differ in their ability to handle parasite infestation; in general, a few horses will have high fecal egg counts (FEC), a few will have low or zero FEC, and the majority will be grouped around the average.

If egg count measurements are repeated the following year, one would find that most horses occupied their similar, prior rankings. In other words, the few horses with low counts in 2002 would likely occupy the lower end of the curve again in 2003 (in the absence of treatment). The individual differences in egg counts are considered to be manifestations of differences in immunity.

Horses develop nearly absolute acquired immunity to two parasites: *Strongyloides westeri* (threadworms) and *Parascaris equorum* (ascarids or roundworms). Both of these are rarely seen in horses after six months and two years of age, respectively. These are exceptions to the rule, however; few other domestic animals are able to control their parasites as effectively as horses manage these two.

Because horses apparently don't develop effective immunity against more harmful parasites such as large and small strongyles, controlling these parasites becomes a lifelong challenge for horse owners. Stay with us over the course of this series for some detailed information about how this can be accomplished.

A Short History of Deworming

Mankind has been aware of the impact of parasites almost as long as we've practiced animal husbandry. But for many centuries, efforts to tackle the problem were at best ineffective, and at worst life-threatening for the horse as well as the worms!

A 1999 paper in the journal *Veterinary Parasitology* by Gene Lyons, PhD, and Sharon Tolliver, BS, of the Gluck Equine Research Center at the University of Kentucky; and Hal Drudge, DVM, professor emeritus of the University of Kentucky, took a look at equine parasite control from a historical perspective and described several rather questionable early "cures" for worms.

Back in 1610, veterinary surgeons were inclined to use bloodletting—drawn from the horse's palate—to address parasites, with the notion that making the horse drink the blood afterward "would kill the worms and help most inward maladies." (How exactly one induces a horse to drink its own blood is not described, which is perhaps a mercy!)

Other alleged anthelmintics in history include chicken eggs, chicken dung, human feces, the guts of young hens or pigeons, black (carbolic) soap, aniseed, aloe, antimony, licorice, linseed (flaxseed), and even quicksilver (mercury). Horse owners were cautioned not to use some of these treatments in pregnant mares because they often triggered "violent purges."

Tobacco was another popular home remedy for internal parasites, and one that some horses readily ate. But although nicotine does have some anthelmintic properties, Kaplan notes that it's extremely toxic in the doses needed to kill worms. "Just giving a plug of tobacco isn't going to do anything, and the correct dose would make a horse quite sick," he says.

There are a number of herbs to which anthelmintic properties have been attributed, but they tend to have the same problem—what's poisonous to the worms is also

DOROTHY WOODWARD

Prior to the 1960s, horse owners had no set deworming routine. They just tended to administer an anthelmintic when they noticed signs of a heavy parasite load, such as a dull coat, a pot belly, frequent colic, or weight loss. Nowadays, most farms use deworming as preventive rather than treatment.

poisonous to the horse. Wormwood (artemisia) was named for its antiparasitic qualities, but it is also the basis of the "tonic" absinthe, much abused in Victorian times (and responsible for some cases of brain damage and death among its devotees). Wormseed, a.k.a. *Chenopodium anthelminticum*, is another plant long pressed into service (the oil pressed from its seeds was used to dose horses and other livestock). There is documentation from the early 1900s indicating that oil of *Chenopodium* was a popular deworming treatment for both horses and humans, but it has also been described as causing terrible side-effects.

Colonel Floyd Sager, an equine practitioner who wrote a book reflecting on his almost 60 years as a veterinarian, recalled that horses dosed with oil of *Chenopodium* often would not eat or drink for three to four days afterward and dropped significant amounts of weight.

Tobacco was a popular home remedy for internal parasites in the past, and one that some horses readily ate. But although nicotine does have some anthelmintic properties, it's extremely toxic in the doses needed to kill worms.

The first "chemical" cures for worms came in the form of carbon tetrachloride and carbon disulfide, both in regular use by the early 1900s for eliminating ascarids and bots. They had some effect, but were quite toxic. It wasn't until the 1940s that the modern age of deworming began with the introduction of phenothiazine.

"It wasn't a great drug, either," says Kaplan, "as it was pretty toxic. But it represents the first of a string of anthelmintic drugs introduced to the market at the rate of about one a decade from then on."

Phenothiazine did have the virtue of being the first drug active against strongyles (bloodworms), the most destructive class of equine parasites. It continued to be used into the early 1960s, by which time strongyle resistance to it was documented both in the United States and England. It was also the first drug to be made available as a daily dewormer when top-dressed on the feed. This low-level administration didn't remove strongyle infestations, but it did block or reduce transmission of the worms by affecting their reproduction.

Piperazine, which arrived in the 1950s, was the first medication with broad-spectrum activity (meaning it was effective against more than one type of equine worm). Useful in combating ascarids, small strongyles, and pinworms, it still wasn't effective against large strongyles. A mixture of piperazine, carbon disulfide, and phenothiazine made available in the 1950s and 1960s addressed the problem of toxicity to some degree by reducing the amount of each drug needed. This deworming "cocktail" had to be administered as a large-volume drench given by nasogastric tube, an unpleasant and slightly risky procedure that required a visit from the veterinarian.

Organophosphates—including trichlorfon and dichlorvos—showed up on the market in the early 1970s, along with levamisole, a drug which, while useful for some other types of livestock, quickly proved to be too toxic for horses. Organophosphates, which are pesticides, were useful against bots, pinworms, and ascarids, but not strongyles. So, they also were mixed with piperazine and phenothiazine to attack more worm species. But with a very narrow safety margin, and extreme toxicity when other species (such as birds) were exposed, these drugs were tricky to use at best.

The 1960s and 1970s saw parasite control take a giant leap forward with the discovery of the benzimidazoles, a class of drug that had unparalleled broad-spectrum action and could be delivered at a much lower dose rate, with a much wider margin of safety, than anything previously available. Thiabendazole was the first of these drugs to hit the market, followed quickly by cambendazole, fenbendazole, mebendazole, oxfendazole, and oxibendazole. Many of these remain available today.

The 1970s also took deworming out of the exclusive province of the veterinarian. With the introduction of convenient paste formulations, horse owners could administer dewormers themselves.

Pyrantel (known to most of us as Strongid) was the next class of drug to surface in the 1970s. It provided an alternative to the benzimidazoles when resistance began to be a problem, and it had a broad margin of safety. It also earned a reputation for being effective against tapeworms when given in a double or triple dose, although it was never officially labeled for that use.

But the real "silver bullet" in parasite control was ivermectin, introduced in the early 1980s. The first drug to kill migrating larval stages of worms, as well as the adults, in a regular dosage, it was (and remains) extraordinarily safe and dramatically effective against virtually every class of nematode and arthropod, including external parasites. Ivermectin would quickly come to dominate the market, particularly once it became available in an easy-to-use paste formulation (it was originally introduced as an injectable, which proved not to be the ideal format for horses).

Ivermectin would prove almost *too* good. Thanks in part to throttled-back research

and development programs, it would be another 20 years before any new anthelmintics came down the pike. Moxidectin, in the same drug "family" as ivermectin, was developed as a horse dewormer in the late 1990s. It is the first drug shown to be effective at a single dose in killing the encysted larval stages of small strongyles hiding in the horse's intestinal wall, but has a narrower safety margin than ivermectin. Kaplan notes that "there are no reports of toxicity with moxidectin when administered at a correct dose."

The Evolution of Rotation

Prior to the 1960s, Kaplan says, horse owners had no set deworming routine. They just tended to administer an anthelmintic when they noticed signs of a heavy parasite load, such as a dull coat, a pot belly, frequent colic, or weight loss. An article published in the *Journal of the American Veterinary Medical Association* (JAVMA) in 1966 changed all that. In it, parasitologists Lyons and Drudge looked at worm biology, egg output, and worm epidemiology in horses, and they recommended a deworming routine of every six to eight weeks with thiabendazole (together with other various drugs in combination) to minimize egg shedding on pasture. Preventing the pasture from becoming heavily re-infected with worms was a new idea, and it became deeply embedded as a parasite control routine.

But as Kaplan points out, "That protocol was designed at a time when horses were loaded with all sorts of worms. Ninety percent of equine colics at the time were related to large strongyles. The deworming routine proved extraordinarily successful, and colic incidence dropped dramatically. It was a chemical assault which changed worm populations in domestic horses forever.

"Then ivermectin came along in the 1980s, killing all stages of worms rather than just the adults," Kaplan continued. "Parasites in horses became much less common. With a diligent routine, you could totally prevent large strongyle egg shedding onto pastures. As a result, *Strongylus vulgaris*, once the most common and important species of large strongyle, has become an endangered species in managed horses. And other problems, such as the summer sores caused by *Habronema spp.*, are far less common than they used to be."

However, with these changes have come new problems. Small strongyles are now recognized as the most important equine parasites, and drug resistance is making it increasingly difficult to adequately control these worms.

With resistance to phenathiazine first recognized in the late 1950s, and to thiabendazole just a few years after that, the idea of rotating different chemical classes of drugs was introduced. It also was eagerly accepted, and fiercely adhered to over the following decades.

"Rotation has become accepted as what you have to do," says Kaplan, "and the concept is important, historically. But over the years, resistance has become so prevalent-except against ivermectin, which remains a bit of a mystery—that rotation now means you're often failing to adequately

COURTESY DR. DENNIS BROOKS

With regular deworming, problems such as the summer sores caused by *Habronema spp.*, are far less common than they used to be.

control parasites even when you treat frequently. We've studied farms that follow the most common protocol, which is every eight weeks with a different drug, and found that the horses on many farms using such a program essentially only get two effective dewormings a year. Those are the times they're dosed with ivermectin. The rest of the treatments do little to control small strongyles, leading us to believe that traditional rotation is no longer the best approach."

Several factors are continuing to contribute to drug resistance, which brings us to something of a 21st Century crossroads in parasite control. Should we depend on the now tried-and-true ivermectin and moxidectin and let older drugs fall by the wayside? Or will putting all of our eggs in one basket prove to be a massive mistake?

One thing is certain—it's time for a major re-evaluation of our parasite control routines. Stay tuned as we take the series in-depth over the next 11 issues and explore the best options for our horses' health.

TAPEWORMS:

An Underrated Threat

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

PARASITE PRIMER—PART 2

Of all the types of internal parasites that plague our horses, the one that was long considered fairly inconsequential was the tapeworm-an intestinal innocent bystander, if you will. Parasitologists knew that horses, like most mammals, harbored a few species of these cestodes (flat, segmented worms; in contrast to most equine worms, which are nematodes, or roundworms). However, parasitologists felt tapeworms were uncommon and did little damage in horses. As a result, there were few chemical means of addressing tapeworm infestations; the vast majority of the drugs available, including ivermectin and moxidectin, were developed to deal with the nematodes that were considered the *real* threat to your horse's health.

Recent research, however, has given us startling news: Not only are tapeworms far more prevalent in the equine gut than was previously suspected, but they might well be responsible for some serious health issues in horses.

Here's an update on what we now know about the secret lives of tapeworms.

Tapeworms at Work and Play

Tapeworms are so named because the body of an adult typically is flattened and segmented at regular intervals, like a measuring tape. Each body segment (proglottid) is a separate unit, like the box cars that form a train. Single proglottids, or several linked ones, can break off from the worm's body without killing the worm, which remains attached to the horse's intestinal wall. Because the segments of most equine tapeworms disintegrate within the large intestine, it's rare to see intact proglottids passed in the feces of horses. That fact gave us the illusion for many years that tapeworm infections in horses were uncommon.

The tapeworm's mouth parts are contained in a scolex, which has four suckers enabling it to attach quite fiercely to the horse's gut wall. Below these suckers are lappets, tiny ear-shaped flaps. As we'll see, tapeworms can cause severe inflammation at their attachment sites. They have no digestive system to

ANNE EBERHARDT; INSET COURTESY PFIZER ANIMAL HEALTH

Although tapeworms haven't previously been considered a major health risk for horses, more recent findings indicate that they can cause several types of colic, some more dangerous than others, in the horse (inset shows a tapeworm attached to the gut wall).

speak of, absorbing the nutrients they need into each proglottid through their integuments (skins).

All tapeworms are hermaphroditic, meaning that there are no separate males or females; each individual worm contains the reproductive organs of both sexes. Tapeworms produce eggs, but they usually don't deposit them singly or continuously like most common internal parasites of horses. Rather, tapeworm eggs come in packets that break away from the end of the worm when they've matured, then are carried out of the horse with the manure.

All tapeworms have indirect life cycles, meaning that they must develop initially within a different animal before they can be transmitted to their final host. The first animal infected in this cycle is known as an intermediate host. Apparently, some critical, biological change occurs in the intermediate host that renders the parasite capable of infecting its final host. Familiar examples of intermediate hosts and their respective parasites include mosquitoes for heartworms and fleas or rodents for the tapeworms in your barn cat.

The intermediate hosts for equine tapeworms are oribatid mites, which exist as free-living forms on pasture and often can be found in very high numbers. Oribatids are more common on permanent pastures than on cultivated or newly sown grasslands. The mites apparently swallow tapeworm eggs while feeding on organic material in equine feces. The eggs then hatch and develop to an infective stage (cysticercoid) within the body cavity of the oribatid in about two to four months. Horses get tapeworms by swallowing infective mites while grazing, and the worms mature within the horse in six to 10 weeks.

Tapeworm Species

Three species of tapeworms are known to infect horses in North America, and each has staked out its own little area of the equine intestine. *Anoplocephala perfoliata*, the most common tapeworm of U.S. horses, is commonly found at the junction of the small and large intestine (the ileocecal valve). *A. perfoliata* are usually just over one inch in length and can accumulate in large numbers.

The smallest of the tapeworms, *Paranoplocephala mamillana*, is usually in the range of a half-inch long, and it inhabits the small intestine and stomach.

The monster of the trio is *Anoplocephala magna*, which is also found in the small intestine or stomach of infected horses. This impressive (some might say horrific) tapeworm can range anywhere from four to 30 inches in length.

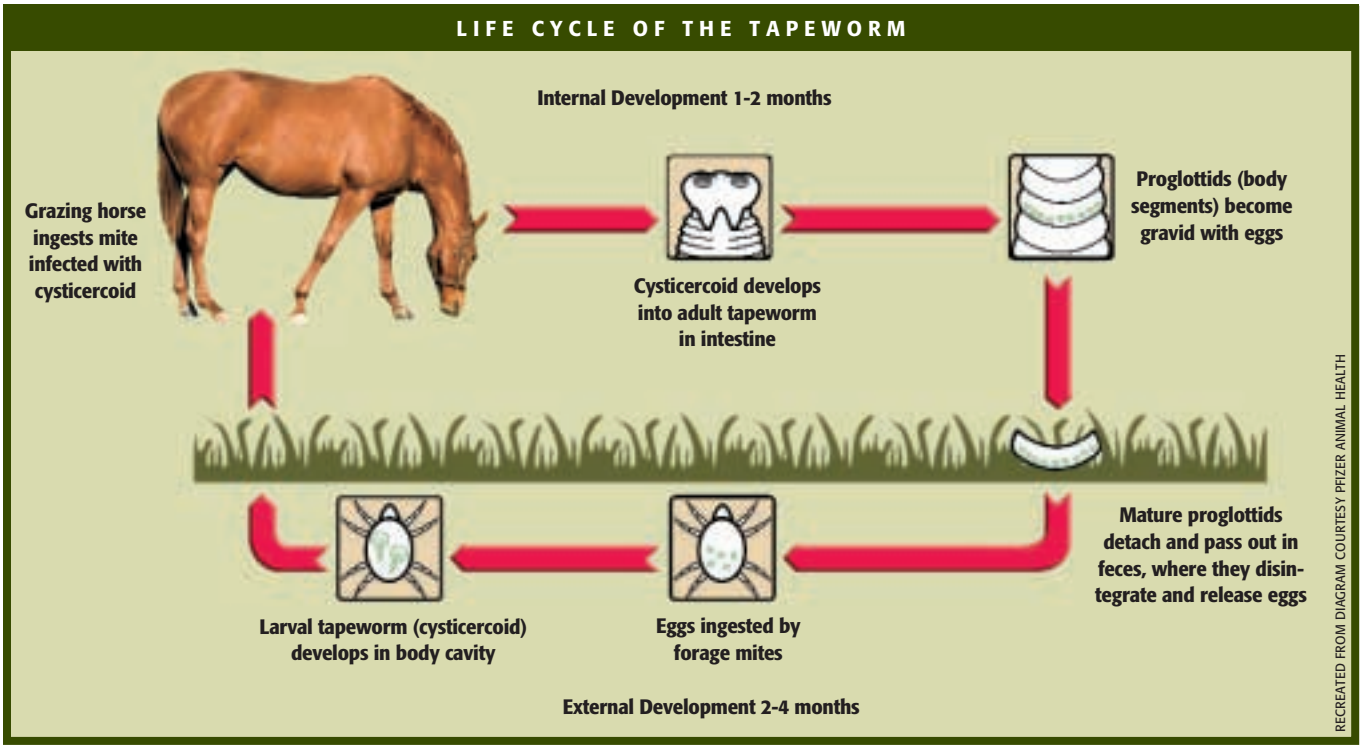
Tapeworm infections were first described in North American horses during the late 1800s. Curiously, *A. magna* was the most common tapeworm in those early descriptions, but this species is uncommon today. In contrast, *A. perfoliata* was a rare finding a century ago, but today it is the most prevalent species worldwide.

Population Patterns of Infection

Historically, it has been difficult to study the population patterns of tapeworm infections because no highly sensitive diagnostic methods were available. Fecal examination, which is excellent for detecting the presence of ascarids and strongyles, frequently doesn't reveal the presence of tapeworm segments or eggs. Parasitologists speculate that the disintegration of tapeworm segments within the bowel results in uneven distribution of eggs within feces. It's also suspected that tapeworms might not release ripe segments on a regular basis. So it is difficult for a veterinarian to prove that a given horse is infected with tapeworms.

The one definitive way to demonstrate tapeworm infection is direct examination of the gut, either post-mortem or during surgery. Numerous post mortem surveys have been conducted in various countries, and several have reported that 50-60% of horses examined were infected with tapeworms. The most thorough investigations in the United States were done in Kentucky, where three surveys found the prevalence of tapeworm infection in adult horses to be 53% (1983), 54% (1984), and 64% (1992).

In 1995, researchers in the United Kingdom developed a test that could detect antibodies to *A. perfoliata* in the blood of horses. A positive result indicated exposure to tapeworms, but not necessarily a current infection with adult worms. This technique was adapted by Stephen Kania, PhD, of the University of Tennessee's College of

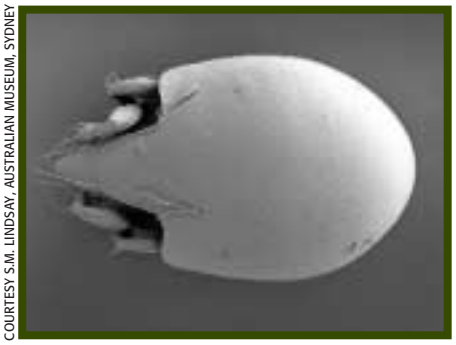


Veterinary Medicine, and it was used in a nationwide survey in 2002 and 2003. Veterinary diagnostic labs in 19 states were asked to submit excess serum from blood samples submitted for Coggins (equine infectious anemia) testing from horses which were at least one year of age. In addition to age, the labs also provided information about the donor horse's sex, breed, and state of residence. Serum samples were frozen and shipped to a lab in Tennessee, where they were examined for antibodies to tapeworm infection.

The survey found that more than 54% of all horses sampled had antibodies to *A. perfoliata* infestations. Prevalence estimates ranged from below 2% in California to higher than 98% in Minnesota; in all, the prevalence of tapeworm antibodies was greater than 30% in 15 out of 19 states—a startling finding.

Analysis of the results found that horses older than 15 years were significantly more likely to have antibodies than younger horses. (Researchers already knew that foals rarely harbor significant numbers of tapeworms before weaning, but once forage becomes a significant part of the diet, exposure to tapeworm infection becomes a lifelong possibility.) The study also demonstrated that stallions were less likely to have been exposed than geldings or mares. Both of these patterns might be explainedby relative amounts of pasture exposure: A lot of older horses are turned out permanently, rather than kept in stalls, and stallions are more likely to be confined indoors.

Also, according to the analysis, mustangs, Arabians, and Quarter Horses were significantly less likely to have antibodies to tapeworms. The pattern in mustangs was attributed to their arid residence (Nevada), but no explanations were found for the differences in Arabians and Quarter Horses.



COURTESY S.M. LINDSAY, AUSTRALIAN MUSEUM, SYDNEY

The intermediate hosts for equine tapeworms are oribatid mites, which exist as free-living forms on pasture. They are more common on permanent horse pastures than on cultivated or newly sown grasslands.

Tapeworms Cause Problems

We know very little about the horse's reaction to tapeworms. Several authors have expressed the opinion that horses do not develop acquired immunity to these parasites, based on the observation that horses of all ages appear to be infected, with some of the most severe infestations found in older horses. Here's some of what we do know:

- High tapeworm burdens are more likely to be associated with serious clinical manifestations, such as spasmodic colic or severe colic due to an ileal impaction or ileocecal intussusception.
- The pathologic findings associated with tapeworms include severe local inflammation of the ileocecal valve (or other attachment sites), ulceration of the mucosa, increased fluid retention and thickening of the gut wall, and local scarring. Sometimes the lesions associated with the attachment sites can partially obstruct the horse's bowel.
- The typical tapeworm infection is not associated with any subtle or chronic clinical signs that would be obvious to the owner or to an examining veterinarian. Most of the clinical events, then, are acute and severe.
- Tapeworm infection is an important cause of various types of colic.

For years, tapeworm infection has been associated with ileocecal intussusceptions in young horses. In this condition, the last foot of the small intestine (ileum) telescopes into the first section of the large intestine (cecum), and swells, blocking passage of intestinal contents. The condition is a result of tapeworms setting up shop at the ileocecal junction, where their pointy mouth parts (which they use to adhere to the gut wall) cause intense inflammation and irritation in a small area of the gut. That stimulates exaggerated peristalsis (the waves of muscle contraction that push food through the gut), resulting in the small intestine itself getting pushed through the opening.

Tapeworms are now considered the primary cause of ileocecal intussusceptions, which can only be corrected surgically (even then, the prognosis is guarded).

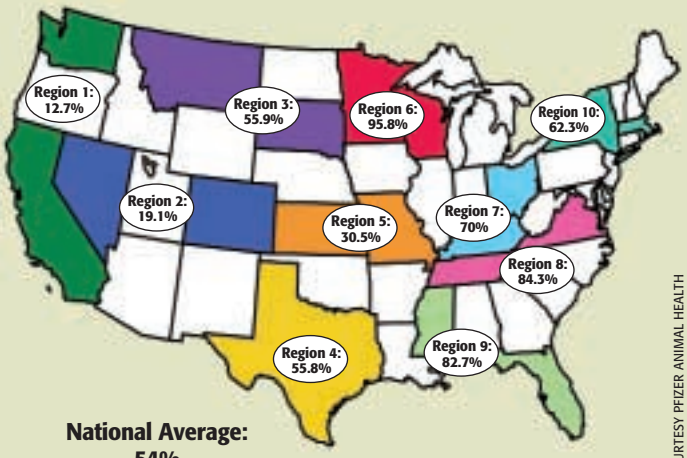
Tapeworms also contribute to ileal impaction colic, in which the wall of the ileum becomes thickened and inelastic, slowing or blocking the passage of food materials. Like intussusceptions, these can only be addressed surgically. In severe cases, they can even trigger ruptures of the small intestine. Chris Proudman, MA, VetMB, PhD, CertEO, FRCVS, of the University of Liverpool, estimated in a 1998 study that some 81% of ileocecal impaction colics were tapeworm-related, and he demonstrated that horses with tapeworms were 28 times more likely to experience this condition than uninfected horses.

Tapeworms also have been shown to cause spasmodic (gas) colic. The presence of tapeworms made horses eight times more likely to experience this than uninfected animals. Spasmodic colic is one of the most common types and can be treated medically.

Region	States	True Prevalence (%)
1	CA	1.3%
	WA	33.3%
2	CO	20.3%
	NV	17.5%
3	MT	44.8%
	SD	70.2%
4	TX	55.8%
5	KS	9.1%
	MO	51.8%
6	MN	99.7%
	WI	92.5%
7	KY	70.6%
	OH	61.8%
8	TN	79.2%
	VA	89.5%
9	FLA	80.9%
	MS	82.5%
10	MA	49.8%
	NY	74.3%

Estimates of Tapeworm Exposure

Percentages based on regional averages.



National Average: 54%

MAP COURTESY PFIZER ANIMAL HEALTH

Although we don't yet fully understand the relationship between tapeworms and some types of colic, researchers speculate that the parasites somehow interfere with normal nerve transmission in the gut. *A. perfoliata* has been shown to contain large quantities of a chemical called acetylcholinesterase, which could interfere with normal peristalsis in the gastrointestinal tract. Any disruption of normal gut motility could certainly be a plausible explanation for signs of colic.

Tackling the Tapeworm

Until recently, no equine anthelmintics were approved for use against tapeworms. In the absence of labeled products, many practitioners recommended doubled or tripled dosages (13.2 or 19.8 mg/kg) of pyrantel pamoate paste or suspension because these regimens were reportedly effective against *A. perfoliata*. Limited studies also have indicated that pyrantel tartrate (2.64 mg/kg) fed daily for 30 days is effective against tapeworms in horses. But at this writing, none of the pyrantel formulations are approved in the United States for use against tapeworm infections.

Gene Lyons, PhD, at the University of Kentucky's Gluck Equine Research Center in Lexington, Ky., demonstrated several years ago that praziquantel was highly effective against *A. perfoliata* infections at all dosages greater than 1 mg/kg. Praziquantel was already in common usage to treat tapeworms in cats and dogs, but its use in horses had not been explored. When praziquantel subsequently came off patent, several veterinary pharmaceutical companies simultaneously began to develop it for equine applications. These efforts culminated in 2003 with the approval of three new deworming products that offer a complete anti-parasitic spectrum, including tapeworms, bots, and most types of nematodes.

All of these products are combinations of a macrocyclic lactone anthelmintic (ivermectin or moxidectin) plus praziquantel (PRZ) at various dosages (see "What Works Against Tapeworms?" above). The concentrations of praziquantel vary among the three competing products, but this is strictly a formulation issue and it has absolutely no impact on efficacy or safety as long as an appropriate amount of drug is delivered to a horse for his respective bodyweight. The label dosages of praziquantel also vary among the combinations. Again, any dosage of 1 mg/kg or greater is virtually 100% effective, so there should be no differences among these products in their efficacy against tapeworms. Finally, praziquantel is an extremely safe compound, so toxicity is unlikely within the range of dosages currently indicated by the labels of available products.

WHAT WORKS AGAINST TAPEWORMS?

PRODUCT (MANUFACTURER)	INGREDIENTS			LABEL CLAIMS	
	FORMULATION	NEMATOCIDE DOSAGE	PRAZIQUANTEL DOSAGE	PREGNANT MARES	MINIMUM AGE
Equimax (Pfizer)	Paste	Ivermectin (0.2 mg/kg)	1.5 mg/kg	Yes	1 month
Quest Plus (Fort Dodge)	Gel	Moxidectin (0.4 mg/kg)	2.5 mg/kg	No	6 months
Zimecterin Gold (Merial)	Paste	Ivermectin (0.2 mg/kg)	1.0 mg/kg	No	5 months
ComboCare (Farnam)	Gel	Moxidectin (0.4 mg/kg)	2.5 mg/kg	No	6 months

Comparison of combination anthelmintics currently approved for treatment and control of tapeworm infections in horses

An Ounce of Prevention

The arrival of deworming products approved for, and effective against, tapeworms just as we begin to understand the true impact of these parasites is one of veterinary science's little serendipities. But the best time to use a product with praziquantel is still something of a question.

A practical approach is to see your objective not as eradication of tapeworms, but as prevention of the accumulation of large numbers of worms that are associated with severe conditions. So how do we do that?

Since horses infect themselves with tapeworms while grazing, one of the simplest methods of preventing tapeworm infection would be to deny them access to pasture. However, for most of us, this is an impractical control recommendation.

At present, we really don't know enough about tapeworm biology to make definitive recommendations for various climatic regions. The most critical piece of missing information is the length of time that infective oribatid mites can survive in the environment. If tapeworms are like most other equine parasites, they probably persist much better in cool or freezing conditions than in hot, dry weather. Logical, evidence-based control recommendations will only be possible after the regional survival patterns of tapeworms and oribatid mites are known.

In the meantime, let's theorize that oribatid mites are likely active only during the warmer months of the year. There are two schools of thought on the best time to treat horses against tapeworms—late winter/spring or autumn/early winter.

Deworming horses in late winter or early spring, before the start of the annual grazing cycle, could help eliminate egg passage as a potential source of infection for mites. A subsequent treatment two to four months later would go a long way toward reducing the numbers of tapeworms that could accumulate in grazing horses.

On the other hand, treating in late autumn or early winter can get horses "cleaned out" soon after transmission ends from the mites. By waiting until spring to deworm horses, tapeworms might have more time to cause damage and seed pastures with eggs. Also, with the combo products available, a late autumn treatment serves dual purpose of bot and tapeworm cleanout.

One bit of good news is that because praziquantel is a completely new class of anthelmintic to horses, there are currently no problems with drug resistance. Coupled with its extremely high margin of safety and almost 100% efficacy, it's a treatment that is bound to be embraced by North American horse owners as part of a comprehensive deworming program.

We'll be looking at recommendations for that deworming program, and other important parasite issues, over the next 10 months in our continuing 2004 series on parasites.

ASCARIDS:

A Growing Problem

PARASITE PRIMER—PART 3

BY KAREN BRIGGS, WITH CRAIG REINEMEYER, DVM, PHD; DENNIS FRENCH, DVM, MS, DIPL. ABVP; AND RAY KAPLAN, DVM, PHD

In the world of internal parasites, ascarids get no respect. Unlike strongyles, they aren't a high-drama threat to your horse's health, and they aren't a "hot topic" parasite like the tapeworm. But that doesn't mean they should be overlooked or discounted in your war on worms. Ascarids, or roundworms, wreak their havoc largely on young horses with naïve immune systems, and that can set your youngster up for depression, stunted growth, and potentially fatal colic.

What Are They?

Ascarid is a general term referring to a large family of closely related parasites that infect a variety of vertebrates, including dogs, cats, horses, cattle, swine, birds, skunks, raccoons, and even humans. Most species of ascarids are host-specific, meaning they will grow to adults and reproduce in only a single type of host animal. So the ascarid of equids, *Parascaris equorum*, occurs in horses, donkeys, and zebras, but is not capable of infecting pigs or dogs.

ARND BRONKHORST

Ascarids, which are usually problematic only for horses up to 15 months of age, were some of the first internal parasites to be recognized by man.

Most ascarids are comparatively large parasites, ranging in width from one to two millimeters (imagine a pencil lead) to three-eighths of an inch, and in length from one to 14 inches. Their large size meant ascarids were some of the first internal parasites to be recognized by man. Adult specimens of *P. equorum*, by far the largest of the common species to infect horses, are approximately the dimensions of a pencil. Ascarids are easily observed in the manure of infected horses, especially after treatment with an effective anthelmintic.

Unlike the tapeworms discussed last month, ascarids have a relatively conventional sex life, developing as separate sexes, males and females. Like most other parasitic nematodes (a phylum of elongated cylindrical worms), the femalesare much larger than the males because they are the egg factories and have a more critical role in propagating the species.

The female ascarid is a prolific egg layer, producing hundreds of thousands of eggs per day, each with a thick, multilayered shell that makes them resistant to desiccation (drying) and freezing. They're also equipped with a sticky protein coating that enables them to adhere to any surface they come into contact with, once they pass into the environment with the manure.

The usual route of transmission for most species of ascarids is through ingestion of infective eggs. Some ascarid species, however, have very complex life cycles. The common ascarid of dogs (*Toxocara canis*), for instance, can be transmitted from the bitch to the pups before they are born, and its counterpart in cats (*Toxocara cati*) can infect suckling kittens through the milk. In addition, each of these species can survive in the tissues of small mammals, and dogs or cats are infected when they exercise their predatory instincts.

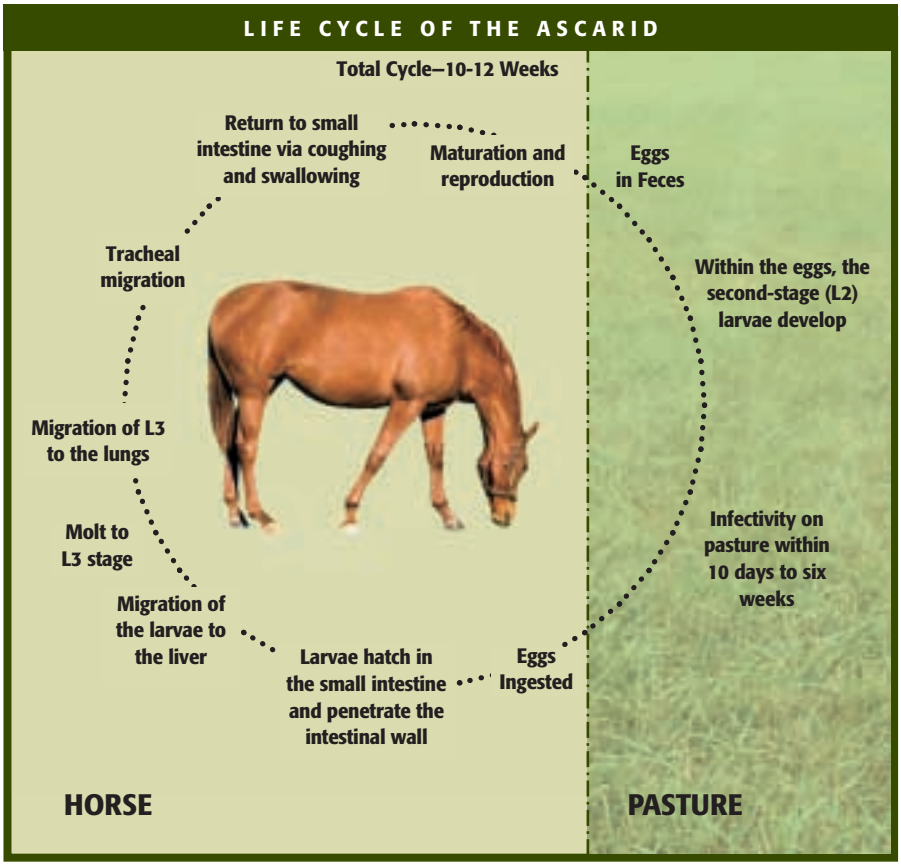
Equine Ascarids

The ascarid of horses has the simplest life cycle of the entire family. The sole route of transmission is via ingestion of infective eggs, usually while grazing. Ascarid eggs become infective in the environment after incubating at moderate temperatures for a few weeks. During this process, an immature worm or larva develops within the egg shell. Once they become infective (or larvated), equine ascarid eggs might remain viable in the pasture for a decade or longer. (An interesting item of equine trivia is that the first descriptions of mitosis, the basic cellular phenomenon that makes cloning possible, were based on observations made with *P. equorum* eggs.)

P. equorum adults usually hang out in the small intestine. But like a racehorse, they have to go around the track once to get to the finish line. When a horse swallows an infective ascarid egg, the larva inside is released in the small intestine. This larva then invades the gut wall and is carried to the liver, either through the circulation or the lymphatic system.

Once in the liver, the ascarid larva migrates through hepatic (liver) tissues for about one week and molts to the next larval stage. It proceeds from the liver, riding the bloodstream to the lungs, where it breaks out of tiny blood vessels and enters the terminal air sacs, or alveoli. The ascarid larva then migrates up the airways, through bronchioles and bronchi, and eventually upthe trachea. This process is greatly accelerated whenever the host (your horse) coughs.

When the migrating ascarid larva reaches the back of the throat (pharynx), it is swallowed and returns to the small intestine after a whirlwind tour of the host's viscera. In the small intestine, the larva becomes mature and finally begins to reproduce. Female ascarids generally start to lay eggs within 10-12 weeks after infection.



Patterns of Infection

P. equorum infection occurs worldwide on every continent where horses reside. But unlike strongyles, bots, and tapeworms, which tend to be concerns of adult horses, ascarids are largely an issue with youngsters up to about 15 months of age. Although adult horses continue to swallow some ascarid eggs as they forage, infections are clinically significant only in youngsters because equids develop excellent acquired immunity to *P. equorum* infection during their second year of life.

Accordingly, mature horses rarely harbor significant numbers of adult ascarids, and few if any eggs are ever detected in their feces by microscopic examination. But ascarid populations in weanlings often number in the hundreds, and associated fecal egg counts can reach several thousand eggs per gram.

It is uncommon for grazing animals to develop absolute immunity to an internal parasite, but *P. equorum* is one of the best-known examples of this unique phenomenon. As we'll discuss later, these differences in host susceptibility influence recommendations for parasite control.

What They Do

The signs of infection with *P. equorum* vary according to the phase of the worm's

ANNE EBERHARDT

Ivermectin is highly effective against migrating stages of ascarids, so treatments with this compound more or less "clean out" an infected host.

life cycle and the total parasite load in the horse. Small numbers of worms probably have a negligible impact on the host's health, but heavy adult ascarid infections can trigger weight loss or poor growth, a rough hair coat, pot-bellied appearance, lethargy, and/or colic.

The exact mechanisms of ascarid disease are unknown, but we suspect that these large worms compete with the host for nutrients in the small intestine. Therefore, despite receiving an adequate diet, a foal might experience secondary malnutrition if a large population of ascarids is hijacking the nutrients before they can be absorbed from the gut.

During the migratory phase of the life cycle, foals often exhibit signs of respiratory disease, including fever, coughing, and nasal discharge. Ascarid-induced respiratory signs do not respond to antibiotic treatment because the cause is not a bacterial infection. Respiratory signs might be worse in weanlings and yearlings than in foals, due to greater levels of acquired immunity and exacerbated host reactions to migrating parasites.

Despite its persistent popularity as one explanation for how parasites cause disease in domestic animals, most parasites are totally incapable of causing a mechanical blockage of the alimentary tract. Ascarids are a notable exception. A tangled mass of ascarids can indeed block the small intestine of a foal or weanling, resulting in severe colic. Most ascarid impactions are precipitated by anthelmintic (dewormer) treatment, and signs of colic usually develop within 24 hours after deworming. The obstructive mass is comprised of dead worms, and severe impactions might progress to intestinal rupture unless the blockage is relieved surgically.

Diagnosis and Treatment

Fecal examination is considered the "gold standard" for demonstrating adult ascarid infections in live horses. But because fecal exams detect eggs, they are only useful for diagnosing infections with mature worms, the sole segment of the worm population that is capable of reproducing. It is extremely difficult to find immature ascarids while they are migrating through the liver and lungs. Blood counts are unreliable, although they might indicate increased numbers and proportions of eosinophils (white blood cells that proliferate in the presence of parasitic invaders); this could also be a non-specific response to other disease conditions or parasites of horses.

Their prominent size and the obvious clinical signs associated with infection probably made *P. equorum* one of the earliest targets of anthelmintic remedies. Our ancestors could not have chosen a tougher objective for their first parasiticidal efforts. That is because ascarids are usually

the "dose-limiting" parasite for equine anthelmintic products. This means that of all the target parasites listed in the spectrum of activity for a dewormer, ascarids require the highest dosage for effective removal. In other words, they are the hardest to kill, and they require a bigger gun to bring them down.

The first historical dewormer with acceptable efficacy against ascarids was piperazine, which was developed in the 1950s. Organophosphates such as dichlorvos and trichlorfon were used in the 1960s, and the 1970s brought a multitude of benzimidazole (BZD) and pro-benzimidazole compounds that made attacking ascarids much less problematic, with their easy-to-administer paste formulations.

The BZD group of drugs is currently represented by Panacur, Safe-Guard, Benzelmin, and Anthelcide E.Q., all of which are effective against ascarids at a dosage of 10 mg/kg. Interestingly, Panacur (fenbendazole) is labeled for use in foals at 10 mg/kg, but the indicated dosage for mature horses is only 5 mg/kg. Why should foals get a proportionally higher amount of drug than their dams? The difference isn't any age-specific differences in the metabolism of fenbendazole by horses, but age differences in their susceptibility to parasites. All target parasites except *P. equorum* are susceptible to fenbendazole at 5 mg/kg, but a higher dosage (10 mg/kg) is needed to kill ascarids. With adult horses being immune to ascarids, there is no need to treat them with the higher dosage; only foals harbor the one parasite that requires a bigger gun.

Another effective way of attacking ascarids is with pyrantel pamoate (Strongid), a drug which is available either as a paste or in liquid suspension. Its chemical cousin, pyrantel tartrate (Strongid-C and others), which is designed to be fed as a daily dewormer, can also be used therapeutically. It has a significant advantage over some other dewormers in that when fed daily, pyrantel tartrate kills ascarid larvae as they emerge from recently ingested eggs, before they can leave the gut to begin their migration through the liver and lungs. Most other dewormers exert their activity only after parasites have been allowed to invade tissues and inflict some initial damage.

Macrocyclic lactones (ivermectin, moxidectin, and abamectin in other countries) also are highly effective against ascarids at relatively low dosages. In addition to killing adult and immature worms in the small intestine, ivermectin is highly effective against migrating larvae in the liver and in the lungs. Moxidectin probably has similar larvicidal activity, but none of the Quest products marketed in the United States are approved for use in foals younger than six months of age.

WHAT WORKS AGAINST ASCARIDS?

CHEMICAL DEWORMER CLASSES	BRAND NAMES	ACTIVE INGREDIENT	DOSAGE
Benzimidazoles	Panacur, Safe-Guard	Fenbendazole	10 mg/kg*
	Benzelmin	Oxfendazole	10 mg/kg
	Anthelcide E.Q.	Oxibendazole	10 mg/kg
Tetrahydro-pyrimidines	Strongid-P, Strongid suspension; Exodus; Pyrantel Pamoate Paste; various generics	Pyrantel pamoate	6.6 mg/kg
	Continuex; Strongid-C; Strongid-C 2X; various generics	Pyrantel tartrate	2.64 mg/kg daily
Macrocyclic Lactones	Equell; EquiMax; Eqvalan; Ivercare; Phoenectin; Rotation 1; Zimecterin; Zimecterin Gold; various generics	Ivermectin	0.2 mg/kg **
	Quest; Quest Plus, ComboCare	Moxidectin	0.4 mg/kg***

* The 10 mg/kg dosage is for foals that might harbor *Parascaris*; older horses presumably immune to ascarids are dosed at 5 mg/kg.
** Zimecterin® Gold is not approved for use in foals younger than five months of age.
*** Quest, Quest Plus, and ComboCare are not approved for use in foals younger than six months of age.

Very recently, there have been several reports of suspected resistance to ivermectin and moxidectin in *P. equorum* on horse farms in North America and northern Europe. All of these reports were based on the failure of ivermectin to eliminate ascarid eggs in the feces of foals. Although this method for detecting resistance is generally accepted for the strongyle parasites, there are no established guidelines for interpreting fecal egg count data for *P. equorum*. Although unlikely, it is possible that something other than resistance was the cause of the apparent inability of ivermectin to eliminate the ascarid eggs in the feces. Therefore, it is important that controlled studies are done to confirm whether worms really are resistant. If confirmed, *P. equorum* will be the first equine parasite known to be resistant to ivermectin, although ivermectin resistance is quite common in parasites of sheep and goats.

As might be expected in a parasite of foals, these reports of suspected resistance have come from breeding farms. Because of frequent movement of foals (and mares) to and from these farms, if ivermectin resistance has occurred, then it is virtually assured that resistant worms have been spread over many farms and geographic regions. Parasite resistance to dewormers is becoming an extremely important problem in all livestock. Therefore, monitoring of control programs by use of fecal egg counts is critical to maintaining effective parasite control and healthy animals.

Warning...

When deworming your youngsters, remember that ascarid impactions are a well-known consequence of therapeutic deworming in juvenile horses. The best way to avoid impactions is a regular control program that never allows a large population of adult ascarids to develop within an individual foal. But how should deworming be implemented for foals which haven't been on a regular control program, or worse, for those which already have developed obvious signs of ascarid infection?

The first step is to enlist the assistance of your veterinarian. The conventional wisdom in these cases is to attempt to remove only a portion of the worms, if possible, to thin out the sheer numbers and thereby reduce the probability of a subsequent blockage. This can be tricky, though, because most approved dewormers are more than 90% effective, and results are very unpredictable when dewormers are administered at less-than-recommended dosages.

Fortunately, it has been reported that fenbendazole (Panacur), at the 5 mg/kg dosage, is somewhat less than 50% effective against *P. equorum*. Therefore, a heavily infected foal (little more than "worms and eyeballs") is one situation calling for use of the lower dosage of Panacur in a juvenile horse. This "partial treatment" should be

followed about one week later by another treatment with fenbendazole at the 10 mg/kg dosage, or with any other effective dewormer at its recommended label dosage.

In an effort to prevent impaction colics, some veterinarians concurrently administermineral oil when deworming foals. But we don't really know whether the presence of oil might interfere with the absorption and distribution of the anthelmintic.

Prevention and Control

Ascarid eggs can be found in any environment occupied by foals, weanlings, or yearlings, and *P. equorum* is one of the few equine parasites that can be transmitted in stalls. Because infective ascarid eggs remain viable for a decade or longer, inadequate control can have chronic consequences. Heroic attempts have been made to eliminate environmental sources of infection, but disinfectants such as bleach, iodine, cresol, and quaternary ammonium compounds aren't very useful against ascarid eggs. Even steam-cleaning of concrete block stalls in research facilities has not been completely effective. Apparently, the thick shell renders ascarid eggs resistant to attack by most chemical and physical agents.

So what's the best solution? The manure of young horses should be picked up frequently and disposed of, and bedding should be stripped regularly. Composting might kill ascarid eggs if fairly high temperatures are sustained for several days to weeks. Stalls that have held foals or weanlings should be cleaned as thoroughly as possible, which is facilitated if the stall design incorporates non-porous surfaces. Due to the sticky protein coating on ascarid eggs, they can adhere to virtually any surface, including vertical elements of stalls and the hair coat of the mare. For this reason, it is good practice to bathe mares prior to introducing them to a foaling stall, and their udders should be cleaned thoroughly.

Preventing ascarid infections in pastured youngsters is tougher, but one practical provision is to feed them from containers so they aren't eating directly off the ground. Remember that the objective of any parasite control program is prevention of environmental contamination with infective stages. Thus, the primary goal is to keep foals from passing *P. equorum* eggs in their feces. Foals can pass eggs within 10 weeks after initial infection, so ascarid control efforts should begin when foals are two months old. At that age, most (but not all) of the dewormers listed above would be suitable choices.

After the initial deworming, how often must we repeat treatments to prevent foals from becoming re-infected and passing more eggs in their manure? Following the use of benzimidazoles or pyrantel compounds (see "What Works Against Ascarids?" on page 13), the subsequent treatment should be scheduled 56 days later. This recommendation is based on the fact that drugs of these two classes only kill worms that have returned to the small intestine, and have no effect against larvae migrating in the liver and lungs. The minimum interval between arrival in the gut and production of eggs is approximately 56 days.

Ivermectin is highly effective against migrating stages, so treatments with this compound more or less "clean out" an infected host. Then, the clock starts ticking all over again as if the foal were a newborn, so the appropriate interval between ivermectin treatments for ascarids is 60 days. (Keep the preceding information about possible resistance to ivermectin in mind, however, and resist the urge to over-use it.)

You can also use a daily dewormer such as Strongid C, but check the labeling carefully beforehand to make sure the product is approved for use in foals.

Whichever mode of attack you choose, expect to continue your assault until the foal is approximately 15 months old. This schedule represents the minimum program for juvenile horses. After that, you can rely on your youngster's natural immunity to have kicked in and kicked out the vast majority of the ascarids.

PAULA DA SILVA

Preventing ascarid infections in pastured youngsters isn't easy, but one practical provision is to feed them from containers so they are not eating directly off the ground.

STRONGYLES:

The Worst of the Worms

PARASITE PRIMER—PART 4

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

Ever since the battle against internal parasites began, researchers, veterinarians, and horse owners have recognized a common enemy-strongyles, sometimes called bloodworms (or, in the United Kingdom, redworms). The largest and most significant family of worms in horses, they're also the most dangerous. In fact, they're considered responsible for the vast majority of serious parasite-related health problems in adult horses, and they have the capacity to kill.

Strongyles are nematodes, with roughly cylindrical bodies that are round in cross-section. Most species range from a half-inch to two inches in length. As their common name suggests, a few varieties of strongyles are blood red in color, although most species are white. The adults are equipped with well-defined buccal capsules (mouth parts) with teeth, the better to latch on to your horse's intestinal wall.

Unlike the tapeworms we discussed in February, strongyles have separate sexes, and males can be distinguished from females by the shape of their tails. Few worms are more prolific-female strongyles lay eggs almost constantly, making it easy to detect a horse infected with adult strongyles by examining manure for eggs.

All strongyles of horses have direct life cycles. This means that they can be transmitted between hosts without involving a different species of animal (tapeworms need another species besides horses to complete their life cycle).

NANCY STEVENS-BROWN

Manure management is essential to parasite control. Strongyle eggs hatch in the fecal pile when environmental temperatures range from 45-85° F.

However, when strongyle eggs are passed in manure, they are not capable of infecting a horse. They must first develop through three distinct stages before becoming infective.

The Climate-Controlled Worm

Strongyle eggs hatch in the fecal pile when environmental temperatures range from 45-85°F. That range is critical: Temperatures below the stated range are too cold for hatching to occur, and freezing is usually fatal to strongyle eggs. And although eggs hatch quickly at higher temperatures, the resulting first-stage larvae (designated L1 by parasitologists) die very rapidly.

At moderate temperatures, the L1 stage larvae consume bacteria and other organic material present in feces, and they eventually molt into second stage larvae (L2). All told, there are three larval stages the young strongyle must go through in the outside environment before it becomes capable of infecting a horse as an L3.

The rate at which strongyle eggs hatch and larvae develop from L1 to L3 is directly proportional to the environmental temperature. In warm weather, eggs can hatch and yield infective larvae in as little as three days, but the process might take several weeks in cooler months.

Once a strongyle egg develops to the L3 stage, however, the environmental conditions that favor its survival are quite different. Third-stage larvae are completely surrounded by a membrane that protects them from drying out. However, the membrane doesn't have a mouth opening. Therefore, L3s cannot feed and must survive on energy that has been stored in their intestinal cells. The quantity of this stored energy is limited, and once it is gone, the larva dies of energy exhaustion and starvation. How quickly this happens is, once again, directly proportional to the environmental temperatures. In warm weather, stores are used up rapidly, but at very low temperatures, little if any are consumed.

What this means for the horse world is that larvae disappear rapidly from pastures during hot, dry weather, but they survive extremely well in freezing conditions. In most regions of the United States, infective larvae present on pasture in October can persist until the following May or June. In climates with hot summers, grazing horses are at far greater risk of parasitism in December than in July.

Horses pick up strongyle larvae through the normal process of grazing, as L3 larvae crawl up blades of grass. The examination of a single early-morning dewdrop on a grass blade might reveal thousands of them. Horses can also ingest the larvae directly from the soil or from drinking contaminated water.

Know the Enemy

Although dozens of species of strongyles are known to infect horses in North America, they can be divided into two major groups—large strongyles (*Strongylinae*, or large bloodworms) and small strongyles (the *Cyathostominae*, also called cyathostomes or cyathostomins). These two groups differ in several major and minor features, but their developmental patterns and responses to environmental conditions are virtually identical.

Size is the most obvious difference between the large and small strongyles. Large strongyles are relatively stout worms up to two inches long, whereas small strongyles are small, hair-like worms, yet they can still be seen with the naked eye.

Although their life cycles outside the horse are practically identical, the large and small strongyles have very different approaches to infection once they've arrived in the horse's gastrointestinal tract. Large strongyle larvae take the grand tour of the equine interior, leaving the intestine soon after infection and migrating through various tissues for the next six to 11 months. The path they take depends on the species of the worms.

The best-known large strongyles are *Strongylus vulgaris*, whose larvae invade the lining of arteries supplying the gut, and *Strongylus edentatus*, the larval stages of which migrate through the liver and peritoneum (the membrane that lines the cavity of the abdomen). A third species, *Strongylus equinus*, tours the liver and pancreas.

Regardless of the route taken, the worms' destination is the same. Eventually, large strongyle larvae return to the gut to mature and lay eggs.

The 40-odd species of small strongyles that infect horses suffer far less from wanderlust than their larger cousins. Instead of taking the migratory approach, they set up housekeeping immediately and provide themselves with defenses that make it nearly impossible for the horse's immune system to attack them.

Shortly after being swallowed, small strongyle larvae invade the lining (mucosa) of the large intestine, where a thin, tough capsule of scar tissue forms around each worm. Within these bubbles, larvae undergo further development. The capsule serves two functions. First, it (temporarily) protects the horse from the parasite, and there is remarkably little inflammation around these cysts as long as the walls remain intact. Simultaneously, the capsule protects the larva from its host's immune reactions, and also from the majority of equine dewormers that are currently marketed.

Researchers have counted up to 60 reddish-black capsules per square centimeter of intestinal tissue in severely infected horses.

The cyathostome stage that first enters the tissues is known as an early third stage larva (EL3). After an EL3 becomes encapsulated or "encysted," it can follow one of two developmental patterns. It might mature progressively, turning into a late third-stage larva (LL3), then a fourth-stage larva (L4), all within the same cyst. Or the EL3 might disrupt further maturation and remain stalled in the early third stage for up to two years or more-a pattern known as arrested development. This happens when there is already a large population of adult small strongyles in the hollow center (lumen) of the gut; the immature larvae appear to be able to wait their turn to come to maturity.

When the adult population dies off, either through "old age" or thanks to being purged by a deworming drug, the encysted larvae eventually emerge from the tissues as L4s, sometimes in huge numbers. Within a few weeks, cyathostome larvae in the lumen mature into adults and begin to lay hundreds of thousands of eggs, which can be observed in the manure of infected horses.

All strongyle eggs are similar in appearance, so one cannot determine whether a horse is concurrently infected with both large and small strongyles by a fecal egg

count. This can be determined only if feces are cultured in a laboratory and the distinctive L3 stages are recovered for identification and differentiation.

Population Patterns

Unlike the ascarids we discussed last month, strongyles, both large and small, are a concern throughout a horse's life.

Although very young foals might pass strongyle eggs in their feces, these could just be the result of coprophagy, i.e., the foal eating his dam's manure (a normal behavior that helps inoculate the foal's cecum with beneficial fiber-digesting bacteria). Researchers believe the ingested eggs are just passing through and do not represent a true infection. Foals begin to acquire strongyle infections as soon as they can nibble at forage, however, and foals as young as six weeks can harbor small strongyles and pass typical eggs in their manure.

Strongyle infections accelerate when grazing becomes a horse's major source of nutrients. In fact, the transmission of strongyles is almost totally limited to pastures, and very little infection is thought to arise in stables or on dry lots. Although some immunity to strongyle infection occurs, it usually amounts only to a reduction of strongyle disease rather than the elimination or prevention of infections. Therefore, horses tend to maintain strongyle infections for their entire lives if not on a deworming program.

Individual horses vary markedly in their susceptibility to strongyle infections. A certain proportion of the herd consistently has very low fecal egg counts, even in the absence of anthelmintic treatment, whereas a similar proportion will probably have high counts and be responsible for the majority of pasture contamination.

The Damage Done

The internal thoroughfares through which large strongyles travel in their migration through the horse suffer greatly from the traffic. The "footprints" these destructive worms leave can include:

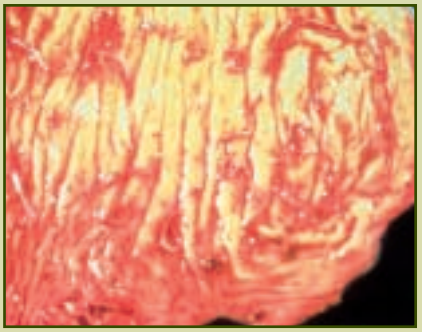
- Rapid weight loss, loss of appetite, fever, lethargy, dull hair coat, poor performance, a "pot-bellied" appearance, diarrhea and/or constipation—the classic signs of a severely parasitized horse;
- Localized hemorrhage, swelling, and small bleeding ulcers in the lining of the cecum and colon, thanks to adult large strongyles attaching with their damaging mouth parts and sucking blood (the worms might move to several different sites over their life spans);
- Anemia and hypoproteinemia (decreased levels of protein in the blood);
- A swollen, bluish-red liver, which can develop chronic fibrosis (caused by *S. edentatus*);
- Inflammation of the abdominal lining (peritonitis) (*S. edentatus*);
- Submucosal cysts in the liver, pancreas, and intestine (*S. equinus*);
- Irritated and thickened arterial walls in the cranial mesenteric artery and its branches, which supply blood to the small intestine, colon, and cecum (*S. vulgaris*);
- Restricted blood flow to the gastrointestinal tract, thanks to partial (or complete) blockages by worms, which can lead to infarctions (areas of dead tissue) (*S. vulgaris*);
- Ballooning of the mesenteric artery, called a verminous aneurysm (a sac formed by the stretching of the wall of an artery), can occur in the intestine, heart, kidney, liver, or legs, which can lead to thrombi (blood clots) gathering there like clusters of grapes. If these clots break free, they can block vessels further downstream (*S. vulgaris*);
- Severe thrombo-embolic colic due to disruptions of the blood supply to the intestine (*S. vulgaris*); and
- In rare cases, complete rupture of the mesenteric artery, which is usually fatal (*S. vulgaris*).

Small strongyle infections have more variable effects. During the initial phase of infection, when larvae are ingested from pasture, massive invasion of the gut can cause local inflammation that might be manifested as diarrhea, loss of appetite, and weight loss. Later, during larval development, there is remarkably little host response to the encysted larvae. They can lurk in the intestinal lining for months or years with no discernible effect on the horse.

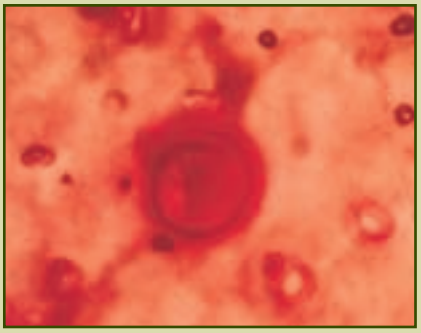
The rupture of the cyst capsules by emerging larvae, however, is accompanied by intense local inflammation. Tissues around ruptured cysts suffer hemorrhage, edema, and local infiltration of inflammatory cells, and the horse can become anemic. The gut damage from emerging larvae can manifest as diarrhea, weight loss, and severe hypoproteinemia (decreased levels of protein in the blood).

There's also a severe syndrome known as larval cyathostomosis associated with the synchronous emergence of large numbers of encysted larvae. Larval cyathostomosis occurs seasonally (often in winter or

LARGE AND SMALL STRONGYLES



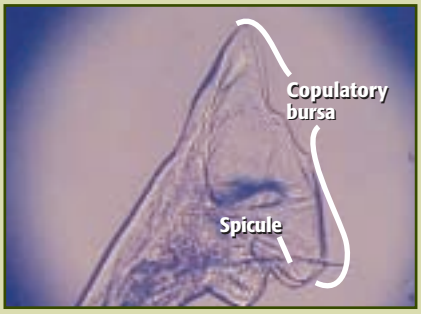
Large strongyles.



PHOTOS COURTESY FARNAM

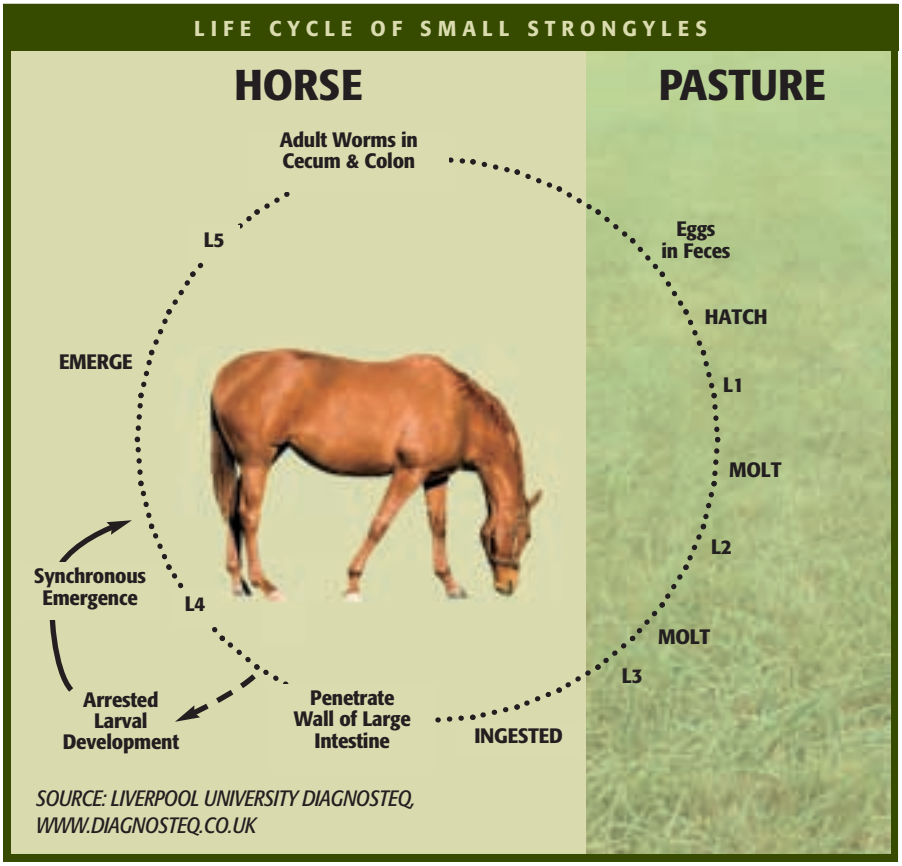
Small strongyle encysted in the intestinal wall.

This is an infective third stage larva of a cyathostome (small strongyle) that would be ingested from pasture.



PHOTOS COURTESY DR. RAY KAPLAN

This is the posterior end of a male cyathostome (small strongyle) showing the copulatory bursa and spicule that are used to grab onto and inseminate the female worm.



spring), and can lead to intense irritation of the mucosal lining of the cecum and colon, impaired gut motility, a sudden onset of diarrhea, weakness, muscular wasting, and severe colic. Rarely, horses can suddenly die with few outward signs of disease, the cause being revealed only on necropsy.

Larval cyathostomosis has a guarded prognosis at the best of times, and it is now considered one of the most serious parasite-related diseases in horses, making small strongyles a much more deadly foe than we once thought.

It should be kept in mind, however, that small strongyles are usually present at all stages of their developmental cycle, each causing different pathologies to the horse. Consequently, with the exception of the severe disease caused by larval cyathostomosis, it is not usually possible to distinguish symptoms caused by the various stages of these worms.

Beating Back the Invasion

Nearly all equine dewormers marketed today are termed "broad spectrum," meaning they're effective against large strongyles, small strongyles, ascarids, and pinworms. The only exception currently available in North America is piperazine, which has no activity against large strongyles.

But there's a catch. All dewormers with label claims against strongyles are effective against the adult, egg-laying stages, but only two classes demonstrate efficacy against migrating large strongyle larvae. These are the macrocyclic lactones (see "What Kills Larval Strongyles?" below left), which include ivermectin and moxidectin, and elevated dosages of certain benzimidazoles. Currently, Panacur and Safe-Guard (fenbendazole) are the only benzimidazoles with label claims against larval large strongyles, and this is achieved by administering elevated dosages (10 mg/kg) daily for five consecutive days (marketed as the Panacur Powerpak).

It's only quite recently that we've been able to tackle the problem of encysted small strongyles, which are left completely unscathed by most deworming drugs, including ivermectin. Only two drugs are considered larvicidal against encysted small strongyles: Moxidectin (at 0.4 mg/kg) and fenbendazole (10 mg/kg daily for five consecutive days), which are both known to kill significant proportions of the encysted cyathostome larvae within the gut mucosa.

Recent studies have found that moxidectin's larvicidal effect was evident within nine days after treatment, and that larvae died within the cysts without inciting any inflammatory reaction. Various researchers have noted improvements in the appearance of the equine gut after treatment with larvicidal dewormers.

Prevention

One of the simplest methods of preventing strongyle infection would be to deny horses access to pasture. Unfortunately, this is an impractical control recommendation, and it comes with its own set of downsides, including increased feed and bedding costs and the potential for the development of vices when your horse is bored and deprived of equine company.

Eradicating strongyle populations from pastures is also a tall order, considering the larvae can survive drought conditions and even the perils of winter. But instituting a control program can at least help prevent accumulation of large numbers of infective larvae on those blades of grass-and that is the surest way to limit worm burdens in your horse. We'll focus on the specifics of pasture management in a future article.

What Kills Larval Strongyles?

ANTHELMINTICS CURRENTLY APPROVED FOR TREATMENT AND CONTROL OF LARVAL STRONGYLE INFECTIONS OF HORSES

			EFFECTIVE AGAINST:		
				Encysted cyathostomes	
Dewormer	Brand Names	Dosage/Regimen	Migrating Large Strongyles	EL3	LL3/L4
Fenbendazole	Panacur, Safe-Guard	10 mg/kg/day; 5 days	Yes	Yes	Yes
Ivermectin	Equell; EquiMax; Eqvalan; Ivercare; Phoenectin; Rotation 1; Zimecterin; Zimecterin Gold; various generics	0.2 mg/kg/once	Yes	No	No
Moxidectin	Quest; Quest Plus; ComboCare	0.4 mg/kg/once	Yes	No	Yes

SOURCE: CRAIG REINEMEYER, DVM, PHD

BOTS & BEYOND

Little-Known Parasite Enemies

PARASITE PRIMER—PART 5

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

When it comes to parasites, worms aren't your horse's only worry. Although nematodes and cestodes (or more colloquially, roundworms and tapeworms) make up the biggest demographic in the parasite "neighborhood" in your horse's insides, there's one other major class of internal pest you need to be concerned about—the larvae of the bot fly. Many other insects and arthropods feed from your horse's resources—lice, mites, and chiggers are three of the most common parasites—but only bots take up long-term residence deep in your horse's interior, sometimes clustering thickly all along his intestinal mucosa like particularly unappetizing gnocchi.

In addition to bots, there are other classes of internal parasites that while not as "high-profile" as strongyles or tapeworms, are important to recognize (and combat!). This month, we'll take a look at these lesser-known interlopers and make some recommendations on how to best limit their impact on your horse's health.

KIM AND KARI BAKER

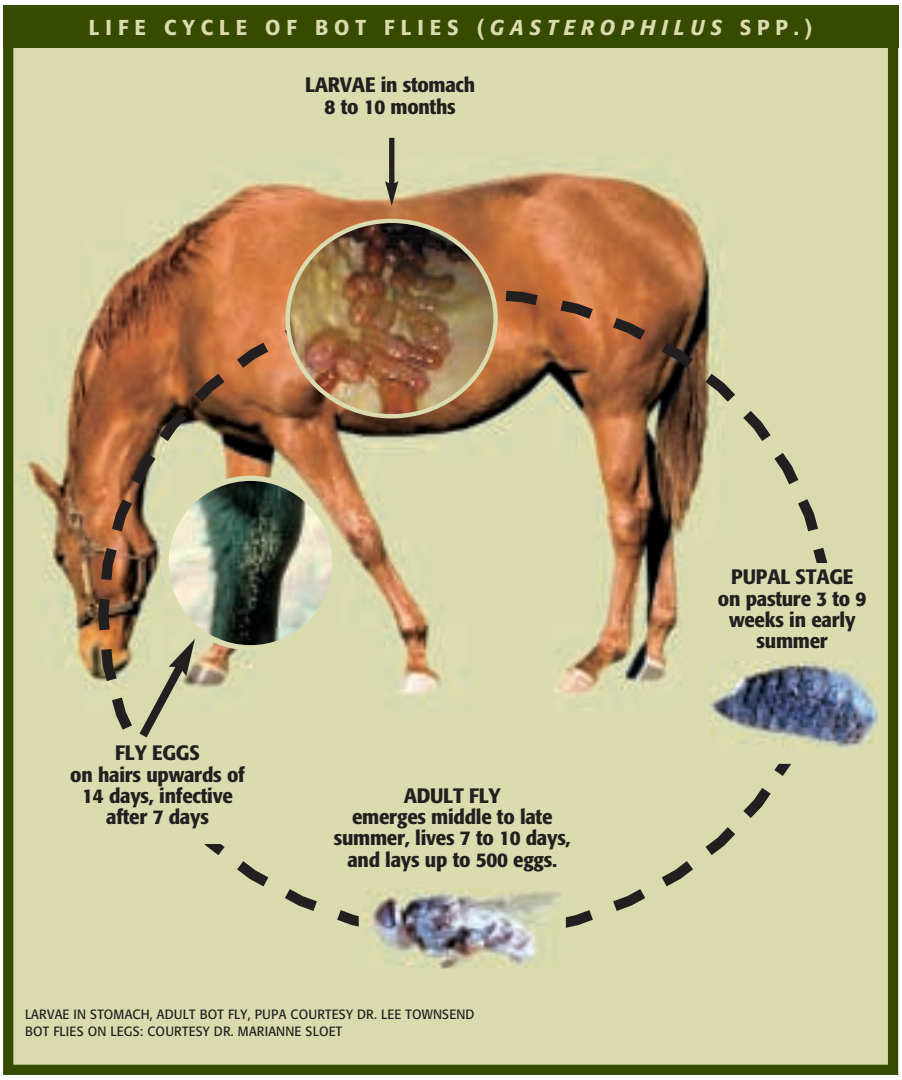
Those tiny yellow bot eggs glued onto your horse's legs, chest, shoulders, and maybe under his chin can hatch and cause health problems in your horse unless you remove them with a sharp blade or bot block.

The Bountiful Bot

Many of us have observed horses in the middle of summer zooming about the pasture in a vain attempt to escape swarms of annoying flies. If you see an insect that looks something like a honeybee buzzing industriously around your horse's legs, chances are it's not a bee but an adult bot fly, looking to deposit her yellow eggs on the horse's leg hairs with the long, curved ovipositor attached to her striped abdomen. Female bot flies are often observed on warm, sunny days hovering near horses and darting in rapidly in an attempt to attach eggs with a sticky "glue" that makes the eggs adhere tightly to the leg.

Bot flies are common virtually everywhere horses are kept, with two major species found in the United States. *Gasterophilus nasalis* lays its eggs on the hairs of the intermandibular space (under the jaw). The eggs hatch spontaneously five to six days after being deposited, and the larvae crawl downward to the chin until they pass between the lips.

In contrast, the females of *G. intestinalis* lay their eggs on the hairs of the forelegs and shoulders of the horse.



Far removed from their destination, the eggs depend on assistance from the horse to find their way into the mouth. Five days after the eggs are laid, they contain first-stage larvae that are ready to hatch rapidly in response to the sudden rise in temperature that occurs when the horse brings its muzzle in contact with them. When an itchy horse rubs and scratches himself with his muzzle and teeth, this allows the larvae to enter the horse's mouth and burrow into the epithelium (skin) on the top of the tongue.

First- and second-stage larvae of *G. intestinalis* spend about a month in the oral cavity. The white first-stage larvae drill burrows that can extend up to five inches (about 13 cm) into the mucosa of the horse's tongue, with air holes about every 0.16 inches (4 mm) through which they breathe. These burrows typically extend in a front-to-back direction along the tongue. The larvae will double in size while they live in the tongue, then will enter pockets in the interdental spaces of the upper molar teeth where they molt from first to second stage.

The second-stage larvae develop a red color due to the synthesis of their own hemoglobin (an oxygen-carrying protein), which is an adaptation necessary to compensate for the low-oxygen environment they will encounter in their migration to the stomach. When they finally leave the interdental spaces, they briefly attach to the root of the tongue before they proceed to the stomach, where they molt to the third larval stage or full-grown bot.

The oral migrations of the other species of bots have not been described in the detail of *G. intestinalis*. However, it is thought that first- and second-stage larvae of *G. nasalis* usually remain completely hidden well below the gum line in interdental pus pockets that extend into the root sockets of the molar teeth. Parasitologists believe that the tissue migration of these parasites helps protect them from the host's teeth, while putting them in a position where they can draw nourishment.

Inside your horse, the larvae of each type of bot have their preferences as to where they set up shop. The red *G. intestinalis* larvae attach in clusters to the non-glandular part of the stomach near the margo plicatus, which is the line of demarcation between the glandular and non-glandular portions of the equine stomach. *G. nasalis* larvae, which are yellowish, are usually found in a small dilatation in the first few inches of the duodenum.

Third-stage larvae attach by their mouth hooks to the wall of the stomach or duodenum for up to 12 months. Prime real estate for both *G. intestinalis* and *G. nasalis* is above the fluid level in the intestinal tract. In these locations, the bots are surrounded by gas pockets that supply them with sufficient oxygen.

As maturity beckons in late spring, the larvae release their grip on the mucosa and pass out with the manure to pupate (mature into adult flies) in the soil. Adult bot flies emerge from the pupae in three to nine weeks depending on the ambient temperature and start the cycle anew. (There is generally only one generation a year.) Bot fly activity continues throughout the summer and fall, but ceases completely with the onset of cold weather.

The Damage Done

While most of us have seen photos of bot larvae lining the intestine or stomach, the oral lesions caused by migrating first- and second-stage larvae might come as a shock. They can be extensive, and they can have an impact on your horse's oral and dental health-to say nothing of the chronic lesions in your horse's gastric and intestinal mucosa.

Yet there is very little evidence associating *Gasterophilus* infections with clinical illness. Most horses can support substantial populations of these parasites without apparent disease. This is not to say that bots don't have an impact on the horse's health; their presence can cause disease too subtle for current detection methods. And particularly heavy *Gasterophilus* infections have been associated with subserosa (membranous) abscesses and gastric ulceration. Some researchers have implicated them in cases of stomach rupture and peritonitis (inflammation of the membrane lining of the abdomen), but others argue that the presence of bots might be coincidental, not causative, in such cases.

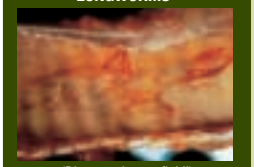
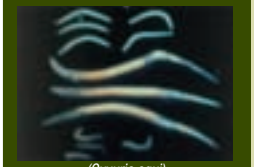
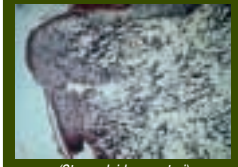
Other Invaders

Stomach worms—Like *G. intestinalis* bots, the spiral-shaped stomach worms

CHARLES MANN

Five days after *G. intestinalis* bot eggs are laid, they contain first-stage larvae that are ready to hatch rapidly in response to the sudden rise in temperature that occurs when the horse brings his muzzle in contact with them. When an itchy horse rubs and scratches himself with his muzzle and teeth, this allows the larvae to enter the horse's mouth and burrow into the epithelium (skin) on the top of the tongue.

LESS-KNOWN INTERNAL PARASITES OF EQUIDS

PARASITE	AGE AFFECTED	SIGNS	TREATMENT
LUNGWORMS  *(Dictyocaulus arnfieldi)*	Any age group (more common in donkeys)	Coughing, lung irritation	Ivermectin
STOMACH WORMS  *(Trichostrongylus axei; Habronema muscae; H. microstoma; Draschia megastoma)*	4 months and up	Loss of appetite, weight loss, poor growth, itchy, persistent sores	Ivermectin
PINWORMS  *(Oxyuris equi)*	6 months and up	Tail rubbing, rat-tailed appearance, weight loss	Broad-spectrum anthelmintics*
THREADWORMS  *(Strongyloides westeri)*	1 to 5 months	Diarrhea	Ivermectin Oxibendazole at 1.5 x label dose

* BROAD-SPECTRUM ANTHELMINTICS—ANY COMPOUND THAT DEMONSTRATES EFFICACY AGAINST FOUR DISTINCT GROUPS OF WORMS: SMALL STRONGYLES, LARGE STRONGYLES, ASCARIDS, AND PINWORMS.

PHOTOS COURTESY FARNAM; LUNGWORM COURTESY PARASITOLOGY SECTION, UNIVERSITY OF KENTUCKY

Habronema muscae, H. microstoma, and *Draschia megastoma* inhabit the glandular portion of the equine stomach, with a special predilection for the margo plicatus. There is also a stomach worm that is more common (and pathogenic) in sheep and cattle called *Trichostrongylus axei,* which horses sometimes contract when they're pastured with ruminants.

Stomach worms are transmitted by house and stable flies, which transfer the larvae when they land on your horse's muzzle, eyes, sheath, or an open wound. Those larvae that enter wounds reach something of an impasse in their life cycles, because they can't access the interior of the horse from there, but they do create granulomas on the wound surface (more on these in a minute). Larvae that are swallowed by the horse eventually make their way to the stomach; *Draschia* are associated with the formation of fibrous nodules riddled with pus-filled cysts, but with the advent of modern dewormers these have become rare in horses.

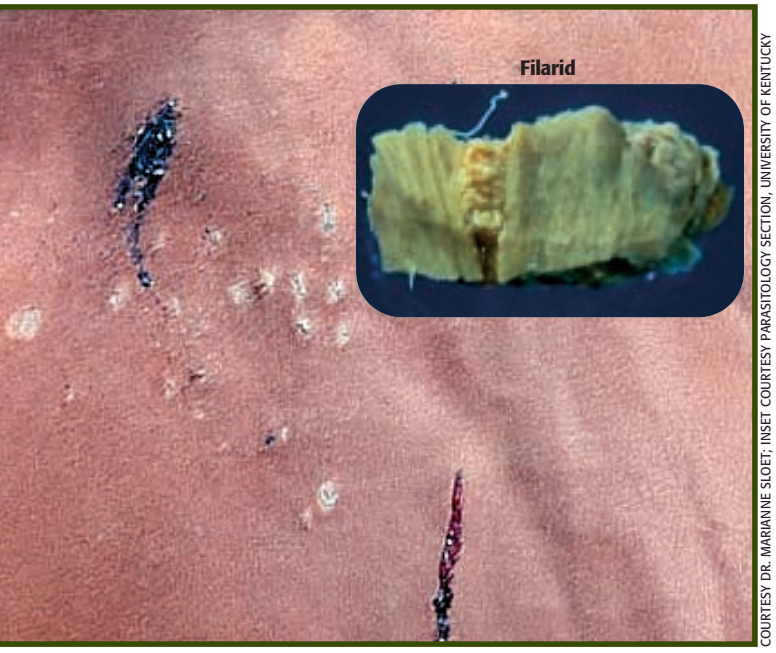
Trichostrongylus species overwinter on pasture, so horses are exposed to them as soon as they are turned out on grass in the spring. The infective larvae die off as the weather warms, and by summer the generation that survived the winter will be gone. But egg production from the new infections rapidly re-contaminates the pasture and continues well into the fall to produce the next season's over-wintering *Trichostrongylus* population.

How significant is the damage done by *Draschia* and *Habronema* stomach worms? Not much, at least not in the stomach. However, their larvae are responsible for a skin condition known as cutaneous habronemiasis, a.k.a. summer sores or swamp cancer. These granulomas develop when the parasites infect minor wounds and moist areas of skin, such as the conjunctiva of the eye, which in pastured horses is often drenched in tears due to stimulation and irritation by flies. Characterized by very rapid production of granulation tissue that refuses to heal during the fly season, the granulomas are also extremely itchy, and secondary trauma often occurs as the horse attempts everything possible to find relief. Granulomas, especially those around the eye, might end up severely ulcerated.

Trichostrongylus axei infection in the horse is most often asymptomatic, but in large numbers it is possible for them to trigger a protracted and debilitating watery diarrhea, especially in stressed individuals.

Lungworms—If your horses share space with donkeys, they might be at risk for contracting lungworms. *Dictyocaulus arnfieldi* is a primary parasite of donkeys that has also found horses to be a passable host, and they can be very pathogenic.

As the common name suggests, this long, white worm hangs out in the respiratory tract. Adults can be up to three inches (about eight centimeters) long. The eggs already contain first-stage larvae when laid, which hatch *before* they're passed out in the donkey's manure. (It is rare for lungworms to successfully reproduce in horses.) The larvae become infective in pasture in about five days, and when they're ingested they migrate by way of the lymphatic system to arrive in the lungs in another five days. Egg laying begins about 28 days after initial infection in the lungs, and the larvae travel up the trachea via coughing. Once in the throat, they're swallowed and make their exit via the intestinal tract.



COURTESY DR. MARIANNE SLOET; INSET COURTESY PARASITOLOGY SECTION, UNIVERSITY OF KENTUCKY

When *Onchocerca cervicalis* larvae (called microfilariae) migrate to the skin, they produce an intense itchy skin reaction (shown above) that horses respond to by scratching and rolling, subsequently leading to redness and hair loss.

Larval lungworms live in the lumen (cavity) of the bronchial tree (the larger air passages of the lungs), where in horses they can cause chronic bronchitis, coughing, and atelectasis—a collapse of the alveoli (air sacs), which can compromise the ability of that part of the lung to exchange oxygen-all while remaining practically undetectable. The lung damage can have a serious impact on any high-performance horse. Interestingly, donkeys can harbor lungworms without any outward sign of disease, but it's in donkeys that the worms can successfully complete their life cycles.

A minor infection of lungworms imposes only a mild burden on the horse. Heavier infections, however, can lead to partial or complete obstruction of the air passages, with clinical disease developing in proportion to the degree of obstruction. These horses might be difficult to distinguish from those with other types of respiratory problems, such as chronic obstructive pulmonary disease (COPD, also known as heaves). The easiest way to distinguish a lungworm infection is to consider the horse's history; if he has been housed with donkeys, lungworms are a real possibility.

Threadworms—The genus *Strongyloides* (commonly called the threadworm) is mostly of concern in foals. The species that infects horses, *S. westeri*, is commonly transmitted from mare to foal through nursing, a mode of transmission with important implications for disease induction and control. Larvae in the tissues of mature mares are induced to migrate to the mammary glands by the hormones of pregnancy and lactation, so they're present in colostrum and in the first few days of milking.

Mares infected with *S. westeri* are asymptomatic, but once the larvae find a home in a foal, they mature rapidly in the small intestine. Within 10 to 14 days after birth, foals begin to shed eggs from the parasite. Diarrhea is a possible sign at this time. These "foal scours" can be coincident with the mare's first return to estrus, and numerous investigators have questioned the correlation between infection with *S. westeri* and foal heat diarrhea, although no definitive connection has been established. Heavy infections in foals can persist for 10 weeks, while lighter infections can last two to three times as long. Fortunately, horses develop immunity to these worms by about six months of age, and egg-shedding is never seen in mature animals.

Pinworms—Most horse people have heard about *Oxyuris equi*, the pinworm, a common and fairly large parasite with a long, tapering tail (hence the name).

Pinworms lurk in the large intestine. Instead of simply discharging their eggs with the horse's manure, the females migrate through the rectum and cement their eggs in masses to the skin around the anus. Each mass consists of a thick yellow or gray fluid, containing up to 60,000 eggs. The eggs develop into the infective stage in four to five days, during which the cementing fluid dries and cracks, allowing for detachment from the skin. The flakes that fall away from the skin adhere to mangers, water buckets, and walls, effectively contaminating the environment of the stable. If a sponge or towel is used to wipe down the horse's perineum following work or grooming and that sponge is used again to clean a bit, transmission has been accomplished for the pinworm. The entire life cycle for these parasites takes up to five months to complete.

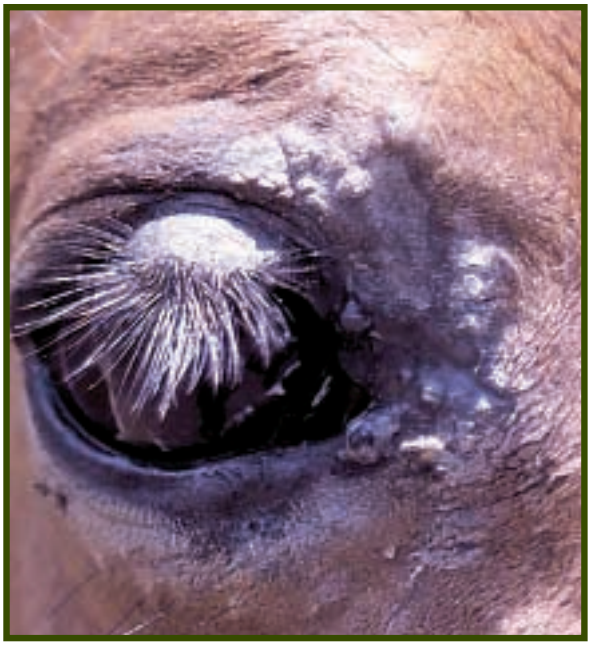
Severe infections with third and fourth stage larvae of *O. equi* can produce inflammation of the cecal and colonic mucosa, resulting in mild colic signs. The most common problem associated with pinworms in horses is the intense itchiness caused by the adhesive egg masses. In an effort to relieve the itching, horses will rub their tail heads against any surface they can find. Numerous horses have lost beautiful tails thanks to pinworm eggs. Cleansing the perineum with paper towels and warm water will give an affected horse some relief.

Filarial parasites—*Onchocerca cervicalis* adults are found in the nuchal ligament of the horse, a very strong, elastic ligament originating at the withers and extending to insert at the poll. *O. cervicalis* adults are large parasites, intricately woven into the deep connective tissues of the nuchal ligament, so it is very difficult to isolate them.

Deep in the ligament, the adults cause little damage. But the larvae (called microfilariae) migrate to the skin, where they produce an intense reaction that horses respond to by scratching and rolling, subsequently leading to redness and hair loss. A skin biopsy is sometimes needed to confirm the diagnosis—but some horses can harbor large numbers of microfilariae with no evidence of skin disease, while in other animals a relatively minor infection can produce dramatic clinical signs.

The distribution of microfilariae within the skin is not random. The highest densities are found along the ventral (lower)

midline, but can also be found in the forelegs, chest, eyelids, and withers. Eventually, they are ingested by insect vectors, most notably *Culicoides* gnats, where they complete their larval development.



COURTESY DR. MARIANNE SLOET

***Draschia* and *Habronema* larvae are responsible for a skin condition known as cutaneous habronemiasis, a.k.a. summer sores or swamp cancer, in which infected minor wounds quickly develop large amounts of granulation tissue and itchiness.**

Dealing With Multiple Enemies

The parasites discussed in this month's article might not be associated with the severe clinical disease that some of the other internal parasites of the horse cause, but they still represent a threat to the overall health of the horse.

Fortunately for horses and horse owners, all of the parasites discussed this month are fairly easy to treat. Most will be killed by treatment with macrocyclic lactones (moxidectin or ivermectin) given at the correct dosage, based upon the weight of the horse.

Ivermectin is highly effective in removing bots from the equine stomach. Bot larvae are the "rate-limiting parasite" for moxidectin, but efficacy against *G. intestinalis* usually exceeds 95% and moxidectin is nearly 100% effective against *G. nasalis*. A dose-limiting parasite defines the dosage necessary to be effective in removal of the parasite. This is the targeted parasite that needs the highest dose for control. If the horse is underdosed by underestimating his body weight, the parasite might not be completely eliminated.

The dermatitis caused by *Onchocerca* microfilariae is 100% responsive to ivermectin at the regular dose. Disappearance of the skin lesions also validates the diagnosis, although complete resolution of the lesions might take up to a month. Midline edema within the first 48 hours after treatment was a common problem when ivermectin was first introduced. This was thought to be due to the acute death of the microfilariae. Relapses might occur within two to eight months because adult *O. cervicalis* continue to live happily in the nuchal ligament. They are not killed by any of the anthelmintics that are available and can continue to produce filariae, making repeat treatments necessary at appropriate intervals (although ivermectin might disrupt their reproductive success).

Summer sores can be frustrating to treat. Ivermectin and moxidectin effectively kill these parasites if they can get into the lesion from the bloodstream, but the amount of fibrous tissue generated around some of these ulcerated areas can preclude good drug penetration. It might be necessary in some cases to prepare a topical concoction of organophosphates, steroid cream, and anti-inflammatory agents as an adjunct to oral administration of either ivermectin or moxidectin. These topical preparations are difficult to keep on the wound and may involve multiple applications to be effective.

Treatment of *Dictyocaulus* (lungworms) and *Strongyloides* is best accomplished with macrocyclic lactones. Attempts to block transmission of *Strongyloides* by treating mares on the day of parturition with ivermectin have not been successful. However, the number of *S. westeri* eggs passed in the feces of foals from treated mares was significantly lower than those of foals from control mares. (Moxidectin is currently not labeled for use in foals younger than six months.)

Numerous anthelmintics are effective against pinworms. Remember that the adults are really not the problem with these parasites; it is the sticky egg mass laid by the female in the perineal area that produces the most visible clinical signs. Benzimidazoles, pyrantel salts, and macrocyclic lactones all have demonstrable activity against *Oxyuris equi*.

DIAGNOSIS

Examining the Evidence

PARASITE PRIMER—PART 6

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

How do you really know if your worm control program is working? If your horses are looking good, are they doing as well as they could be? If they are not doing as well as you would like despite frequent deworming, is the problem due to worms or to something else? How can you really tell?

The truth is that few horse owners know for sure if they are properly controlling worms in their horses. Most people equate treating a horse with dewormer on a regular basis with providing adequate control. Unfortunately, this is almost always not the case.

Why? Largely because high levels of drug resistance, present throughout North America, render treatments on many farms relatively ineffective. To further complicate matters, the biology of the parasites we need to control has changed over the years—so the commonly used every-eight-week treatment program many of us subscribe to often fails to adequately control worms even when drug resistance is not a major problem.

KIM AND KARI BAKER

New recommendations for parasite control include examining the feces of horses to determine worm egg counts. Money spent on proper diagnosis will lead to less money spent on dewormers for horses that don't need them (or need them less often), less drug resistance, and thus better control of worms.

Another important issue is the way we view our treatment programs. Most horse owners treat all horses with the same regimen, which has been taught in the past. The reality, however, is that worm demographics might not be the same from horse to horse. Studies have demonstrated that about 20-30% of your horses harbor about 70-80% of all the worms (see page 25). Some horses carry extremely high worm burdens, while other horses—for reasons we still don't fully understand—have strong immunity and are infected with fewer worms.

Knowing this, does it really make sense to deworm all of our horses the same way? The answer is a definitive no. And new recommendations for worm control in horses (which will be detailed in a later article) are based on treating some horses more and others less. A "selective" deworming program will result in fewer treatments given (leading to less drug resistance), but if used properly will still result in better overall parasite control.

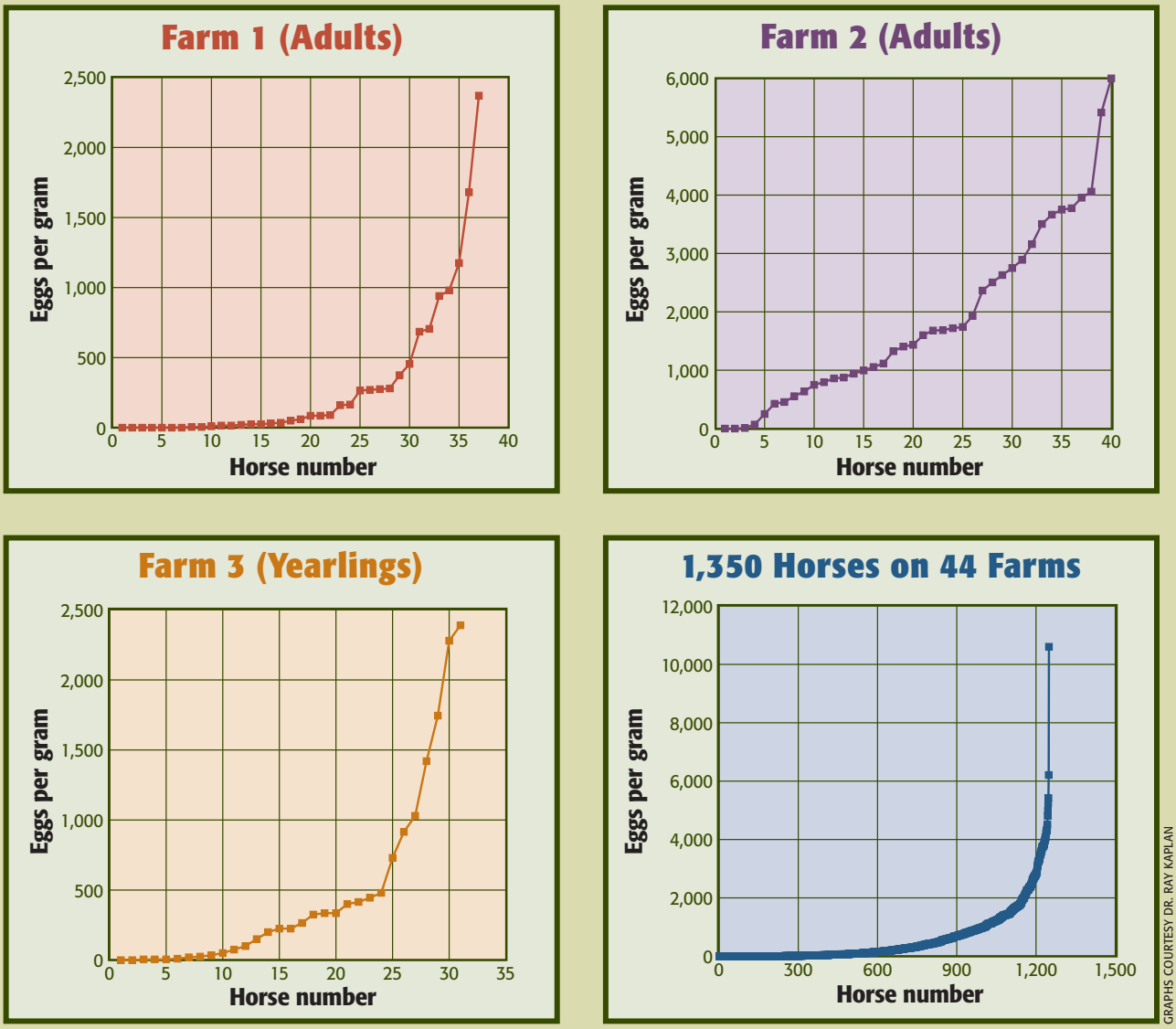
How do we know which horses to treat and not to treat? The answer is simple—we must examine the feces of horses to determine worm egg counts. Money spent on proper diagnosis will lead to less money spent on dewormers, less drug resistance, and better control of worms. Doesn't it make sense to control worms using a medically based approach that considers the needs of individual patients, rather than a recipe-based approach that treats all horses the same?

Eggs by the Dozens

Fecal examinations are done much less frequently than they ought to be, and when they are performed, most times they are only qualitative in nature, meaning that they merely demonstrate the presence or absence of parasite eggs. This can be helpful on occasion, but most often does not yield useful information. For example, small strongyle eggs account for at least 95% (and up to more than 99%) of all worm eggs passed in the manure of adult horses. Essentially, then, we can assume that all horses which have the opportunity to graze are infected with small strongyles. The presence of their eggs in a fecal sample doesn't tell the whole parasite story. Other worm eggs are less commonly seen, and when present, are usually present in low numbers.

Instead of just determining if eggs are there or not, fecal examinations should be quantitative, meaning that an actual number of eggs per gram of manure is determined. Quantitative techniques require that a standard amount of feces be tested, that the quantity of flotation solution is measured, and that a standardized volume of the mixture is examined under the microscope for counting the worm eggs

DISTRIBUTION OF FECAL EGG COUNTS IN HORSES



GRAPHS COURTESY DR. RAY KAPLAN

These graphs show the distribution of fecal egg counts (FEC) in horses on three farms in Georgia (Farms 1-3) and all horses on 44 farms in Florida, Georgia, Kentucky, and Louisiana. Farms 1 and 2 only had adult horses, farm 3 had only yearling horses, and the combined graph represents horses of all ages. Each colored square represents the FEC of a single horse, which is read on the Y axis. Note that in each case the distribution of FEC in the stable is the virtually the same. The shape of these graphs shows the aggregated nature of parasite infections, where a small percent of the animals harbor most of the parasites. In yearlings, because many have not yet reached their immune potential, the shape of the graph is a little less steep.

This aggregated pattern of parasite distribution among animals is always seen. The only thing that changes is the magnitude of the parasite level. From these graphs it is obvious that some horses need much more attention to worm control than do others. Therefore, only by performing FEC can worm control programs be optimized to achieve the desired level of control.

—Ray Kaplan, DVM, PhD

therein. Fortunately for the long-suffering soul squinting through the microscope, a quantitative test doesn't need to enumerate the whole spectrum of parasite clues; it's sufficient to quantify just the strongyle and ascarid eggs, which are the primary targets of worm control programs. Numbers of eggs of other worms, including tapeworms, are less informative, and typically are just noted as being present or absent.

Determining the number of small strongyle eggs being passed in the manure is the only reliable means of estimating the number of adult small strongyles infecting a horse. In young horses (less than 15 months of age), eggs of ascarids (*Parascaris equorum*) can also be present in large numbers.

Results of fecal egg counts (FEC) can be evaluated to:

- Identify the animals in need of the most intensive control measures;
- Assist in stable-wide parasite management decisions;
- Determine if the drugs you're using are effective; and to
- Monitor the success of your worm control program on an ongoing basis.

Checking FEC on a regular basis is the single most important thing you can do to improve your parasite control program. Future articles in this series will address how to interpret and use the results of an FEC to keep your anti-parasitic efforts at their peak, but first, let's discuss how a FEC is performed. This can be done in a lab or in your home with the right equipment.

MCMASTER METHOD

PHOTOS COURTESY DR. RAY KAPLAN

The preferred quantitative method for performing FEC is the McMaster method. A McMaster FEC is a fairly easy procedure to perform, and it is something that individual horse owners can do provided they receive adequate training and obtain the necessary equipment and supplies. Top photo: Materials required; bottom photo: McMaster's egg counting slide.

D.I.Y. Egg Counting

The preferred quantitative method for performing FEC is the McMaster method. A McMaster FEC is a fairly easy procedure to perform, and it is something that individual horse owners can do provided they receive adequate training and obtain the necessary equipment and supplies. Alternatively, veterinarians and local diagnostic labs often will perform quantitative FEC if the service is requested. If this service is not offered, ask your veterinarian why not, and encourage the vet's lab to provide this valuable test. The procedure for performing McMaster FEC is explained in detail shortly and an outline of the procedure is given in "Modified McMaster Egg Counting" on page 27.

Fecal worm egg examination methods are based on the principle of differential density. In other words, parasite eggs sink in water, but they will float in various chemical solutions that are more dense than water (technically, they have a higher specific gravity) because the eggs are lighter than the fluid used as a flotation solution. Ideally, you want the manure to sink, and the eggs to float.

The most common solution used for fecal flotation is saturated sodium nitrate, which is inexpensive, stable, and non-toxic. You probably can arrange to buy some from your veterinarian or a pharmacy. Zinc sulfate is also available commercially and can be used for the same purpose. Theoretically, one could use a concentrated solution of many other common compounds such as sugar, table salt, or Epsom salts. However, saturated sodium nitrate is the preferred solution and can be purchased pre-made or easily prepared using the recipe provided in "Saturated Sodium Nitrate For Flotation and McMaster's Techniques" on page 27. One quart of flotation solution is sufficient for about 30 McMaster examinations.

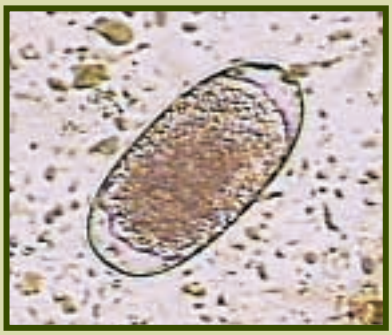
The first step is to collect freshly passed manure balls that are uncontaminated by soil or bedding. (Scavenging manure dropped on concrete or rubber mats, as your horse stands in the cross-ties, is one way to achieve this.) A Ziplock bag can be inverted over the hand and used to pick up one or two fecal balls. Seal the bag and label it with the name of the horse and the date of collection. Fresh samples work best, but accurate results can be obtained up to seven days after collection if the sample is kept refrigerated during the interim. (Do warn your friends and family if you're going to store manure in the fridge, lest they think you've finally lost it!) If samples are not refrigerated, the eggs will hatch within 12 to 24 hours. Once hatched, they cannot be counted.

Just before preparing the sample, the feces should be mixed thoroughly by kneading the bag or by stirring the contents with a wooden tongue depressor or other flat utensil.

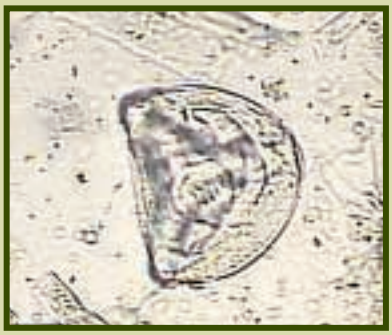
The next step is to obtain *measured* amounts of manure and flotation solution. The solution can be measured easily with a syringe or a graduated cylinder, but

WHAT TO LOOK FOR

Ascarid eggs are round and about 90-100 microns long. They should be counted separately from the strongyle eggs.



Strongyle eggs are oval-shaped and about 90 microns long.



Tapeworm eggs are D-shaped, and are not counted in the fecal egg count procedure (which focuses primarily on strongyles and ascarids).

IMAGES COURTESY DR. RAY KAPLAN

unless one has a postal scale or laboratory balance, it could be difficult to weigh the desired quantity (four grams) of feces accurately. Fortunately, both measurements can be made simultaneously using items that veterinary practices discard on a daily basis. Empty syringe casings from 35- or 60-milliliter syringes work very well for this purpose. Ask your veterinarian to save a few for you. Small translucent plastic cups also work well.

Using a large syringe, add 26 mL of tap water to the empty casing or cup, then use a permanent marker to make a ring around the casing at the top of the water line. Then, using the syringe again, add another 4 mL of tap water and make a second line at the 30 mL mark.

Now you're ready to perform a quantitative egg count.

1. Add flotation solution (saturated sodium nitrate or zinc sulfate) to the first line (26 mL) on the calibrated cup or syringe casing.
2. Next, add small quantities of feces until the fluid level rises to the second line. (The intent is to add four grams of feces, and since one mL of water weighs 1.0 grams, the fluid displacement is a reasonable approximation of weight.)
3. Pour the mixture into a disposable paper cup or a container that can be washed between samples and mix thoroughly with a tongue depressor or flat utensil.
4. An optional—but highly recommended—step is to filter the fecal solution through two layers of cheesecloth or a tea strainer. This step removes the larger particles and makes the sample much cleaner and easier to read under the microscope.

   The next step requires a calibrated apparatus known as a McMaster's slide (see page 26). These can be purchased commercially (see "McMaster Grid Sources" on page 28 for sources), are extremely durable, and they will last a lifetime. A McMaster slide has two chambers, with a calibrated grid superimposed above each chamber. The volume under each grid is 0.15 mL, so if one counts eggs in both chambers, the volume examined is 0.3 mL, or 1% of the total fecal mixture (30 mL).
5. Mix the solution well (remember the eggs are in the flotation fluid and will start to float as soon as the mixing stops).
6. Immediately draw up about one mL of the fecal mixture into a disposable plastic pipette or a small syringe (empty one-mL or three-mL syringes work well).
7. Inject the liquid in the pipette or syringe into both chambers until the area under each grid is filled with liquid. If you get any large air bubbles, you will need to

suck the solution back out and refill the chamber.

8. Wait one or two minutes, then examine under a mechanical stage microscope (which allows the slide to move around) at 100X total magnification (10X ocular lens and 10X objective lens). If the lines in the grid are in focus, you are in the appropriate focal plane to see any parasite eggs that will float to the top of the liquid interface. It is critical that the slide be examined in the correct focal plane. If you are not looking at the top surface of the liquid where the floating eggs are found, you will not see the eggs. You can be sure you are doing this correctly if you see little black circles—these are microscopic air bubbles and are almost always present. These bubbles will float and be in the same place as the eggs. Start at an outside corner of the grid and stay between the lines so you can count the entire chamber without missing individual eggs or counting them twice. At 100X the field of view through the microscope will take up an entire column width of the grid. You will see just the edges of the vertical grid lines on the sides of the field of view. Go down one column and over and up on the next and so forth until you have counted all the eggs in the six columns of the grid. Then repeat on the second chamber grid.
9. When finished, rinse the slide and syringe casing thoroughly with warm water and you're ready to check the next horse. Do not let the slides soak in soapy water for extended periods—this causes them to become cloudy. Instead, rinse them right away with warm water and, if needed, only soak in soapy water for 10 minutes or less before rinsing again.
10. The number of eggs counted should be multiplied by 100. (Because you've examined 1% of the sample, each egg observed within the grid actually represents 100 eggs in the entire mixture.) But because you started with four grams of feces, you must divide this number by four to report the results correctly in eggs per gram (EPG). You'd get the same numerical results if you just multiply the number of eggs counted by 25.

What this means in practical terms is that for every egg you see on the McMaster slide (within the grid area only), there are 25 eggs in one gram of feces. Therefore, if feces have fewer than 25 EPG, the result will be negative (no eggs seen). This does not mean that the horse is not infected, only that infection is at a very low level. As

FECAL EGG COUNT PROCEDURE

Modified McMaster Egg Counting

Materials:

McMaster's egg counting slide*, saturated sodium nitrate**, graduated beaker, tongue depressor, balance, cheesecloth, disposable plastic pipette, paper towels, compound microscope.

Notes:

- A fresh fecal sample should be collected and it should be kept refrigerated until tested.
- A kit containing all needed supplies is available from Chalex Corp.

Procedure:

1. Weigh out 4 g of feces in a small beaker or paper cup.

2. Add 26 mL of sodium nitrate flotation solution (to bring the volume up to 30 mL) to feces. Mix well.

- *Note: If you do not have a scale, you can add feces to the 26 mL of solution and when the volume reaches 30 mL, you have added 4 g.*

3. Strain through one or two layers of cheesecloth (or tea strainer), mix well.

4. Immediately withdraw about 1 mL of the suspension with a pipette or syringe and fill both counting chambers of the McMaster slide. Work quickly, stirring as you draw up fluid. Let the slide stand for one to two minutes to allow eggs to float to top. If visible air bubbles are present, remove the fluid and refill.

- *Steps three and four should be done at the same time without letting the sample sit between steps, since eggs are in flotation fluid and will immediately begin to rise to the top of the fluid. You want to be sure to get a representative sample of the mixed solution.*
- *Once chambers are filled, step three can be started for the next sample.*
- *Once filled, the chambers can set for 60 minutes before counting without causing problems. Longer than this and drying/crystal formation can begin.*

5. Count all eggs inside of grid areas (only count the eggs which have more than half of their area inside the grid) at 100x total magnification (10x ocular lens and 10x objective lens). Focus on the top layer, which contains the very small air bubbles (small black circles). Count both chambers. Count only strongyle eggs (oval-shaped, about 90 microns long; see page 26). Ascarid eggs (round, about 90-100 microns long) can also be counted, but should be counted separately from the strongyle eggs. Do not count strongyloides (oval, about 50 microns long), tapeworm eggs (D-shaped), or coccidia (various sizes)—only notations are made as to the presence of these other parasites.

6. Total egg count (both chambers) x 25 = eggs per gram (EPG).

- *Each chamber has a depth of 1.5 mm and holds a volume of 0.15 mL. Two chambers hold 0.3 mL of fecal mixture, which is 1/100th of the total volume of 30 mL. Therefore, the number of eggs counted must be multiplied by 100. However, since you began with 4 g of feces, to yield eggs per gram, you must instead multiply by 100 and divide by 4 (or multiply by 25).*

**** McMaster Grid Sources***

Chalex Corporation, 5004 228th Ave. S.E., Issaquah, WA 98029-9224; 425/391-1169; fax 425/391-6669; chalexcorp@att.net; www.vetslides.com.
Focal Point, PO Box 12832, Onderstepoort, 0110, South Africa; +27 12 329-1210; www.mcmaster.co.za; e-mail eddy@icon.co.za.

*****Saturated Sodium Nitrate For Flotation and McMaster's Techniques***

- Sodium nitrate (laboratory grade), 400 grams
- Hot water, 1,000 mL
- Heat with stirring until boiling, then let cool at room temp. Store at room temperature—do not refrigerate as additional solute will precipitate.
- Specific gravity should be 1.20 to 1.25. This can be checked with a hydrometer. If specific gravity is too high, additional water can be added.

Note: *Fecal flotation solutions are also commercially available and can provide more accurate results.—Karen Briggs, with Craig Reinemeyer, DVM, PhD; Denny French, DVM; and Ray Kaplan, DVM, PhD*

we mentioned before, if a horse is eating grass, then he is—without exception—infested with small strongyle worms!

You'll probably have to spend a little time with your veterinarian's lab technician to learn to identify strongyle and ascarid eggs confidently, but this is not a difficult task (see images on page 25).

Those Elusive Tapeworms

Although fecal examination is excellent for uncovering the presence of ascarids and strongyles, it is a poor method for detecting tapeworm eggs. The reason for this is that tapeworm eggs are not released from the worms the same way they are from roundworms. Instead of continuously shedding eggs as do roundworms, tapeworms intermittently release segments of the worm that contain the eggs. The eggs are then released into the manure as the segment disintegrates. This results in both uneven shedding of eggs and uneven distribution of eggs within feces. Therefore, it is quite common to *not* find eggs in a fecal examination of a horse which is actually infested with tapeworms—this tends to make it difficult for a veterinarian to prove that a given horse is harboring these parasites.

However, if many horses sharing a pasture are tested, the chance of finding eggs increases. And if one infested horse turns up, chances are that his companions are infested, too.

Because tapeworm eggs are so difficult to find in feces, a method such as the McMaster, which only examines a small percent of the total sample, is not the method that should be used. Instead, a large amount of feces (at least five grams) should be examined using a procedure that concentrates the eggs by using a centrifuge. Theoretically, this procedure can detect one egg in five grams of feces, in contrast to the McMaster method that can only detect 25 or more eggs in one gram of feces. (Needless to say, you'll probably want to rely on your veterinarian for this test, unless you're in the business of collecting and shipping semen and have a centrifuge at your disposal.)

In 1995, researchers in the United Kingdom developed a test that could detect antibodies to the tapeworm *Anoplocephala perfoliata* in the blood of horses. A positive result indicated exposure to tapeworms, but not necessarily a current infection with adult worms. This test was used in a survey in the United States, as described in an earlier article in this series (see www.TheHorse.com/emag.aspx?id=4917). The U.K. test ultimately was refined so the results could be correlated to worm numbers. Unfortunately, this test is not readily available for clinical purposes at the present time.

ANNE EBERHARDT

Next issue, join us for a discussion of your horse's environment and how it contributes to his parasite load, as well as what you can do to reduce the impact!

TESTING YOUR HORSE

How Often Should You Test?

How often should fecal egg counts (FEC) be performed? The answer is not straightforward and will vary from farm to farm. When FEC monitoring is first introduced to a stable, many more samples will need to be tested than later on when your program is established.

To start, all drugs used on a farm should be tested to determine their efficacy. This can be done with one drug at a time, about two weeks after administration. More details on how to do this will be included in our future article on anthelmintic resistance in the September 2004 issue.

Once you know which drugs are doing the best job of keeping egg counts low, FEC should be performed on all horses before the next scheduled deworming with that drug. This will tell you how well your worm control program is working. The reappearance of eggs in the manure will generally take about four weeks if you've used pyrantel or one of the benzimidazoles, six to eight weeks if you've used ivermectin, and 12-16 weeks with moxidectin. These numbers will also depend on the horse—youngsters and those carrying very heavy parasite loads will generally have eggs reappear more quickly.

You can also use FEC before a scheduled deworming to determine which horses need treatment. Some horses in your herd might have low FEC (less than 100 eggs per gram, EPG) and probably don't need to be dewormed. Other horses will have high (greater than 500 EPG) or very high counts (greater than 1,000 EPG), which suggests the interval being used for treatments is too long for these horses. Doing a fecal count first might cut down on the number of tubes of dewormer your farm goes through and help fight the battle against drug resistance.

Performing FEC at frequent intervals in this manner during the first year or two (at every scheduled deworming) will show you some important things that will enable you to improve your worm control, including:

- Which drugs are effective on your farm;
- Which horses tend to always have low FEC (require less frequent deworming);
- Which horses tend to always have high FEC (require more frequent deworming);
- The interval between treatments that is required for the anthelmintics you're using (this may vary from drug to drug); and
- Are fecal egg counts getting lower over time, indicating that worms are being controlled better than when the program was started?

Once you've established an effective routine on your farm, the need for frequent FEC will be reduced because you'll have identified patterns in your herd. Thereafter, you can probably get by with only periodic monitoring.—*Karen Briggs, with Craig Reinemeyer, DVM, PhD; Denny French, DVM; and Ray Kaplan, DVM, PhD*

ENVIRONMENT

Development and Persistence of Parasites

PARASITE PRIMER—PART 7

BY KAREN BRIGGS, WITH CRAIG REINEMEYER, DVM, PHD; DENNIS FRENCH, DVM, MS, DIPL. ABVP; AND RAY KAPLAN, DVM, PHD

Back in January, in our first installment of this series, we described what makes a parasite different from other kinds of infectious organisms. One of those factors is that the offspring of adult parasites must return to the environment outside the horse in order to become capable of infecting a second animal, or re-infecting the original host.

So while discussing the environment might seem esoteric when we're talking about equine parasites, it's really one of the most critical elements to consider if you're going to construct an effective anti-parasite strategy. Equine parasites must undergo some change away from the horse—so the environment presents unique opportunities for disrupting transmission. That's a tremendous advantage compared with diseases that are directly contagious from one horse to the next.

The key to exploiting environmental events is understanding when and how Mother Nature works—for us or against us—in our perpetual battle against worms.

PAULA DA SILVA

The key to exploiting environmental events is understanding when and how Mother Nature works—for us or against us—in our perpetual battle against worms.

Environmental Factors

So what qualifies as "environment" for your horses? In the case of most domestic horses, the answer would be stalls, pastures, and paddocks. These are the battlefields on which we wage war against our major parasitic enemies—strongyles (bloodworms), ascarids (roundworms), and tapeworms.

Environment in a broader sense includes literally anything outside of the equine host. So, for certain equine parasites such as stomach worms and *Onchocerca* (which we discussed in the May issue), the flies that transmit them also must be considered a part of the environment.

Let's briefly review the changes undergone by each of the three major parasites during their sojourn in the environment.

Strongyles, ascarids, and tapeworms all produce an egg stage that passes from the horse in manure. Ascarids (as we discussed in the March 2004 issue) lay eggs that can develop to the infective stage in two to four weeks. The potential new roundworm is protected within the egg, and it can remain infective for perhaps as long as a decade. Strongyle eggs (see the April 2004 issue) hatch over a wide range of temperatures, and the rate of development varies with thermal conditions. The first-stage larva that emerges from the egg feeds in the environment, and it eventually molts to a

second- (L2) and a third-stage (L3) larva. Only the third stage is potentially infective to another horse.

Tapeworm eggs (February 2004 issue) must be eaten by free-living mites found on pasture. They then develop into the infective stage (cysticercoid) inside the mite. All three parasites are transmitted into your horse when he inadvertently ingests the infective stage while grazing in the pasture.

Microenvironmental Variables

Temperature, moisture, and the availability of oxygen all have the potential to affect parasite development in the environment. Let's take a detailed look at each one.

Temperature—Ascarid and strongyle eggs are capable of developing or hatching over a wide range of environmental temperatures. As a general rule, the rate of development increases in direct proportion to the environmental temperature. Thus, strongyle eggs can hatch at 45°F, but it could take several weeks for them to reach the infective (L3) stage. At 80°F, however, strongyle larvae can become fully infective within five days of eggs leaving the horse.

There are two dynamic processes going on here. The first is development, which is the formation of new infective stages from a reproductive product (egg). The environmental conditions that favor parasitic development are virtually identical to those required for germination of a plant seed. Both respond to the same conditions—they will germinate (hatch) and grow above some critical temperature, but the rate of growth is accelerated in warmer conditions.

Development is only half of the picture for parasite transmission. The other factor is persistence, or how long infective stages survive in the environment. Persistence has a greater impact on the cumulative numbers of parasites acquired, and thus on the likelihood of associated disease (see "The Power of Persistence" above).

The environmental conditions that influence persistence are nearly the opposite of those required for development. Let's return to our gardening analogy. After the successful germination and growth of a tomato plant, the gardener can harvest and enjoy the ripe, red fruits. But what conditions would allow her to preserve that tomato for the longest period of time—or in terms of parasites, what conditions would allow them to persist for the longest time in the environment? If the tomato were stored in a refrigerator, it would remain fresh longer than if it were placed out in the sun.

This analogy applies directly to the infective stages of many common parasites because they persist far longer at cold temperatures than in warm ones. This difference shouldn't be a huge surprise to us because equine parasites probably evolved along with their warm-blooded hosts in temperate, rather than tropical, climates. Accordingly, the environmental stages of most equine parasites are more at home on the steppes of Siberia than in the steamy jungles of India or on the sands of Saudi Arabia.

Let's examine this phenomenon by using strongyles as our model. After a strongyle egg hatches, the first- (L1) and second-stage (L2) larvae feed actively in the environment, ingesting organic material and bacteria from the manure. The third-stage

HOW MANY WORMS ARE OUT THERE?

The Power of Persistence

What's the difference between development and persistence in terms of disease risk? Development essentially describes the numbers of new larvae that appear in the environment, whereas persistence determines how long they stick around.

For example, if 10,000 new larvae appeared on a pasture each day, but they only lived for 24 hours, the maximum daily risk would be 10,000 larvae. The previous conditions could be described as favoring development, but not persistence. Alternatively, if only 1,000 new larvae arrived each day, but they were able to survive for months, it would only take about two weeks before the maximum daily risk would exceed the previous example. Thus, conditions that favor persistence are more important for determining the likelihood of parasitic disease.

In continental North America, conditions during autumn and spring favor both development and persistence. Southern horses face a much greater risk of strongyle infection during winter than in summer. Northern horses at pasture are challenged almost perennially, and stabling them during the winter months might be the only practical respite from strongyle exposure.

Regardless of climatic influences, the most effective strategy is to maintain low fecal egg counts in horses. Eggs turn into larvae, and larvae turn into parasites. Once the cycle has entered the environment, we have few control options other than confining our horses. Therefore, the most logical approach is to minimize contamination with eggs in the first place. Upcoming articles will describe the implementation of this strategy in greater detail.

—Karen Briggs with Craig Reinemeyer, DVM, PhD; Dennis French, DVM, MS, Dipl. ABVP; and Ray Kaplan, DVM, PhD

larvae (L3), however, which are infective, are covered by a membrane that protects them from desiccation (drying out). This flexible coat of armor contains one major design flaw. It has no mouth opening, so an L3 can no longer feed. It must survive by using stored energy reserves. You'll never see a nematode with love handles, because they have no organs of energy storage. Their sole energy reserves are stored within tiny intestinal cells, and those are very limited in quantity. The rate of consumption of those finite energy reserves is directly proportional to environmental temperature, meaning that their energy is used up more rapidly in hot weather. Just as in high school chemistry lab, firing up a Bunsen burner and applying heat often sped up a reaction. So, too, worms burn energy faster at higher temperatures—and they die when their energy reserves are depleted.

At colder temperatures, energy consumption by third stage strongyle larvae is nil. That is why they survive for months in a refrigerator, and they can persist happily all through winter in most North American climates. Remember that these larvae are cold-blooded invertebrates. They aren't killed by freezing, they don't shiver when they get cold, and they don't need any mechanisms to burn energy in order to stay warm. They just take the weather as it comes and expend their energy in response to thermal conditions.

In comparison, ascarids and tapeworms are less susceptible to environmental conditions. Ascarid eggs still need warmth to develop to an infective stage, but a viable egg can survive for up to a decade. Tapeworm eggs might be killed by freezing, but it is certain that their mite vectors only feed actively during the warmer months of the year. This means mites can only ingest eggs (and acquire new infections) during spring, summer, and autumn in most parts of North America. We don't know how long mites can survive once infected, but it is reasonable to assume that they make it through winter conditions just fine.

Moisture—Because larval stages are more dependent on moisture than worm eggs, we'll use strongyles again to explore this environmental variable. Sufficient moisture is usually present in equine manure to support the hatching of strongyle eggs and promote development to the L3 stage if temperatures are warm enough. But when development is slowed, additional moisture might be required to complete development.

Because all grazing animals demonstrate some degree of fecal avoidance behavior (see "Lawns and Roughs" on page 31), larvae have a better chance of becoming successful parasites if they migrate away from manure and onto forage where they will be consumed. Larvae can be disseminated by mechanical disruption of fecal piles, but the most common method is through heavy rainfall (anything in excess of a half-inch). Parasite transmission is hindered by drought, but helped along by frequent precipitation.

After they leave the fecal pile, most strongyle larvae set up shop in the thatch layer on pasture. This is the dense mat of vegetative detritus (debris) at the root level of pasture grasses, directly on top of the soil. The relative humidity within the thatch layer is considerably higher than in other areas of the vegetation, especially during dry weather.

Because of the critical requirement for a moist microenvironment, strongyle transmission occurs almost exclusively on pastures. Infection from stalls or dry-lot paddocks is negligible—clean horse stalls are simply too dry to allow strongyle larvae to develop or persist, and even in a filthy, damp stall, strongyles are unlikely to develop because the ammonia that comes with urine accumulation is extremely toxic to strongyle larvae. Clean and dry or wet and stinky, horses rarely acquire new strongyle infections in confined conditions.

Oxygen—Because parasite offspring only become infective in an aerobic environment, they all require oxygen for development and growth. However, sufficient oxygen is uniformly available in all but a few extreme circumstances. The relatively loose structure of equine manure ensures that air can penetrate to all areas of the fecal pellet, especially as it dries out.

The Bigger Picture

Your horse's relative risk of exposure to parasitic infection varies in different management situations. A few parasites, such as ascarids and pinworms, can be transmitted in stalls, but pastures are the main battleground for strongyle control. And because pastures are affected greatly by climatic variation, horse owners in Manitoba, Canada, face a different set of challenges than those in Florida.

Across North America, spring and autumn present nearly optimal conditions

ANNE EBERHARDT

Any third-stage strongyle larvae present on pasture in autumn will probably survive just fine through the whole winter, even under six inches of ice and snow.

SHAWN HAMILTON

Spreading fresh manure on pastures is a common management practice that can have a ruinous impact on your pastures. Equine manure should be composted prior to spreading.

for generating wormy pastures. Average daily temperatures facilitate the hatching of eggs and development of larvae without shortening the life span of infective stages.

In northern temperate climates (roughly above the latitude of the Ohio River), summer conditions are favorable for both development and persistence of strongyles, but the cold northern winters do not support hatching of eggs or development of larvae from November through March. Despite that, any third-stage larvae present on pasture in autumn will probably survive just fine through the whole winter, even under six inches of ice and snow. In fact, rested pastures can be an important source of strongyle infection for northern horses when they are first turned out in the spring. The larvae that successfully over-wintered eventually die off, but not until early summer when rising daily temperatures (with the energy expenditure they trigger) do them in.

In southern temperate regions, strongyle eggs hatch very rapidly during summer, but the high daily temperatures are unfavorable for larval survival. The infectivity (numbers of available infective larvae) of southern pastures is at its lowest level of the annual cycle during summer. Southern winters, on the other hand, are cool enough to promote larval persistence, but strongyle eggs can also hatch and develop slowly into new larvae whenever daily temperatures rise above 45°F.

A few years ago in Tennessee, a study monitored the numbers of available larvae on pastures occupied by horses (at approximately one horse per acre) enrolled in various parasite control programs. On the pasture grazed by untreated horses, larval numbers during summer never exceeded 2,000 per kilogram of forage. However, worm numbers increased during autumn, and more than 60,000 larvae per kilogram of forage were recovered during December.

Some aspects of pasture usage are determined by the horses themselves. Left to their own devices, grazing horses segregate pastures into two distinct areas, which have been described as roughs and lawns. Roughs are areas of taller forage, where horses preferentially defecate, but do not graze. The lawns are areas of shorter forage that have been grazed down by feeding horses and are characterized by a relative paucity of manure. In the 1980s, Dr. Rupert Herd of The Ohio State University demonstrated that the levels of strongyle infectivity (numbers of available larvae) were about 15 times higher in roughs than in lawns. So, by not grazing in the roughs, horses naturally practice a behavior that significantly reduces their exposure to parasite infection!

However, we humans are irresistibly compelled to tame our environment,

ANNE EBERHARDT

With a greater density of horses on your pastures, more parasites will be shed that can further infest them. Also, more horses eat more grass, forcing horses to graze closer to parasite-rich defecation areas.

HOW HORSES MANAGE THEIR PASTURES

Lawns and Roughs

All grazing animals practice some degree of fecal avoidance behavior. (Ever seen a new father holding a dirty diaper at arm's length? This response is apparently exhibited by some non-grazers as well.) If you look at an occupied cattle pasture, for instance, you'll see numerous tufts of tall grass distributed at irregular intervals over the entire field. Closer inspection reveals a fecal pat at the base of each of these tufts. The grass in these clumps is not tall because it's being fertilized by the manure, it's high because it's not being grazed. Cattle avoid the manure as well as the grass growing around and through it. Horses do something similar, but they don't defecate as indiscriminately as cattle. Equids deposit their manure in distinct areas known as roughs, and subsequently avoid grazing in those contaminated patches. The closer-clipped areas they do graze are termed lawns.

The behavioral basis of fecal avoidance in horses is unknown, a fascinating phenomenon that awaits further scientific investigation. Why do horses avoid fecally challenged roughs? Is it due to olfactory (smell) cues, visual stimuli, or other, unknown factors? And if one could determine the behavioral basis of fecal avoidance, is it possible that this behavior might be exploited? Could horses be trained to defecate in certain areas of a pasture, perhaps where the forage wasn't so desirable?

In the future, perhaps the equine proclivity for forming roughs and lawns could be manipulated to improve pasture utilization.

—Karen Briggs with Craig Reinemeyer, DVM, PhD; Dennis French, DVM, MS, Dipl. ABVP; and Ray Kaplan, DVM, PhD

ANNE EBERHARDT

Owners should never drag pastures that are currently occupied. For optimal parasite control, pastures should be left vacant for a minimum of two weeks in the south, or four weeks in the north, after dragging in summer months.

including horse pastures, so we use a bush hog to cut down the taller forage. Clipping pastures probably doesn't affect parasite exposure very much, because the horses still know where the manure is concentrated and graze elsewhere. But dragging or harrowing pastures totally disrupts Mother Nature's system for segregating infected from clean areas. Dragging disseminates fecal material, along with its larval passengers, from an isolated area of concentration and spreads it uniformly across the entire pasture. It contaminates the previously pristine lawns with larvae, and exposes horses to more parasites than if the pastures had been allowed to look ragged.

(As a general rule, owners should never drag pastures that are currently occupied. For optimal parasite control, pastures should be left vacant for a minimum of two weeks in the south, or four weeks in the north, after dragging during the summer months. Pastures dragged after Oct. 1 will remain infective until the following July in nearly all regions of the United States.)

Spreading fresh manure on pastures is a common management practice that can have the same ruinous impact as dragging if the manure contains strongyle eggs. Equine manure should be composted prior to spreading. (Look for more specifics on the impact of dragging, spreading manure, and other pasture management techniques in the final two installments of this yearlong series.)

The number of horses on your pasture also has a significant impact on the risk of exposure to parasites. Fecal avoidance is a dominant behavior, but it can be overwhelmed by hunger.

Horses on overcrowded pastures extend their grazing farther into the roughs and thereby presumably increase their risk of parasitism. They also increase their exposure by grazing available forages to ever shorter lengths (remember that the infective larvae tend to be concentrated in the thatch layer at the base of the grass). Part of the reason for high larval numbers on pastures (measured on the basis of a unit weight of forage) during winter months is that grass is often grazed down to the root during winter. In contrast, the lush growth of spring and summer tends to dilute horses' exposure to infective larvae.

In any season, grazing horses should be managed so that forage height remains at a reasonable level, and supplemental hay should be supplied whenever necessary to minimize over-grazing.

Future Directions

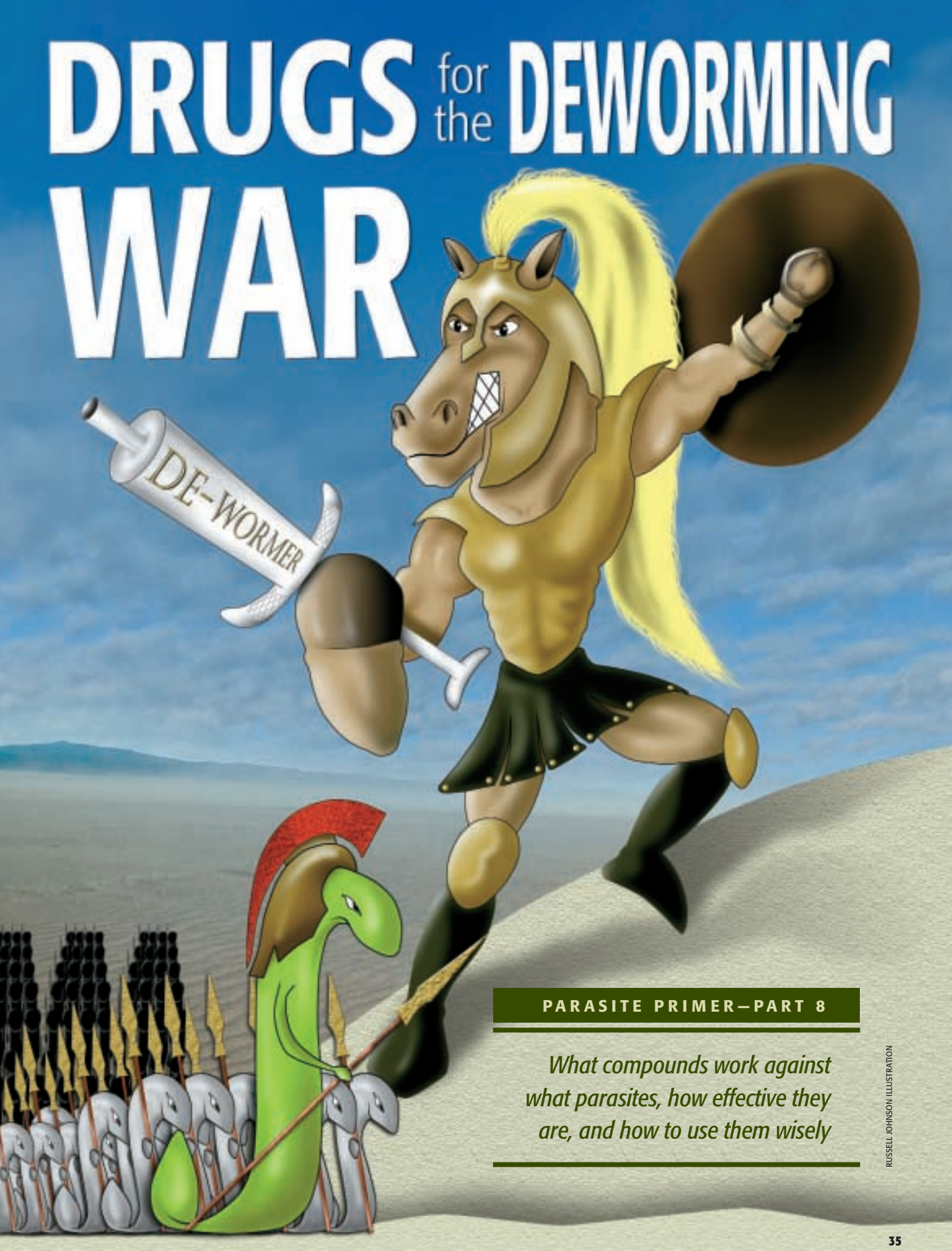
If the environment is so critical in the transmission of equine parasites, why don't scientists just develop some silver bullets that can kill or remove the infective stages from pastures?

It would be wonderful if it were that simple.

Even though parasites with infective egg stages (ascarids and pinworms) can be transmitted in confinement, disinfectants aren't very effective against them. And as we've discussed, strongyles are transmitted almost exclusively on pastures. However, strongyle larvae are far from alone out there. They dwell among a veritable jungle of living organisms, including soil nematodes, free-living mites, beneficial insects, and the pasture grasses themselves. Scientists have not yet discovered any exclusive chemicals or worm-specific viruses that could kill strongyle larvae without disrupting the entire ecosystem. You don't want to kill off the good "bugs" along with the bad ones. So for now, adopt some of the management controls discussed here.

A future installment of this series on parasites will discuss the recent developments that hold some promise for environmental control. But for the time being, the most common approach is the appropriate use of dewormers (which will be discussed next month) to prevent horses from contaminating their environments with worm eggs.

DRUGS for the DEWORMING WAR



PARASITE PRIMER—PART 8

What compounds work against what parasites, how effective they are, and how to use them wisely

RUSSELL JOHNSON ILLUSTRATION

BY KAREN BRIGGS, WITH CRAIG REINEMEYER, DVM, PHD; DENNY FRENCH, DVM; AND RAY KAPLAN, DVM, PHD

The shelves at the local co-op or tack shop, and the pages of your animal health product catalog, all feature a bewildering array of dewormers. If you feel confused every time you have to make a decision about anthelmintic selection, it's no wonder. How do you choose?

In order to make an informed decision, you need to know something about the properties of the drugs. How do they act once they're inside your horse? What about safety and efficacy? Here's a primer on the active ingredients in various dewormers that are currently available.

How Do They Work?

All nematocides (drugs that kill nematode or roundworm parasites) essentially kill worms by either starving them to death or paralyzing them. Because worms have no way of storing energy, they must eat almost continuously to meet their metabolic needs. Any disruption in this process results in energy depletion, and interfering with feeding for 24 hours or less is sufficient to kill most adult parasites.

Parasites will also die if they become paralyzed and temporarily lose their ability to maintain their position in the gut.

Although dewormers are sold under many brand names, there are really only nine distinct compounds currently approved for use against equine parasites (see "Dewormers Available in the United States" below right). These nine drugs belong to only five different chemical classes. Because all drugs in a chemical class kill worms by the same mechanism, we'll use a chemical classification system to discuss each dewormer in greater detail. For a translation to the more familiar brand name, see the table.

Benzimidazoles

Benzimidazoles interfere with a worm's energy metabolism on a cellular level. They bind to a specific building block called beta tubulin and prevent its incorporation into certain cellular structures called microtubules, which are essential for energy metabolism. This is analogous to treating bricks so they won't bind to mortar; walls constructed with such materials would be unstable and ultimately collapse.

Interfering with energy metabolism is a much more basic mode of activity than those of the other classes of dewormers. For this reason, the benzimidazoles are also able to kill nematode eggs, a property not shared by other classes of dewormers.

Benzimidazoles are available in granules, paste, and suspension formulations. As a group, they're more effective when given several days in a row. This property has been exploited in the Panacur PowerPac, which has a five-day treatment regimen for efficacy against migrating large strongyle larvae and encysted cyathostomes.

PAULA DA SILVA

Nearly all of the equine anthelmintics currently marketed could be termed "broad spectrum," meaning they kill a broad variety of parasites, rather than targeting a single species. For horses, broad spectrum products kill large strongyles, cyathostomes (small strongyles), ascarids, and pinworms.

Tetrahydropyrimidines

The tetrahydropyrimidines (pyrantel pamoate and pyrantel tartrate) mimic the activity of acetylcholine, a naturally occurring neurotransmitter that initiates muscular contraction. The action of acetylcholine on nerve cells is usually brief and temporary because it is degraded by a specific enzyme known as acetylcholinesterase. Pyrantel salts induce the same muscle-contracting effect as acetylcholine, but the resulting contraction is irreversible. This results in rigid paralysis, so the worm is unable to feed and quickly starves. Pyrantel has the fastest activity of any of the major deworming classes used in horses, but worm populations tend to bounce back quickly from it—strongylid fecal egg counts are suppressed for only about four weeks. Also, pyrantel salts only affect adult parasites—they don't hurt the larval stages.

Pyrantel pamoate is available in suspension and paste formulations, while pyrantel tartrate is formulated in alfalfa pellets and can be fed daily at 1.2 mg/pound daily for prevention of strongyles and ascarids, or used at a higher dosage as a single treatment to remove existing worms.

Heterocyclic Compounds

Several dewormers are classified as heterocyclic compounds, but piperazine is the only one used in horses. Piperazine works by depolarizing muscular membranes, which renders them resistant to the action of acetylcholine. Worms affected by piperazine are paralyzed and cannot feed properly. The action of piperazine is limited to adult parasites, so it is likely that fecal egg counts would remain low for only a limited time following treatment with this drug.

Although it was popular a few decades ago, today piperazine is used infrequently in horses. It is available as a liquid or powder formulation, and it must be administered in voluminous doses that usually require nasogastric intubation. There is also a pelleted form that can be fed as a supplement.

Macrocyclic Lactones

The macrocyclic lactones act on very specific sites in nematode nerve and muscle cells, disturbing the normal transmission of nervous stimuli to muscles. The result is flaccid paralysis, with an inability to feed or swallow nutrients. Macrocyclic lactones are the most potent killers of worms, being effective at less than one-tenth the dosage of other classes of dewormers. They also have the unique quality of killing external parasites, such as lice, mites, and the cutaneous (inhabiting the skin) larvae of *Onchocerca,*

DEWORMERS AVAILABLE IN THE U.S.

DRUG CLASS	GENERIC ANTHELMINTICS	BRAND NAMES
Benzimidazoles	Fenbendazole	Panacur; Safeguard; Panacur PowerPac
	Oxfendazole	Benzelmin
	Oxibendazole	Anthelcide E.Q.
Heterocyclic compounds	Piperazine	Piperazine
Macrocyclic lactones	Ivermectin	Zimecterin, Rotation 1, Ivercare, Equell
	Moxidectin	Quest, Quest Plus, ComboCare
Tetrahydropyrimidines	Pyrantel pamoate	Exodus; Pyrantel Pamoate Paste; PSI's suspension; Strongid-T; Strongid Paste
	Pyrantel tartrate	Strongid-C; Strongid-C 2X; Continuex; Continuex 2X
Isoquinoline-pyrozines	Praziquantel	Equimax; Quest Plus; Zimecterin Gold

Habronema, and *Draschia*.

But macrocyclic lactones also have a down side—they don't act as quickly as other deworming drugs. Three to four days is required before strongylid egg counts reach their lowest level post-worming.

Macrocyclic lactones are available in paste or solution formulations (ivermectin) or as an oral gel (moxidectin).

Isoquinoline-Pyrozines

Praziquantel is the sole member of the isoquinolone class used in horses. It is also unique in that it has no activity against nematodes (roundworms). Praziquantel is effective only against cestodes, or tapeworms (*Anoplocephala spp.*). It acts by disrupting the integument (the worm's outer layer), which renders the worm incapable of maintaining a balanced equilibrium of body fluids and chemicals. Tapeworms killed by praziquantel look very damaged when they eventually pass out in the feces of a horse.

Praziquantel is currently marketed only in combination with macrocyclic lactones, and the combination formulation is that of the parent compound (ivermectin if paste, moxidectin if a gel).

Common Ground

Despite their differences, all anthelmintics for use in horses must be approved by the Center for Veterinary Medicine of the Food and Drug Administration (FDA). Before receiving FDA approval, however, dewormers must undergo rigorous testing to demonstrate that they are safe and effective for the intended uses. FDA guidelines generally require an anthelmintic to remove at least 90% of the target parasites, and they are very strict about the information that appears on the product label.

Nearly all currently marketed equine anthelmintics could be called "broad spectrum," meaning they kill a broad variety of parasites rather than targeting a single species. For horses, broad spectrum means efficacy against large strongyles, cyathostomes (small strongyles), ascarids, and pinworms. Narrow spectrum is anything less.

THEHORSE.COM POLL RESULTS

Do you deworm your horse...

Every three months:	**35.04% (328)**
Every six weeks:	**31.94% (299)**
Other:	**13.57% (157)**
Daily:	**9.94% (93)**
Every six months:	**8.97% (84)**
Never:	**0.53% (5)**
TOTAL VOTES:	**936**

Over-the-counter anthelmintics do not require refrigeration, but can be damaged by excessive heat. Observe the storage conditions recommended on the packaging, and remember that a truck's dashboard can get darned hot in the summer.

Dewormer labels also include an expiration date, after which the stability and concentration of the product can no longer be guaranteed. Expired drugs are not likely to become toxic, but they might become less effective, and their physical properties might be altered. For instance, if your paste is kept too long it might shrivel into cement.

Dewormer Myths

Forty years ago, dewormers were a much different kettle of fish than they are today. More difficult to administer, and with a smaller margin of safety and a narrower spectrum of efficacy than anything available today, those early dewormers should be just a memory. But some of the beliefs that came with them persist; like so many other things in the equine industry, tradition swings a bigger stick than logic.

Let's tackle a few of the more outdated notions head-on.

Myth #1—"The only way to ensure my horse gets dewormed properly is to get my veterinarian to tube him."

False—Giving an anthelmintic via nasogastric tube is still considered to be the only "guarantee" that all of the drug is ingested. But this practice originated strictly because most early dewormers were either very irritating to oral membranes (like carbon disulfide) or required voluminous doses that could not be delivered in any other way (piperazine and combinations thereof). With the modern compounds and formulations, there is no need to use a nasogastric tube for anthelmintic administration.

Myth #2—"Chemicals toxic enough to kill worms must be toxic to my horse, too."

False—In the 1960s and 1970s, a drug class known as organophosphates was used in horses to remove bots and various other internal parasites. At therapeutic dosages, organophosphates could cause physiologic

MORE DEWORMING OPTIONS

Are Generics Just As Good?

The first company to develop a drug generally secures a patent that guarantees exclusive rights to manufacture and market the compound for 20 years. When the patent expires, individuals or companies that can manufacture the same chemical may also pursue FDA approval to market generic versions of the product.

The approval requirements for generic drugs are not as exhaustive as those needed to achieve the original New Animal Drug Approval. A generic manufacturer must accomplish two things. The first is to demonstrate that their version of the compound is chemically identical to the one first approved (known as the pioneer). Proof of chemical identity mostly involves manufacturing issues, which have very stringent regulatory requirements.

The second issue is demonstrating that the generic candidate acts like the pioneer when given to the intended animal species. The preferred method is demonstrating plasma bioequivalence i.e., that both generic and pioneer compounds are metabolized at similar rates and to a similar extent. Plasma bioequivalence was used to test the generic ivermectin pastes that are currently approved for equine use (e.g., Equell, Ivercare, Phoenectin, Rotation 1).

Some drugs, however, are metabolized in a manner that results in negligible or erratic plasma levels. Generic approvals for such drugs can be accomplished by biological end-point bioequivalence testing, which is a modification of the efficacy studies performed initially by the original manufacturer. Bioequivalence is granted if both drugs significantly reduce worm numbers with greater than 90% efficacy. If the calculated efficacy of either compound is less than 90%, bioequivalence can still be granted if the data meet certain statistical criteria. Biological end-point bioequivalence was used to test the generic pyrantel pamoate products currently marketed for horses (e.g., Exodus, Pyrantel Pamoate Paste, PSI's suspension).

If either testing method shows a generic drug to be equivalent to the pioneer, then the generic can receive all the label claims for the same dosage of the pioneer at the time of comparison. Finally, drugs that are solutions (not pastes or suspensions) may require no animal testing, and the generic manufacturer's only burden of proof is chemical identity.

While a patent is in effect, the manufacturer of a drug operates in the absence of competition and can charge what the market will bear. Generic drugs generally can be sold at lower prices, however, because development costs are significantly lower. Although the term "generic" is sometimes used to suggest inferior or inconsistent quality, the chief purveyors of this notion are pioneer manufacturers who hope to maintain their market share. Approval of a generic drug by the Center for Veterinary Medicine of the Food and Drug Administration means, by definition, that it is considered to be equivalent to the pioneer.—*Karen Briggs*

changes in horses as well as in worms. Toxic effects in horses included colic, salivation, incoordination, and muscular twitching. There also was some concern about organophosphates causing abortions.

Today's dewormers are vastly superior to the organophosphates we once used. They have broad margins of safety and are administered to millions of horses worldwide without incident. Today, the major safety issue with deworming is ascarid impactions following treatment of foals, weanlings, and yearlings harboring heavy parasite loads, and there are strategies to treat those horses without triggering that cascade. Killing other types of adult worms is essentially without consequence, as is killing most larval stages in the tissues.

Myth #3—"Dewormers aren't safe to use in pregnant mares."

False—For a drug label to state that the product is safe for use in pregnant mares, the FDA requires the manufacturer to conduct rigorous testing to prove it. With the exception of the three drug combinations that include praziquantel (see "Dewormers Available in the United States" on page 34), all currently marketed anthelmintics are approved for pregnant mares.

The lack of pregnant mare safety claims for the praziquantel combinations is due to their recent entry into the market rather than to any suspected hazards. Because pregnant mare safety testing usually requires a minimum of two years, most pharmaceutical companies will seek initial approval of their drug while waiting on a pregnant mare safety trial. If and when safety is demonstrated, the label can be amended to reflect that fact. In the meantime, the manufacturer is able to sell a product that is not labeled for use in pregnant mares to begin recouping their research and development investment while the patent life is ticking away.

Myth #4—"I can get the same deworming effect without using toxic chemicals on my horse by giving him a safe, natural remedy like diatomaceous earth."

Questionable—Although many herbal and "natural" products are marketed as being effective against worms, none of them are currently regulated by the Centers for Veterinary Medicine. As a result, they are not subject to the same FDA restrictions regarding label claims.

Also, no scientific evidence supports the efficacy of most herbal dewormers. In the case of diatomaceous earth, which is composed largely of silica (the main component of glass), there is some evidence that it might be abrasive to the integuments of worms in the GI tract, but it can only target those worms in the digestive system and might not be enough to kill them. And, one has to wonder what that same abrasiveness might do to your horse's delicate intestines!

If you choose to try a natural or herbal anthelmintic on your horse, check its efficacy by performing a fecal egg count before and after administration (see "Diagnosis: Examining the Evidence," www.TheHorse.com/emag.aspx?id=5193, for information on doing a fecal egg count). That will give you some indication as to whether they've provided the protection your horse needs.

Myth #5—"To keep worms from developing resistance, I should use a different drug each time I treat my horse."

False—Rotation will be addressed more fully in next month's article on anthelmintic resistance. For now, keep in mind that the practice of rotating equine dewormers began four decades ago, and the rationale wasn't resistance, because that was long before anthelmintic resistance became a prevalent problem. Instead, rotation was recommended because earlier anthelmintics didn't have broad-spectrum activity. Owners alternated among various products to cover any holes in the spectra of the other drugs. (For example, phenothiazine didn't kill ascarids, so piperazine was used from time to time. Neither was effective against bots, so that required dichlorvos.)

Because most modern dewormers are "broad spectrum" and strike down many worm species in a single blow, rotation as most of us understand it is now largely a moot practice for horses. There are routines for managing resistance, however, which we'll talk about next month. Stay tuned!

SAVING A FEW BUCKS

Are Compounded Anthelmintics As Good?

You might have heard about "compounded" dewormers that are much cheaper than those you can buy from a vet or tack shop. They claim to have the same active ingredient as the national brands, so they should be just as good, right? Often, the answer is no.

Manufacturing veterinary drugs is very complicated; to achieve Food and Drug Administration (FDA) approval, manufacturers must demonstrate that a product consistently matches its label's description. A drug is far more than just the active ingredient. Its safety and efficacy depend on the active ingredient plus all of its other components, which can affect its stability, shelf life, and absorption. Overall product quality depends on the quality of each component.

In many cases, compounded drugs are examples of drug piracy, the act of producing and selling a copy of a drug approved by the Food and Drug Administration (FDA).

Many chemicals used in pirated products are bulk chemicals (usually powders) that come from non-FDA-approved sources. They might be smuggled into the Unites States and have no quality assurances or testing for safety or efficacy. It's nearly impossible for pirates to produce a drug that matches FDA quality, safety, and efficacy standards. According to at least one independent study performed by Merial Limited, pirated ivermectin dewormers have been shown to lack the quality and concentration of the FDA approved products (see "Is Your Horse Getting The Right Medications?," www.TheHorse.com/emag.aspx?id=3649).

The other problem with pirated products is that if the drug fails to work or harms your horse, you as an owner have no recourse because drug piracy and the use of these products is illegal. With dewormers, you might not even know the pirated product hasn't done what you expected it to do; the consequences might not be apparent for several months.

Pharmacists can legally compound veterinary drugs *if* there are no FDA-approved drugs for the problem. Given the various dewormers and formulations (pastes, gels, suspensions, solutions, pelleted top-dress) available, that's clearly not the case, so it's hard to conceive of a situation that would justify using a compounded anthelmintic. The main motivation seems to be price. Remember, you usually get what you pay for. Caveat emptor (buyer beware).

The issue of illegal drug piracy for animals has low priority for the FDA, so it might be years before this problem receives serious enforcement. In the meantime, it's up to veterinarians and horse owners to remain vigilant.—*Joseph J. Bertone, DVM, MS, Dipl. ACVIM*

RESISTANT WORMS

Do Your Horses Have Them?

PARASITE PRIMER—PART 9

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

Most people assume that when they administer a tube of dewormer to a horse, the drug is effectively killing worms. The drug must work—it says so there on the label. Right? Unfortunately, the answer frequently is no. All dewormers were highly effective when they were first introduced, but over time parasites have developed resistance to many drugs. The product labels reflect results of studies performed when the dewormers were first developed—before the worms developed resistance—and drug companies have not been required by the FDA to modify labels to reflect current levels of effectiveness. So it's possible that the drug you choose to deworm your horses might not be doing what you expect.

What Is Resistance?

Drug resistance is defined as the ability of worms in a population (e.g., worms on a given farm) to survive a treatment that once was effective against the same population (same drug, same dose, same parasite). It's an inherited genetic trait in the parasite that results from natural selection, the selective pressure being treatment with a drug.

How does this work? Let's use the example of small strongyles. Small strongyle worm populations on farms are extremely large. If you include the infective larvae on pasture, the developing larvae in the horse's intestinal walls, and the adults in the intestinal lumen (cavity), there can be millions or even billions of worms on a farm. This enormous population size, combined with a naturally high mutation rate, gives these worms a tremendously large genetic diversity, and some of them will have the genetic ability to survive treatment with drugs.

BARBARA LIVINGSTON

All dewormers were highly effective when they were first introduced, but over time parasites have developed resistance to many drugs. Are your horses the ones at risk?

ANNE EBERHARDT

Considering that horse populations are transported, mixed, and often graze shared pastures, the transmission and widespread dispersal of resistant parasites is virtually assured.

In essence, it's a numbers game. The resistant worms are actually present before the drug is used, but in extremely small numbers. But because they survive the drug treatment, they have a tremendous advantage over the rest of the parasite population. Each time a horse is dewormed, the resistant worms live to shed their eggs onto the pasture. At first the numbers of resistant worms are extremely low, but over time and with repeated deworming, the numbers of resistant worms in the population increase. Eventually, the resistant worms make up a large proportion of the population and the drugs no longer are effective.

There's also another important factor in this selection process: The number of worms carrying resistant genes increases very slowly at first. But after a certain threshold level is reached, the numbers increase quite rapidly. The reason this pattern develops is a simple matter of mathematics, analogous to the concept of compounding on investments. As any investment advisor will tell you, the length of time you save is more important than the amount you save each year because growth compounds on itself.

Studies on sheep parasites have shown that when resistance is inherited as a recessive trait (which seems to be the most common mode of inheritance for resistance in worms), at least 25% of the worms carry the resistance gene (meaning that 6% of worms will be homozygous for the gene and fully resistant) before treatment efficacy decreases enough to be noticed. In other words, by the time we see treatments not working as well as expected, resistance is on its way to reaching very high levels.

Detecting Resistance

The most accurate way to establish the presence of resistant worms in a population is to compare the number of worms recovered from treated and untreated horses infected with the same population of worms. But because these types of studies require slaughter of the animals, they're not feasible for on-farm diagnosis and are rarely done.

Molecular assays capable of detecting mutations that cause resistance offer great promise, but they are not yet available to the public. Research investigating the molecular basis of resistance should be made a priority, because molecular tests can detect and measure resistance while the gene frequency is still low and the drugs are still effective. Such tests could be used not only to detect resistance if it exists, but also could be used to monitor the development of resistance over time and to prevent entry of resistant worms onto a property. If we can say with relative certainty that resistance genes are starting to accumulate on a given farm (for a given drug), then worm control strategies could be modified to help preserve the effectiveness of that drug.

Until molecular assays become reality, however, diagnosing resistance is more prosaic. Presently, the fecal egg count reduction test (FECRT), while far from

perfect, is considered the gold standard for clinical diagnosis of anthelmintic resistance. When performing this test, one simply compares the number of parasite eggs in the feces after treatment with the number that were there before treatment.

The biggest snag with FECRT is that it's good at measuring resistance in strongyles, but isn't nearly as useful for other parasite species, such as roundworms/tapeworms. Additional research is needed to determine the optimal methods for performing and analyzing results of FECRT when testing for resistance in worms other than strongyles.

As discussed in last month's article on anthelmintics, there are three major classes of deworming drugs: Benzimidazoles, tetrahydropyrimidines (pyrantel, a.k.a. Strongid), and the macrocyclic lactones (ivermectin and moxidectin). When these products were first introduced to the marketplace, the percentage strongyle fecal egg count (FEC) reductions for benzimidazoles were approximately 95-100%, for tetrahydropyrimidines (pyrantel) 90-100%, and for macrocyclic lactones (ivermectin, moxidectin) 99.9-100%. These values can serve as a guideline for comparing results of FECRT when testing for resistance.

As worms develop resistance to these drugs, percentage reductions decrease, so at some point we can unequivocally say that anthelmintic resistance is present. A reduction in FEC of 90% (anthelmintic decreases parasite eggs by 90%) is often used as the cut-off for determining whether resistance is present on a farm. However, because FEC can vary widely between horses and over time, and usually only small numbers of horses are tested, it can be difficult to know if resistance is really present when values decrease only marginally from these established levels of efficacy.

Therefore, the following guidelines should be used when interpreting FECRT results for benzimidazoles and tetrahydropyrimidines: Greater than 90% drop in number of eggs means the drug was effective (no resistance); 80-90% means resistance can be suspected; and less than 80% means resistance is definitely present and the drug wasn't effective.

In contrast, the extremely high efficacy of the macrocyclic lactones (ivermectin/moxidectin) makes any egg reduction of less than 98% a cause for concern.

Eradication Vs. Control

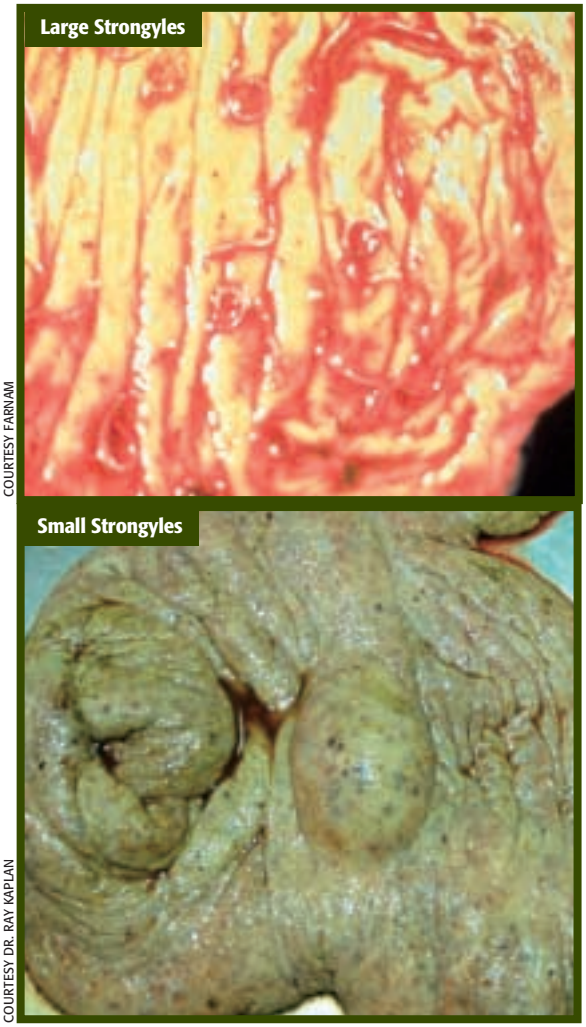
It's tempting to interpret the results presented to mean that only ivermectin or moxidectin dewormers should be used for small strongyle control, but this could be a significant mistake. Remember that oxibendazole and pyrantel still are effective on many farms, and these drugs should continue to be used where they remain efficacious. The only way to know whether these drugs are effective or not is to perform a FECRT. Preliminary results of a study that is currently ongoing suggests that treating with both oxibendazole and pyrantel at the same time can yield clinically significant increases in efficacy. Thus, on many farms using these drugs in combination may prove to be an effective means to decrease the reliance on ivermectin and moxidectin.

It's also important to realize that ivermectin and moxidectin can't be expected to remain effective forever. Furthermore, there are currently no new anthelmintics in development likely to hit the store shelves soon. So the sooner we implement strategies to decelerate any further selection for drug resistance, the better we'll prolong the effective lifespan of the macrocyclic lactones.

Given this situation, it is clear the "no parasites allowed" mentality of horse owners (in which the goal is to treat frequently enough to keep FEC near zero year-round) is neither sustainable, nor medically justified. We have to adjust our thinking.

When the familiar every-eight-weeks deworming program was first recommended in 1965, the highly pathogenic large strongyle, *Strongylus vulgaris*, was the primary target. The strategy has worked. In the past 40 years, *S. vulgaris* has become extraordinarily rare and no longer exists on most well-managed farms. But in their wake, another enemy has become the front-runner—the small strongyles, which were once considered to be little more than a nuisance with low disease-causing potential. Small strongyles are now the primary target of worm control programs in horses. Although the current situation is very different than the one that existed 40 years ago, many owners refuse to adjust their deworming protocols to meet today's realities.

It might seem odd to suggest that we think in terms of control rather than eradication, but in order to select against resistance, we actually have to encourage the presence of anthelmintic-sensitive worms in the population so they don't become outnumbered by the resistant worms. Therefore, the most successful parasite control strategies are those based on performing routine fecal egg counts to identify horses who need treatment vs. those who don't (see "Examining the Evidence" in the June



COURTESY FARNAM

COURTESY DR. RAY KAPLAN

When the familiar every-eight-weeks deworming program was first recommended in 1965, the highly pathogenic large strongyle (top), *Strongylus vulgaris*, was the primary target. The strategy has worked. In the past 40 years, *S. vulgaris* has become extraordinarily rare and no longer exists on most well-managed farms. But in their wake, another enemy has become the front-runner—the small strongyles (bottom). The cecum of a horse infected with small strongyles becomes thickened, roughened, discolored, and full of edema fluid. The peppered appearance is from the presence of worms (small circles are larvae), as well as from the damage they cause when exiting the tissue.

2004 issue, www.TheHorse.com/emag.aspx?id=5193, for instructions on how to perform and interpret a FEC). A deworming program based on treating only the horses who need it will result in better parasite control overall and be accomplished with far fewer treatments than most farms now give.

The Rotation Question

The practice of rotation (using a different class of deworming drug each time you treat your horse) is also an idea whose time has come and gone. It does not appear to significantly slow the progression of resistance, and it can actually mask the clinical effects of using an ineffective drug along with an effective one. As a result, horse owners, stable managers, and veterinarians are almost always unaware of the drug resistance problem.

Some parasitologists recommend "slow" rotation as an alternative to traditional rotation. In other words, use a single anthelmintic for an entire year, then a different drug the following year. This approach has its pluses, although because not all dewormers are broad-spectrum, some might fail to control other important parasites such as bots or tapeworms. Given this, and the current high prevalence of resistance in cyathostomes (small strongyles), this approach can no longer be recommended.

Anthelmintic drugs must be selected based on a number of considerations, taking into account efficacy against a variety of different parasites as well as time of year. In other words, there's no easy answer.

Common Sense Strategies

Small Strongyles

Many farms routinely shove a tube of dewormer in the mouth of each new horse who arrives. But this practice might actually accelerate the spread of resistance. How?

If a treated horse is infected with worms resistant to that drug, he will shed resistant eggs for several weeks following treatment. Furthermore, unless he is treated with a drug that kills the mucosal larval stages encysted in the intestinal wall (which are often much more numerous than the adult worms in the lumen of the gut), over the following weeks the mucosal larval worms will emerge from the intestinal wall and mature into adults, so a new round of egg shedding will occur. All of these eggs will come from the population of worms carried by the horse to its new location, so any drug-resistant worms infecting that horse will rapidly contaminate the new environment with drug-resistant infective larvae.

For these reasons, if a farm does not have resistance to benzimidazoles or pyrantel, long-term additions should be treated upon arrival with a larvicidal drug such as moxidectin to remove as much of the total worm burden as possible. Depending on the circumstances, a second treatment with moxidectin 12 weeks later might be desirable. Fenbendazole at a double dose for five days also has demonstrated excellent efficacy against small strongyle mucosal larvae, but the efficacy of this regimen against benzimidazole-resistant small strongyles has not been established. Given the extremely high levels of fenbendazole resistance known to exist, and results of recent studies suggesting that the effectiveness of this treatment might be only moderate to poor, this treatment regimen cannot be recommended for preventing the introduction of resistant worms.

Short-term additions to your farm (those staying less than six weeks) can be treated with one dose of ivermectin, since the egg reappearance period following

PERFORMING FECAL EGG COUNT REDUCTION TESTS (FECRT)

How Many Parasites are in Your Horse?

The most practical way to perform fecal egg count reduction tests (FECRT) is to examine one drug at a time. If you begin testing one drug, then test a different drug at your next deworming, all of the dewormers you use can be tested within a six-month period.

At the time of your scheduled treatment, collect a fresh fecal sample from each animal. Ziplock bags work well for this; turn the bag inside out, pick up one or two manure balls, flip the bag right-way out, and seal. Make sure you label the bag with the horse's name and the date. Place the samples in the refrigerator or keep them in a cooler with ice packs. If kept cold, FEC can be done at your (or the diagnostic lab's) convenience over the next week. If the feces become warm, the eggs will hatch and counting can no longer be done. If you see larvae inside the eggs, it is likely that eggs have already started to hatch, invalidating the count.

Perform a McMaster FEC on all samples and record results (see "Diagnosis: Examining the Evidence" in the June issue of *The Horse,* www.TheHorse.com/emag.aspx?id=5193, for details on performing FEC).

To reliably measure a reduction after treatment, FEC of should be greater than 100 eggs per gram (EPG) when you run this first egg count, but lower values can still be useful if the drug has a poor efficacy. This is because if egg counts start out low, but treatment still fails to lower them to 0, you can conclude that the drug is not effective. However, when FEC are low and go to 0 after treatment, it cannot be assumed that the drug was highly effective.

To properly interpret results, there should be at least four (preferably six) horses with pre-treatment FEC of 100 EPG or greater. Because many horses will have FEC of less than 100 EPG, if available, eight to 12 horses should be tested. Fewer horses can be used, but confidence in the results diminishes when numbers are small.

Ten to 14 days after treatment, collect a second sample and repeat the McMaster FEC. For each horse, use the following formula to calculate the percent reduction:

FECR% = [(pre-treatment EPG - post-treatment EPG divided by pre-treatment EPG) x 100]

For example, if your pre-treatment EPG was 150 and your post-treatment EPG was 75, then FECR% = (150 - 75 / by 150) x 100 = 50%

Then calculate the average for all the horses tested with a particular drug. For fenbendazole, oxibendazole, and pyrantel pamoate, use the following criteria for interpreting the average percent reduction for the group: Less than 80% means it is not effective (resistance is present).

Because ivermectin resistance has not yet been detected in equine strongyles, we currently lack the knowledge required to properly interpret results of the FECRT. It is assumed for ivermectin that any reduction less than 98% is a cause for concern. Therefore, it is recommended that testing be repeated with another group of horses or wait at least eight weeks (preferably 10-12) and repeat in the same horses. Moxidectin is more potent than ivermectin, so resistance to ivermectin is expected to occur first. Consequently, there is no reason to perform FECRT with moxidectin as long as ivermectin remains fully effective.

If you suspect strongyle resistance to ivermectin, please contact Dr. Ray Kaplan at the University of Georgia via e-mail (rkaplan@vet.uga.edu) with your results.—*Karen Briggs*

ivermectin treatment is six to eight weeks and ivermectin continues to demonstrate virtually 100% efficacy against small strongyles in the gut lumen. However, if you know your farm already has resistance to benzimidazoles and pyrantel, at the present time there's no need for concern about treating upon arrival to prevent introduction of resistant worms. In that case, any treatments given would be based on other worm control considerations.

***Parascaris equorum* (roundworms)**

This parasite is only a concern in foals because horses become immune to roundworms as they reach about 18 months of age. No studies have been performed to investigate the prevalence of resistance in this worm, although researchers do suspect resistance to ivermectin and moxidectin, based on two published and several unpublished reports showing poor FECRT. If these reports of suspected ivermectin and moxidectin resistance are confirmed (which is probable), it is likely that macrocyclic lactone-resistant *P. equorum* are widespread. To prevent introduction of these worms, it would be advisable to treat new or visiting foals with the five-day double-dose fenbendazole regimen, since this treatment will kill both the adult and immature tissue-migrating stages of this worm.

In Conclusion

The importance of small strongyles continues to increase, because extensive reliance on drug treatment for control has led to the development of resistance to all classes of available dewormers except the macrocyclic lactones, and no new dewormers are in advanced stages of development.

It seems extremely unlikely that any new equine dewormers with novel modes of action will be developed and marketed in the foreseeable future. We also don't know how close we are to having parasite resistance to the macrocyclic lactones, but such resistance seems inevitable. If resistance does appear to ivermectin, it will cause major problems for small strongyle control in horses.

Since the drugs we have now are all we can expect to have for quite a while, and because reversion to susceptibility does not appear to occur (resistant worms don't suddenly become susceptible to dewormers again), the aim of resistance control must be to delay the accumulation of resistance genes, although we have no current means of measuring its progress (or lack thereof). Since almost nothing is known about small strongyle genes involved in resistance to dewormers, gaining basic knowledge in this area is a critical need. Without such knowledge, the genetic diagnosis of resistance is impossible, leaving us with the diagnosis of treatment failure as the only alternative.

Considering that horse populations are transported, mixed, and often graze shared pastures, the transmission and widespread dispersal of resistant parasites is virtually assured. We need to be proactive about the problem and let go of outdated approaches. Using drugs that don't work because of resistance is both ineffective and a waste of money. Using only macrocyclic lactones is an alternative, but what happens when resistance to these drugs appears? We'll tackle that thorny issue in the final three articles in this series.

KIM AND KARI BAKER

A deworming program based on treating only the horses that need it will result in better parasite control overall and be accomplished with far fewer treatments than most farms currently give.

LOSING CONTROL OF PARASITES

Parasite Resistance Across Species

Parasite resistance to anthelmintics is not limited to horses, but is a worldwide problem, where producers of all livestock species must judiciously use chemicals to eliminate parasites in order to keep this problem from skyrocketing. Peter J. Waller, BSc, BVSc, FRCVS, of the National Veterinary Institute in Stockholm, Sweden, examined many different studies that measured parasite resistance on sheep and goat farms in Africa, Australia, Asia, Europe, North America, and South America after increasing unthriftiness caused suspicion that the parasites that plague these animals were becoming resistant to the anthlemintics used. Fecal egg counts were taken before and after either benzimadazole, levamisol, or ivermectin were administered to determine the level of parasite resistance.

The results of these investigations were startling-there were high levels of resistance shown in both sheep and goats to all of the anthelmintics. Most importantly, ivermectin was shown to be the least effective anthelmintic for goats in the southern U.S. Recently in Malaysia, shepherds are faced with total parasite resistance in their flocks to all classes of anthelmintics. Macrocyclic lactone resistance is also beginning to appear in cattle populations throughout the world.

Although many factors are involved in the development of resistance, the single most important of these is frequent treatment of all animals.—*Marcella M. Reca*

DEWORMER ADJUNCTS

Control Without—or Along With—Chemicals

PARASITE PRIMER—PART 10

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

Our horses are lucky to live in an age when modern deworming drugs can pretty much rid them of parasites. They're living longer and healthier lives than ever before as a result of the easy availability of these drugs. But as we discussed last month, drug resistance is a looming problem, and questions still exist as to the most effective deworming program. Are there other things you could be doing, not instead of, but in addition to using anthelmintics, to help keep your horse's parasite load at a minimum?

Yes, there are several strategies you can implement to reduce the risk of parasite transmission in your own herd. Not all of them might be practical for your operation or circumstances, but at least some could have a place in your management routine.

To understand how these ancillary measures work, let's review the basic cycle of parasite infection, so we can identify the opportunities for intervention.

The Parasite Life Cycle Reviewed

1. Female parasites living inside the horse's gut lay eggs that are passed into the environment with manure.
2. Under conditions of favorable temperature and humidity, worm eggs hatch and develop into infective larvae.
3. Infective larvae survive in the environment for varying intervals, depending on climatic conditions.
4. Pastured horses swallow infective larvae as they graze.
5. Larvae mature within the horse, develop into reproductive adults, and the cycle starts again with the next worm generation.

ANNE EBERHARDT

One strategy for minimizing equine parasite loads on pastures is to allow non-equid species to graze the pasture for a while. Equine strongyle larvae are quite host-specific; they cannot infect cattle, sheep, goats, camellids (llamas or alpacas), or humans. When pasture contamination becomes dangerous for one bunch of residents, it's time to move them off into another area and bring in the substitutes, and the original parasites will die off because their hosts aren't around.

Let's examine some control options designed to interfere with various elements of this basic life cycle.

Fecal Contamination

Because each individual parasite began life as an egg in a manure pile, we could win the battle against equine parasites if it were feasible to keep horses from contaminating their environment with manure. How could we do that? Although carriage horses must be "diapered" in some urban areas, this isn't considered practical for horses in more usual habitats. But as a point of illustration, diapering would be a highly effective, non-chemical method of parasite control that would greatly reduce the risk of re-infection with most equine parasites (as long as you properly disposed of the manure).

In lieu of preventing primary fecal contamination, prompt and thorough removal of manure from the environment can be very beneficial. In a bygone era, the most elite stables employed pasture grooms, who followed grazing horses with a scoop shovel and a broom. Their job was to remove manure as quickly as it was dropped. But apart from it being very labor-intensive, this practice was probably not completely effective. After falling a distance of four or more vertical feet, some manure balls disintegrate on landing, so it isn't possible to recover every bit of manure nor the worm eggs contained therein.

In the 1980s, a similar approach was evaluated using updated technology. Studies at Newmarket in Great Britain examined the efficacy of cleaning horse pastures with a large commercial vacuum unit that was originally designed for golf course maintenance. Twice-weekly vacuuming was demonstrated to control pasture infectivity more effectively than routine deworming. However, the cost of the vacuum units was prohibitively expensive for the average horse owner, and the process only worked well on level pastures.

Even if one could afford to do it, the best intentions of removing manure at regular intervals can be totally confounded by a heavy rainfall. Precipitation in excess of one inch is the single most important agent for disseminating infective larvae away from a manure pile and onto forage.

Finally, because most stall habitats do not support the development of infective strongyle larvae, regular stall hygiene does little to reduce the risk of strongyle infection for confined horses. It's true, however, that stabled horses can acquire ascarids and pinworms when confined to an infected stall.

Larval Development

Eggs hatch and develop into infective larvae under conditions of moderate temperature and moisture. Cold slows the rate of development or stops it altogether, and excessive heat kills eggs and larvae. Could we capitalize on these weaknesses? Is it possible to heat manure sufficiently to kill the parasites contained therein?

Yes, it is, and it doesn't even require a fancy autoclave or a steam generator. Proper composting of manure and soiled bedding will generate relatively high internal temperatures, and strongyle larvae in manure are virtually eradicated by exposure to temperatures over 90ºF for a minimum of two weeks. Composting is a practice that should already be in place at any stable. (For more on composting, please see "Living on the Edge" in our March 2004 issue, www.TheHorse.com/emag.aspx?id=5045.)

A composting corollary is that fresh or non-composted horse manure should never be spread on pastures. Spreading fresh manure can deposit as many potential worms as would result from weeks of grazing.

Larval Survival

Leaving pastures unoccupied for several months of the year might reduce the risk of infection, but remember the seasonal patterns we discussed in "Development and Persistence of Parasites" (in the July 2004 issue, www.TheHorse.com/emag.aspx?id=1523). Strongyle larvae can survive for only a few weeks during summer, but for as many as six to nine months during colder weather—and few horse owners can afford to leave a contaminated pasture ungrazed long enough for the worm larvae to die of attrition.

However, it might be possible to temporarily turn a grazing pasture into a hay field and recover the forage in a baled format. Or you might want to allow non-equid species to graze the pasture for a while. Equine strongyle larvae are quite host-specific; they cannot infect cattle, sheep, goats, camellids (llamas or alpacas), or humans. Given a herd or flock of "alternative livestock" that consumes about the same amount of pasture as your horses, effective parasite control could be achieved by routinely alternating the use of pastures between horses and other species. This is a complex solution, but one that could work extremely well if the species were rotated at intervals that were appropriate for the local climate, i.e., when pasture contamination becomes dangerous for one bunch of residents, it's time to move them off into another area and bring in the substitutes.

A deposit of horse manure in a pasture can serve as a protective reservoir for parasitic larvae if the manure dries gradually over a period of several weeks. Larvae within fecal masses can be evicted from this protected habitat and exposed to desiccation and ultraviolet light by "dragging" or chain-harrowing pastures to break up the manure. However, dragging should only be performed on unoccupied pastures, and only during hot weather. Horses can be put back on dragged pastures to graze within two to three weeks after dragging during summer conditions, but the risk of infection will not decrease until the following spring if pastures are dragged in autumn or winter.

Ingestion of Infective Larvae

Probably the single most effective recommendation for preventing parasitism in horses is to limit their pasture exposure. This is easy to say, but hard to do. Pasture is an important source of exercise and provides cheap nutrients for most horses, and pastured animals are far less labor-intensive than confined horses. Grazing muzzles can be used on some horses to minimize ingestion of forage while they are turned out—a good solution if you have easy keepers on lush pasture or any horse with a previous founder issue.

Although the parasite control impact of this management practice has not been formally evaluated, anything that reduces ingestion of forage and infective larvae should theoretically decrease the risk of transmission.

KIM AND KARI BAKER

One can reduce the level of parasite challenge in a horse pasture by removing manure at regular intervals, but this practice can be totally confounded by a heavy rainfall. Precipitation in excess of one inch is the single most important agent for disseminating infective larvae away from a manure pile and onto forage.

Customized Control Program

Previous articles in this series have explained that individual horses vary considerably in their susceptibility to parasitism, as evidenced by differences in the magnitude of fecal egg counts. If we were to monitor those fecal egg counts on a regular basis, we might be able to identify horses that are naturally more resistant to strongyle infection. These gifted animals could be dewormed less frequently than the typical hayburner, and the most intensive efforts could be reserved for horses that consistently have higher egg counts than the norm.

The differences in parasite susceptibility among individual horses are likely to have a genetic basis, as has been demonstrated unequivocally in ruminants. Certain breeds of hair sheep (the Barbados Black Belly, Florida Native, and St. Croix) are extremely resistant to parasitism, as are certain individuals within most of the classic meat or wool breeds. In fact, Merino rams with a high degree of genetic parasite resistance are highly prized in wool-producing countries because their first-generation offspring are 50% more resistant to worms than their dams. Admittedly, Thoroughbred Triple Crown aspirants are a long way from selecting sires and dams based on their genetic parasite-resistance properties, but nevertheless, it is entirely possible that one could breed a horse herd that had few parasitic problems and could remain healthy without the use of anthelmintics.

"Alternative" Dewormers

Various compounds, including chewing tobacco and garlic, have been purported to have activity against intestinal parasites of horses. In recent years, the most common subject of such claims has been a naturally occurring substance known as diatomaceous earth (DE). Diatomaceous earth is a whitish powder made up primarily of the exoskeletons of fossilized algae (diatoms). It's almost pure silicon dioxide, the same chemical formula as quartz, and its microscopic sharp edges make it useful as a filtering agent and an abrasive for industrial uses.

Theoretically, the abrasive nature of DE particles damages the integument (outer skin layer) of nematodes as they pass through a horse's gastrointestinal tract. Unfortunately, DE's reputation does not bear up under scrutiny. It has been the subject of only limited formal evaluation in sheep and horses (American Association of Veterinary Parasitology Proceedings), but those researchers failed to detect any anthelmintic effect associated with feeding DE, as measured by fecal egg count reduction in treated horses compared to untreated control animals.

One must also wonder about the effect of such an abrasive silica product on the horse's intestinal tract!

Along similar lines are the increasing number of so-called organic or herbal dewormers appearing in tack shops. To the best of our knowledge, the efficacy of these products has never been demonstrated in formal, controlled evaluations. These products exist primarily because they exploit differences in the labeling requirements for drugs vs. non-drug items. Before a drug can earn label claims for activity against parasites, this fact must be proven unequivocally to the Food and Drug Administration by extensive efficacy and safety testing. And once a dewormer is approved by the FDA, the claims that can be used in advertising that drug are regulated by the FDA.

Curiously, products that are not drugs per se do not require FDA approval for marketing, so advertisers of non-FDA approved products can say just about anything they want, and their products don't have to be effective. Caveat emptor, buyer beware.

Next month, we'll offer you some specific recommendations for controlling parasites in your young horses.

"NATURAL" DEWORMING OPTIONS?

PHOTOS.COM

Diatomaceous earth (DE) is a whitish powder made up mostly of the exoskeletons of fossilized algae (diatoms). Theoretically, the abrasive DE particles damage the integument (outer layer) of nematodes in a horse's gastrointestinal tract. However, DE's reputation doesn't bear up under scientific scrutiny.

Pumpkin seeds have been used as an herbal remedy for treating parasite infections in horses because the seeds contain the amino acid cucurbitin, which many herbalists consider to be a natural anthelmintic. However, the efficacy of organic/herbal dewormers has never been demonstrated in formal, controlled studies.

AGE-RELATED PARASITES

Scourges of Foals and Young Horses

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

PARASITE PRIMER—PART 11

For the past 10 months in this series, we've examined a host of issues relating to equine parasites and their control. Now it's time to get down to the nitty-gritty: How to ensure that parasites have the minimum impact on your horse's health and well-being. Let's start with young horses, because parasite populations in immature animals are often quite different than those found in adults. Some kinds of worms, in fact, prefer young horses so strongly that they're almost never found in equines past a certain age. Infections with threadworms, roundworms, and pinworms, for example, are found almost exclusively in horses less than six months, 18 months, and 24 months old, respectively. Because of the age distribution of these worms, parasite control recommendations for immature animals are necessarily different than those followed for adults.

Susceptibility of Youth

Most of the common nematode (roundworm) parasites of horses are transmitted by ingestion, so the risk to young foals is diminished somewhat while they're nursing rather than grazing. But as curious colts and fillies investigate

ANNE EBERHARDT

Weanlings and yearlings on pasture are particularly susceptible to parasitic disease because unlike their dams, they don't yet have the advantage of acquired immunity or resistance to some types of parasites

their environments, they inevitably come into contact with, and swallow, infective stages of various parasites, including large and small strongyles, roundworms, pinworms, tapeworms, and possibly threadworms (see "Parasites Affecting Juvenile Horses" below).

Weanlings and yearlings on pasture are particularly susceptible to parasitic disease because unlike their dams, they don't yet have the advantage of acquired immunity or resistance. And foals that are under stress from shipping, weaning, or environmental changes, to name only a few, can suffer from immune system suppression, making them more susceptible to infectious diseases and interfering with optimal response to vaccination or anthelmintics.

Parasites Affecting Juvenile Horses

COMMON NAME	SCIENTIFIC NAME
Pinworms	*Oxyuris equi*
Roundworms (ascarids)	*Parascaris equorum*
Large strongyles	*Strongylus spp.*
Small strongyles (cyathostomes)	*Numerous species*
Tapeworms	*Anoplocephala perfoliata*
Threadworms	*Strongyloides westeri*

Although we've talked about the specific parasites listed in the table in earlier installments of this series, let's review the major targets of parasite control programs for the juvenile horse, in the approximate chronological order they are encountered by a foal.

Threadworms

Strongyloides westeri, the equine threadworm, is usually the first parasite a foal encounters. It is commonly transmitted from mare to foal through nursing when larvae in the body tissues of mature mares migrate to the mammary glands when signaled by the hormones of pregnancy and lactation. Threadworms are so well-adapted for this mode of transmission that they're often present in the colostrum mares produce immediately after birth, and in the first few days of milking.

Threadworm infections also can be acquired from the environment, so foals should not be exposed to wet pens or muddy paddocks.

Adult horses infected with *S. westeri* suffer no clinical signs (unthriftiness, scours) because the threadworms live in the horse's tissues. In foals, once the larvae find a home in a naïve host, they mature rapidly in the small intestine. Within 10 to 14 days after birth, foals begin to shed eggs from the parasite. Historically, threadworms in foals were thought to be the cause of diarrhea, but research has shown that this is inaccurate. The majority of foals show no clinical signs whan infected. Heavy infections of threadworms in foals can persist for 10 weeks, and lighter infections can last two to three times as long.

Controlling *Strongyloides* infections can take a two-pronged approach. The first is to treat the foal early with an anthelmintic that is effective against threadworms. Ivermectin is labeled for this application and probably provides persistent protection against re-infection for a week or two following treatment. Some practitioners suggest treating foals with ivermectin within a few days of birth.

The second plan of attack is to treat the mare with ivermectin within 24 hours of foaling. This treatment kills migrating larvae in the mammary tissues of the mare, thus removing the immediate source of infection for her foal.

It can also be helpful to examine the manure of foals routinely at two to four weeks of age and treat accordingly if positive (ivermectin or oxibendazole at 15 mg/kg). Any foal with diarrhea that is due to *Strongyloides* will always have numerous eggs in the manure. Therefore, fecal exams should always be conducted on any foal that develops diarrhea prior to weaning.

JOHANE JANELLE

Threadworms are commonly transmitted from mare to foal through nursing when larvae in the body tissues of mature mares migrate to the mammary glands when signaled by the hormones of pregnancy and lactation.

The good news is that the current prevalence of *Strongyloides* in foals is fairly low, and the damage done by infection is usually not severe. So, a wait-and-see approach is probably justified for most breeding operations.

Roundworms

Parascaris equorum, the well-traveled equine roundworm, finds its way inside young foals as they begin to sample pasture grasses. The infective eggs, once swallowed, release their larvae in the small intestine, and the larvae then invade the gut wall and travel to the liver, either via the circulatory or the lymphatic system. After a one-week sojourn, they hitch a lift in the bloodstream and migrate to the lungs, where they work their way from the terminal air sacs called alveoli, up through the bronchioles, and eventually to the trachea. When the migrating ascarids reach the back of the throat, they're swallowed and return to the small intestine, where they finally settle, become mature, and start to reproduce. Female ascarids generally start to lay eggs within 10-12 weeks after infection.

It's almost inevitable that foals on any breeding farm will be exposed to roundworm eggs. Since these parasites have

COUNTING EGGS

Fecal Exams for Juvenile Horses

What can you expect to see under the microscope in routine fecal examinations of foals, weanlings, and yearlings? The eggs of threadworms might appear in the manure of foals by two weeks of age, but the infection will disappear permanently by five to six months. Ascarid eggs first appear in manure as early as 70 days following infection, and an infected weanling can pass several million eggs per day. Roundworm infections are generally controlled by acquired immunity, and it is unusual to see ascarid eggs in a fecal sample from any horse older than 18 months. Pinworm eggs are rarely observed during fecal examination because the females lay their eggs on the skin around a horse's anus, rather than mixed in with the manure like other parasitic nematodes.—*Karen Briggs*

What Kills Ascarids (Roundworms)?

DRUG ACTIVITY	CHEMICAL NAME
Adulticidal	Fenbendazole (10mg/kg) [1]
	Ivermectin
	Moxidectin [2]
	Oxfendazole
	Oxibendazole
	Piperazine
	Pyrantel pamoate
	Pyrantel tartrate
Larvicidal	Ivermectin
	Fenbendazole (10 mg/kg) daily for 5 days [3]

[1] *The dosage of fenbendazole required for efficacy against ascarids is higher than the dosage recommended for removal of other worms.*

[2] *Although very effective, moxidectin is not approved for use in foals less than six months of age.*

[3] *Although shown to be effective in research studies, there is no label claim for activity against migrating* Parascaris.

significant pathogenic potential, roundworms are the main target of parasite control programs for immature horses. The goals of ascarid control are twofold—to kill worms (thus minimizing the negative effects on health and performance), and to prevent the maturation of worms so eggs are not shed into the environment.

Anthelmintics that are effective against ascarids fall into two broad categories: Those that kill adult and juvenile worms in the small intestine, and those that are also effective against roundworm larvae migrating through the liver and lungs of a foal (see "What Kills Ascarids?" above).

Depending on the type of drug used in foals, the timing of scheduled treatments can be tricky. It takes about two to three weeks for larval worms to complete their migration and reach the intestine, and another eight weeks or longer for the worms to mature and begin shedding eggs. Therefore, if adulticidal drugs are used, the first treatment should be given when the foal is approximately 60 days of age. If ivermectin is used for the initial treatment, it can be given as early as 45 days because it is larvicidal. Thereafter, treatments should be administered at 60-day intervals.

Delaying treatment for intervals longer than 70 days could allow ascarids to mature and contaminate the environment with eggs. Remember that roundworm eggs can survive for a decade or longer, so one break in protocol can have very long-lasting consequences with ascarids. Bimonthly treatments should be continued until horses eventually acquire immunity at 15-18 months of age.

Many veterinarians suggest deworming foals at 30-day intervals, and some recommend ivermectin exclusively. The rationale of this program is prevention of pneumonia associated with larvae migrating through the lungs, but this practice apparently has had some negative consequences.

Within the past two years, some breeding farms in Canada and the United States have reported strains of *Parascaris* that are resistant to ivermectin—one of the first

FOAL DEWORMING POLL

Q: At what age do you begin deworming your foals?

1-2 months .....**50.54% (47)**
3-4 months .....**25.81% (4)**
5-6 months .....**11.83% (11)**
After weaning .....**9.68% (9)**
7-8 months .....**2.15% (2)**

** Answers from TheHorse.com poll*

documented cases of ivermectin resistance in horses. The evidence suggests that this resistance evolved in the face of 30-day treatments.

Regardless of the anthelmintics used, fecal exams should be performed on a representative sample of the juvenile population at each deworming interval to monitor the ongoing efficacy of the resident control measures.

Large and Small Strongyles

Strongyles (bloodworms) are considered the most dangerous equine parasite, and the most prolific. There are dozens of species of large and small strongyles, but only three or four large strongyle species are commonly found in North America. Horses infected with either large or small strongyles will shed eggs in their manure almost constantly, re-infecting their environment with the next generation of parasites.

Although youngsters can become infected with strongyles, controlling them is most important in adult horses, so we'll focus on them next month when we discuss parasite control programs for mature animals. The methods recommended here for controlling ascarids in youngsters should be adequate for strongyles as well, although it's wise to introduce either ivermectin or moxidectin into your program for juveniles at least once every five months to help eradicate large strongyles from the herd.

PAULA DA SILVA

Many a luxurious tail has been destroyed as horses rub their hindquarters against any surface they can find in an effort to relieve itching caused by pinworms.

Pinworms

Oxyuris equi, the pinworm, is a common and fairly large parasite with a long, tapering tail (hence the name). Pinworms lurk in the large intestine, and the females cement their eggs in masses to the skin around a horse's anus. As the eggs develop into the infective stage over four to five days, the cementing fluid dries and cracks, creating intense itching for the horse. Many a luxurious tail has been destroyed as horses rub their hindquarters against any surface they can find in an effort to relieve this itching.

Fortunately, nearly all equine dewormers are effective against pinworms, and the treatment intervals recommended for other parasites should control *Oxyuris* well.

Tapeworms

The only significant cestodes (flatworms) to infect horses, tapeworms are segmented at regular intervals. Each egg-containing body segment, or proglottid, is a separate unit, like the box cars that form a train. Single proglottids, or several linked ones, can break off from the body of the worm without killing the parasite, which remains attached to the intestinal wall.

Tapeworms have indirect life cycles, meaning they must develop within a different animal before they can be transmitted to their final host. Tiny oribatid mites, which live in vast quantities on many pastures, are the intermediate hosts for equine tapeworms; horses swallow these mites in the normal course of grazing, allowing the worms to grow and mature in the equine digestive tract over a six- to 10-week period.

CHEMICAL CLASSES

Which Dewormers Are Safe For Young Horses?

The list of dewormers available for use in foals is somewhat shorter than the one for adults. The labels of a few anthelmintics specifically indicate that they may not be used in foals less than five months (Zimecterin Gold) or six months of age (ComboCare, Quest, Quest Plus).

Many horse owners are unnecessarily concerned about the safety of dewormers used in foals. There is really no need to be anxious if the product is labeled for use in foals, and if it is dosed appropriately. Admittedly, it can be difficult to estimate the body weight of a foal, but an accidental overdose of any product labeled for foals is unlikely to cause adverse effects. Rare reports of deaths in young foals from moxidectin (Quest) treatment resulted from accidental administration of a full tube of dewormer intended for a 1,200-pound horse.

The only serious consequence of deworming that is more common in juvenile horses than in adults is the possibility of ascarid impaction (a colic that arises from large numbers of dead roundworms accumulating in the gut after treatment with a deworming drug). However, ascarid impactions should never occur in foals that have been on an effective control regimen.

If you suspect large numbers of ascarids have accumulated in a youngster's gut, whether from lapses in protocol or resistance to ivermectin (imagine, for example, you buy a pot-bellied weanling at auction and have no deworming history on him), the best approach is to treat the foal with a half-dose of fenbendazole (5 mg/kg) to kill some of the worms. Then, a second treatment with an effective adulticide (see "What Kills Ascarids?" on page 49) should be administered one week later to remove the remainder of the population.

There is experimental evidence that the use of daily dewormers (pyrantel tartrate) in juvenile horses interferes with the development of acquired immunity to strongyles. However, we don't know whether this practice diminishes protection against ascarids as well. Acquired immunity normally develops against invasive or migratory larval stages, but daily regimens are designed to kill parasites before they enter any tissues. Arguably, a horse on daily preventive is protected as long as the program continues and the drug remains effective. Therefore, a horse raised on a daily dewormer could be at great risk for severe parasitic disease if the regimen were disrupted and the animal were exposed to high numbers of infective stages—something to keep in mind before you commit to a daily dewormer program.

—Karen Briggs

Tapeworms attach fiercely to the intestinal wall, which can cause severe inflammation at the attachment site. They're also associated with several types of severe colic, especially ileocecal intussusceptions in young horses. In an intussusception, the last one foot of the small intestine (ileum) telescopes into the first section of the large intestine (cecum), and swells, blocking passage of the gut's contents. It's also suspected that chemicals the tapeworms release can interfere with normal gut motility.

PAULA DA SILVA

Recommendations for deworming foals start as early as 2 weeks of age.

Horses acquire tapeworm infections while they are grazing infective pastures. Research conducted in Scandinavia indicated that foals are first exposed to tapeworm infection while still nursing, but most do not pass tapeworm eggs in the manure until seven months of age or older. Therefore, there is little need to incorporate drugs specifically indicated for treatment of tapeworm infection (those containing praziquantel) until after foals have been weaned.

It's only recently that parasitologists have begun to look seriously at equine tapeworms, so evidence-based recommendations for tapeworm control have not yet been generated. Based on the tapeworm life cycle, however, many experts recommend treating horses in spring and autumn.

General Recommendations

Some general management practices that are commonly recommended for parasite control in mature horses are equally applicable to their young offspring. These include reserving the cleanest pastures available for weanlings, yearlings, and mares with foals. Clean pastures include those that have been vacant for at least two months during the warm season of the year, fields that were used recently to produce hay, or pastures that were grazed by an alternate livestock species, such as cattle or sheep.

Wherever possible, horses should not be fed off the ground. Soil is an excellent reservoir for infective ascarid eggs, and the greatest concentrations of strongyle larvae occur near the roots of plants. Environmental hygiene should be practiced for young horses maintained in stalls or small "dry lot" paddocks. Stalls should be cleaned thoroughly and disinfected prior to any introduction of new stock, manure should be removed from them daily, and all bedding should be stripped from stalls at regular intervals.

Deworming Recommendations

Threadworms—Using ivermectin, treat the mare within 24 hours before foaling or treat the foal during its first two weeks of life. Alternatively, if a fecal examination is positive for *Strongyloides* infection, treat the foal with ivermectin or oxibendazole (15 mg/kg). Fortunately, threadworms are controlled completely by acquired immunity in all foals older than six months.

Ascarids—Start treatments with pyrantel pamoate (a.k.a. Strongid) or benzimidazole paste (fenbendazole, oxfendazole, or oxibendazole; all 10 mg/kg) at 60 days of age. Or, if using ivermectin, treatment can be initiated at 45-60 days of age. Repeat subsequent treatments at 60-day intervals until the horse is 15-18 months old.

Rotation among benzimidazoles, pyrantel, and ivermectin can be practiced; after the foal is six months old, moxidectin can be used as well. Fecal exams should be performed on a representative sample of the juvenile population after each scheduled deworming to monitor effectiveness of the control program. Most horses develop excellent acquired immunity to ascarids by the time they reach 18 months of age.

Strongyles—No special program is required for juvenile horses, other than to use a macrocyclic lactone (ivermectin or moxidectin) at least once every five months to help eradicate large strongyles from the herd. If the farm population of small strongyles is resistant to benzimidazoles and/or pyrantel, the efficacy against strongyles might be less than satisfactory whenever these products are used at 60-day intervals against ascarids. Horses remain susceptible to strongyle infection for their entire lives.

Pinworms—Proper implementation of the recommendations for ascarids should achieve effective control of pinworms. Horses develop acquired immunity against adult pinworms around the second or third year of life.

Tapeworms—After weaning, treat juveniles during spring and/or autumn with a compound containing praziquantel. Some horses apparently develop immunity to tapeworms, some maintain small burdens if not treated, and about 10% of horses will harbor large numbers of tapeworms for life regardless of deworming.

In the next issue will be our final installment in our parasite series: A realistic look at parasite control programs for mature horses.

Foal Deworming Timeline

	ANTHELMINTIC	PARASITES OF CONCERN
1-2 weeks	Ivermectin	Threadworms
2 months	Pyrantel pamoate or benzimidazole	Ascarids and Pinworms
	(subsequent treatments with rotational deworming every 60 days until 15-18 months old)	
5 months	Macrocyclic lactone (ivermectin, moxidectin)	Strongyles
	(and every 5 months thereafter)	
after weaning	Praziquantel	Tapeworms

CONTROL PROGRAMS FOR MATURE HORSES

Get Your Veterinarian Involved in Deworming

BY KAREN BRIGGS, WITH
CRAIG REINEMEYER, DVM, PHD;
DENNIS FRENCH, DVM, MS, DIPL. ABVP;
AND RAY KAPLAN, DVM, PHD

PARASITE PRIMER—PART 12

In last month's article, we discussed certain parasites that are found almost exclusively in horses less than two years of age—roundworms, threadworms, and other nasties that target the naïve immune systems of youngsters. After equines reach their second birthday, however, the picture changes dramatically, as large and small strongyles become the major focus of our parasite control programs. There are other parasites that are a concern in adult horses—bots and tapeworms, to name only two—but their impact is fairly minor in comparison to strongyles (see "Major Parasites Affecting Adult Horses" on the page 51).

Knowing how dangerous strongyles are, we want to be able to keep our horses strongyle-free as much as possible. But are our current parasite-control programs doing the job?

In far too many herds, parasite control for adult horses can be characterized as compliance with a recipe. It is still common practice at many farms to

JANICE TREMPPER

Getting your veterinarian involved in the proper deworming procedures of your horses is the first step to healthier animals.

Major Parasites Affecting Adult Horses

COMMON NAME	SCIENTIFIC NAME
Large Strongyles	*Strongylus spp.*
Cyathostomes (small strongyles)	*Cylicocyclus, Cyathostomum, Cylicostephanus, Coronocyclus, Cylicodontophorus, Poteriostomum, Petrovinema spp.*
Bots	*Gasterophilus intestinalis* and *G. nasalis*
Tapeworms	*Anoplocephala perfoliata*

deworm all horses with Drug A, then wait a stereotypic interval before all are treated with Drug B. After another period of identical duration, horses are treated yet a third time, perhaps with Drug C, or maybe another dose of A or B, and the pattern is repeated throughout the entire calendar year, according to a rotation recipe that is now decades old.

Such rote approaches to parasite control for mature horses are long overdue for major revision for several reasons. First, recipes cannot determine whether dewormers A, B, or C are still effective in a particular herd. Second, a standard rotation formula also fails to exploit the unique characteristics of dewormers A, B, and C, such as differing durations of egg count suppression following treatment. Third, rote programs usually don't attempt to discriminate between horses pastured in Florida and those stabled in Saskatchewan. Finally, recipes do not acknowledge that individual horses vary widely in their susceptibility to parasites, that their contributions to environmental contamination are distinct, and that their respective parasite control needs might differ from the rest of the herd.

With resistance patterns to anthelmintics changing, and an ever-shrinking spectrum of effective drugs to choose from, there's an element of urgency to implement some important changes in parasite control practices for mature horses. You might find some of the suggestions in this article radical, even heretical, but we contend that adoption of these methods will simultaneously provide superior parasite control and decrease selection for anthelmintic resistance, and it could probably accomplish both at a lower cost than current practices.

Target Parasites

The biology of the major parasites affecting adult horses was discussed in earlier installments of this series, but let's review the important details.

Tapeworms—As we've noted previously, tapeworms (*Anoplocephala perfoliata*, the only major variety of cestode, or flatworm, to infect horses) were long thought to be very minor players in the equine parasite game. Recent studies have given us a clearer picture of their significance and the harm they do.

Tapeworms attach fiercely to the intestinal wall, which can cause severe inflammation at the attachment site. They're associated with several types of severe colic, especially ileocecal intussusceptions. In an intussusception, the last one foot of the small intestine (ileum) telescopes into the first section of the large intestine (cecum) and swells, blocking passage of the intestinal contents. The worms can sometimes cause impaction colic, and it's suspected that chemicals they release might interfere with normal gut motility.

Structurally, tapeworms are made up of egg-containing segments, or proglottids. The separate units are like the box cars making up a train. As proglottids mature, they break off from the end of the worm either singly or in small groups without killing the worm, which remains attached to the intestinal wall.

Tapeworms have indirect life cycles, meaning that they must develop within a different animal before they can be transmitted to their final host. Horses acquire tapeworms when they swallow tiny oribatid mites, which live in vast quantities on many pastures. The worms then grow and mature in the equine digestive tract over a six- to 10-week period.

Evidence-based recommendations for tapeworm control have not been generated to date, but many experts recommend at least one annual treatment with a dewormer containing praziquantel during spring or autumn, for horses from weaning age on up. Where there is no evidence of tapeworms causing health problems, one treatment each year might be enough to keep tapeworms at a low level. A second or third treatment might be advisable on properties where tapeworm eggs are frequently seen on fecal egg counts, or where there is a history of tapeworm problems.

Bots—The larvae of the bot fly are the most significant "non-worm" parasites harbored by horses.

MARIANNE SLOET; INSET—LEE TOWNSEND

The bot fly lays its eggs on the hairs under the jaw and on the hairs of the horse's forelegs and shoulders. The eggs hatch when horses rub their warm muzzles on their legs. (Inset) Bot flies are common everywhere horses are kept and their larvae can do significant damage in the oral cavity and stomach.

Interval After Deworming with Various Anthelmintics To Perform Fecal Egg Counts for Determining the Strongyle Contaminative Potential of Individual Horses

ANTHELMINTIC	EXPECTED EGG REAPPEARANCE PERIOD	STRONGYLE CONTAMINATIVE PERIOD
Benzimidazoles (fenbendazole, oxfendazole, oxibendazole)	4 weeks	8 weeks
Pyrantel salts	4 weeks	8 weeks
Ivermectin	8 weeks	12 weeks
Moxidectin	12 weeks	16 weeks

CONTROL BY THE MONTH

Parasite Control for Adult Horses in the United States

It is best to think of worm control as a yearly cycle, starting at the time of year when worm transmission to horses changes from negligible to probable. In the South, this is in late summer/early autumn as temperatures begin to drop. Intestinal strongyles of horses simply do not survive and develop on pasture to any significant level during the hot summers in the South. The goals of the program are to: Keep fecal egg counts (FEC) low, thereby reducing future worm transmission; kill all important parasites at the correct time of the year; and reduce the development of drug resistance.

This is one of many possible programs, and there is room for different opinions. Ultimately, each farm (with veterinary guidance) should develop a program tailored to its needs. Before a rational program can be developed, one must know which drugs work. The only means to learn this information is to perform a fecal egg count reduction test (FECRT; see "Diagnosis: Examining the Evidence" in the June 2004 issue, www.TheHorse.com/emag.aspx?ID=5193, for how to perform one).

This suggested program is only valid in a hot southern climate. For a northern climate, the ideas behind the program (and most of the recommendations) will be the same, but the cycle is different. The first treatment should be given in April and the last treatment can be given in October or November.

SEPTEMBER

In southern climates, September starts the worm control cycle. Treat all horses regardless of FEC with ivermectin or moxidectin. These drugs kill bots acquired since spring, the stomach worms *Habronema* and *Draschia* (which are transmitted by flies and cause summer sores), and sterilize *Onchocerca* females. These drugs also kill migrating large strongyles and any strongyles (large and small) in the intestinal lumen.

Is there a reason to use one over the other?

Moxidectin has the advantage of also killing large numbers of the encysted small strongyles, and it prevents worm eggs from reappearing in the feces for four weeks longer than ivermectin. However, at that time of the year, there are few cyathostome larvae on pasture. Therefore, some parasitologists are concerned that using moxidectin at that time of the year might place more pressure on drug resistance. If FEC are performed ahead of treatment, use moxidectin only in horses with FEC greater than 500 or in horses known to have chronically high FEC, and use ivermectin on the remainder.

Should I perform FEC?

Yes, on all horses. This is probably the single most important FEC performed all year (assuming that you follow this program and have not dewormed in the past few months). The reason is that by not deworming for several months, the FEC seen will be a strong indicator of each horse's innate immunity of small strongyles. Based on this FEC, you can categorize your horses as low (less than 150 epg), moderate (150-500 epg), and high (greater than 500 epg) worm egg shedders. This characteristic of individual horses has been shown to be repeatable between years.

NOVEMBER/DECEMBER

In November/December, treat all horses with FEC greater than 150 epg based on the September fecal check. If your horses were treated with ivermectin in September, treat again in November. If moxidectin was used, wait until December to treat.

Use oxibendazole and/or pyrantel (if effective on your farm). Choose these drugs to reduce the amount of ivermectin and moxidectin used, thereby helping slow the development of resistance to ivermectin and moxidectin. However, there is resistance to oxibendazole and pyrantel, so if using these drugs, before and 10-14 days after treatment a FEC should be performed. Recent data suggests that using oxibendazole and pyrantel together improves the effectiveness of treatment over treatment with the individual drugs.

Is there a reason to use one over the other?

Not unless resistance to one of these drugs is detected.

Should I perform FEC?

Yes, but only on the treated horses (FEC greater than 150 in September). If you haven't performed FECRT previously, FEC should be checked again 10-14 days after treatment.

DECEMBER

Treat all horses regardless of FEC with ivermectin/praziquantel or moxidectin/praziquantel. Tapeworm transmission peaks in autumn, so treatment with praziquantel at this time will remove all the tapeworms acquired over the summer and autumn. Praziquantel is the only FDA-approved drug for tapeworms in horses and is only available in combination with ivermectin or moxidectin. Other reasons to choose one of these combination products are: First, bot transmission will have ended (except in Florida), so treatment with ivermectin or moxidectin will remove the bots acquired since September, and no new bots will infect horses until spring, when the bot flies become active; second, any of the worms targeted in the September treatment that are picked up in the intervening months will be killed.

Is there a reason to use one over the other?

By December, small numbers of strongyle eggs have been deposited on pasture, so there is larval buildup. Therefore, resistance is less of a concern and moxidectin and ivermectin are both reasonable choices.

Should I perform FEC?

Yes, on all horses. It is important to know if the horses with low FEC in September still have low FEC, and if you have been successful in keeping FEC low in the horses that had high FEC in September.

FEBRUARY

If you treated with moxidectin/praziquantel in December, wait until March. If you treated with ivermectin/praziquantel in December, treat again. Only treat horses with FEC greater than 150 epg unless the horse is known to be a chronically high egg shedder. Use moxidectin for all horses that consistently have had the highest FEC (only if moxidectin was not already administered in December; if moxidectin was administered in December, then wait until March to decide what to do). Instead of moxidectin, oxibendazole and/or pyrantel can be used on the horses that have FEC greater than 150, but have not shown high FEC through the year.

Is there a reason to use one over the other?

Horses with chronically high FEC likely also have many encysted small strongyles, and moxidectin has the greatest efficacy against

(continued on page 56)

IS YOUR HORSE AN OFFENDER?

Determing a Horse's Strongyle Contaminative Potential

Individual horses differ widely in their contributions to pasture contamination. The relative magnitude of contamination, as measured directly by quantitative fecal egg counts, is a repeatable characteristic of individual animals. In herds that have not been dewormed recently, certain horses (approximately 20-30% of the herd) have high egg counts, another proportion will have low egg counts (30-50%), and the remainder cluster around the average.

Horses with egg counts less than 150 eggs per gram (EPG) are classified as Low Contaminators, and those with EPGs greater than 500 EPG are classified as High Contaminators. The remainder of horses, with EPGs between 150 and 500 EPG, are classified as Moderate Contaminators.

The contaminative potential of a horse can be determined by examining a fecal sample collected approximately four weeks after the expiration of the Egg Reappearance Period for the last effective anthelmintic it received (see table on page 53). Of course, this might require a break in scheduled deworming treatments, but no negative consequence will result if this occurs during mid-summer for southern horses or in mid-winter for those stabled in the North.—*Karen Briggs*

Bot flies (*Gasterophilus* spp.) are common virtually everywhere horses are kept, with two major species found in North America: *Gasterophilus nasalis*, which lays its eggs on the hairs of the intermandibular space (under the jaw); and *Gasterophilus intestinalis*, which lays its eggs on the hairs of the horse's forelegs and shoulders. *G. nasalis* larvae hatch spontaneously and crawl their way to their host's lips, while *G. intestinalis* eggs hatch when horses rub their warm muzzles on their legs, with the larvae quickly entering the mouth and burrowing into the tissues of the tongue.

Bots can do significant damage both in the oral cavity, where they spend their first month or so of life, and in the stomach, where they set up shop in their final larval stage. They attach to the stomach wall with mouth hooks and spend up to nine months drawing nourishment and ulcerating the tissues. Bot larvae then pass out with the manure to pupate in the soil in late spring, and adult flies emerge by mid-summer to start the cycle anew.

Larval bots must enter the host before winter so they can spend the colder months in the relatively stable habitat of the equine gastrointestinal tract. That's why bot infections are usually managed by using boticidal drugs (e.g., ivermectin or moxidectin) during autumn or early winter.

Large Strongyles—The most dangerous of the equine parasites are the large strongyles, or bloodworms. The three major species of large strongyles (*Strongylus vulgaris*, *S. edentatus*, and *S. equinus*) count among their various crimes against horses such severe symptoms as anemia, liver damage, damage to the cranial mesenteric artery from verminous aneurysms, and severe thrombo-embolic colics.

Furthermore, few worms are more prolific. Female strongyles lay eggs almost constantly, re-infecting your pastures again and again. The eggs hatch in fecal piles and the larvae go through three stages before they become infective, at which point they crawl up blades of grass so they can easily hitch a ride with a juicy mouthful. Horses can also ingest the larvae directly from the soil or from drinking contaminated water. (There can be dozens of large strongyle—and other parasite—larvae in a single drop of water or dew.)

Once inside the horse, large strongyles take an extensive tour of the equine innards, their exact route determined by their species. Regardless of their migration path, they eventually (over the course of six to 11 months) return to the gut to mature and lay eggs. The eggs pass out with the manure, and the cycle begins again.

In some ways, our battle against large strongyles has been very successful. During the past 15-20 years, most horse owners managed to eradicate large strongyles from their herds without realizing they had done so. The use of macrocyclic lactone dewormers (ivermectin or moxidectin) or the larvicidal regimen of fenbendazole (10 mg/kg daily for five days) kills virtually all adult and migrating large strongyles within a horse. Because it takes a minimum of six months to replace a population of reproducing adults, there will be no environmental contamination with large strongyle eggs if all horses on a farm are treated with one of these regimens at intervals of six months or less. Also, because the maximal survival of large strongyle infective larvae on pasture in the continental United States is approximately one year, the use of larvicidal regimens at intervals of six months or less for a total duration of 18 months or longer will effectively eradicate all large strongyles on a farm.

ANNE EBERHARDT

Contrary to popular belief, conditions from autumn through winter are very favorable for the persistence of infective larvae on pasture. Extra management precautions still need to be kept in place to prevent horses from becoming re-infected with larvae after being dewormed.

CONTROL BY THE MONTH (CONTINUED FROM PAGE 54)

these. If a larvicidal treatment is not needed, then use oxibendazole and/or pyrantel for the same reasons these drugs were recommended for the November treatment.

Perform FEC on all horses with greater than 150 epg on the December fecal exam.

MARCH

Treat only horses with FEC greater than 150 epg. The drugs used will depend largely on which drugs were used in the last few months. If tapeworm eggs are seen or the farm has a history of tapeworm problems, give a second treatment for tapeworms using one of the praziquantel products.

Should I perform FEC?

Yes, on all horses that were not treated in February.

APRIL

Treat only horses with FEC greater than 150 epg. The drugs used will depend on which drugs were used for previous treatments.

Perform an FEC on all horses. This is the last treatment of the cycle and the last time you will need to perform FEC until September. Eggs shed after April are doomed to die because temperatures will soon rise to levels that will kill them. This FEC will give you a good indicator of how well you have done controlling worms this year.

Note: *A five-day double dose of fenbendazole (marketed as the Panacur PowerPak) is a moxidectin alternative for removing encysted small strongyles in the spring. However, resistance is known to be quite common to fenbendazole at the single dose rate, and there is mounting evidence that the extended double-dose regimen often fails to provide high levels of control. If you use the PowerPak, do so no more than once a year, and only in a small percentage of your horses, to prevent severe resistance from developing.*

SUMMARY

This program is designed to target bots, tapeworms, *Habronema, Draschia, Onchocerca*, large strongyles, and small strongyles. A few lesser important parasites will also be controlled. Treatments in September and December with ivermectin or moxidectin (with or without praziquantel) will control all of the worms listed above for the entire year with the exception of small strongyles. So all that is left is to do after December is control small strongyles. Horses with naturally strong immunity to small strongyles (demonstrated with low FEC each exam) need no other treatments. In traditional deworming programs, repeated treatment of these horses accomplishes little to nothing.

Some horses will need a third treatment for small strongyles, but only a few horses (probably less than 30%) should need a fourth or fifth treatment, and only 5% or less should need more than that. Compare this protocol to what you are doing now. Many farms are treating all horses six times each year, and likely are getting results that are significantly less than what will be achieved on the program recommended here.—*Ray Kaplan, DVM, PhD*

This program works because the drugs kill all stages within the equine host, scheduled treatments prevent new arrivals from re-contaminating the environment, and time ultimately depletes any potential new infections (larvae) already on pasture.

So many horse farms have implemented deworming practices that are at least this rigorous that large strongyles are now considered a rarity in well-managed herds. It is relatively simple to maintain a large strongyle-free farm. All new animals should be confined upon arrival, treated with a larvicidal regimen, then held off pastures for a minimum of four days. This program will keep large strongyles out of the picture as long as our present dewormers keep their adulticidal and larvicidal properties against *Strongylus spp*.

Small Strongyles—The 40-odd species of cyathostomes (small strongyles) that infect horses are less driven by wanderlust than their larger cousins. Instead of taking the migratory approach, they set up housekeeping in the gut immediately and provide themselves with defenses that make it practically impossible for the horse's immune system to attack them.

Shortly after being swallowed, small strongyle larvae invade the lining (mucosa) of the large intestine, where a thin, tough capsule of scar tissue forms around each worm. These cysts simultaneously protect the larvae from the host's immune reactions and the majority of equine dewormers that are currently marketed. Once securely encysted, small strongyle larvae can enter a period of dormancy or continue to develop, depending on how many other small strongyles are already present in the environment of the horse. All eventually emerge, however, to morph into adults in the lumen (cavity) of the gastrointestinal tract. Individual worms could lay thousands of eggs in one day. An entire population of worms in the horse could produce over a million a day, which are then shed in the manure.

Although small strongyles generally cause less damage than their larger cousins, they can be responsible for intensely irritated intestinal tissues, diarrhea, weight loss, and anemia. Another danger is the risk of huge numbers of them emerging from the intestinal tissues all at once after the adult population dies off (either through "old age" or by being purged with a deworming drug). In essence, the act of deworming can trigger the next wave of larval emergence from the gut wall within a very short period of time (usually seven to 10 days).

On occasion, a severe syndrome called larval cyathostomosis occurs with the synchronous emergence of many small strongyle larvae; it can produce sudden-onset diarrhea, impaired gut motility, weakness, muscular wasting, and serious colic. Rarely, horses can suddenly die with few outward symptoms of disease, the cause being revealed only on necropsy. Larval cyathostomosis has a guarded prognosis at the best of times, and it is now considered one of the most serious parasite-related diseases in horses, making small strongyles a much more deadly foe than we once thought.

We haven't had the same success in controlling small strongyles that we have enjoyed with their larger relatives. Cyathostmes are ubiquitous in pastured horses, and they present the greatest challenge to effective parasite control for adult equines. Their life cycle within the host features extremely persistent larval stages that are not consistently susceptible to any dewormer. Drug resistance is also beginning to make certain classes of anthelmintics ineffective against some populations of small strongyles.

As a result, control recommendations for cyathostomes have to be based on a number of complex factors. Let's look at each in turn, then try to form some coherent parasite control recommendations for mature horses in various regions of North America.

1. Objectives of Control. It's an interesting exercise to ask horse owners, "Why should

Anthelmintics with Adulticidal or Larvicidal Activity Against Cyathostomes

DRUG ACTIVITY	CHEMICAL NAME
Adulticidal	Benzimidazoles, fenbendazole (5 mg/kg), oxfendazole (10 mg/kg), oxibendazole (10 mg/kg), macrocyclic lactones, ivermectin (0.2 mg/kg), moxidectin (0.4 mg/kg), tetrahydropyrimidines, pyrantel pamoate, pyrantel tartrate
Larvicidal	Moxidectin (0.4 mg/kg), fenbendazole (10 mg/kg daily for 5 days)

you control parasites?" Most will offer a response that includes some reference to improved health or enhanced performance. But the answers differ if one refines the question and asks, "What are you specifically trying to do when you give a dewormer?" The most frequent answer is, "To kill worms." However, killing worms per se is *not* the objective of a parasite control program. This is especially true for cyathostomes. It is actually the larval stages, encysted in the mucosa and impervious to most dewormers, that cause the most damage to the horse, and the worst of these effects is when the larvae emerge to repopulate the lumen. The adult worms in the intestinal lumen that are shedding the eggs are much less damaging than their immature siblings.

KIM AND KARI BAKER

The true objective of parasite control is preventing contamination of the environment with the eggs of the target parasites. Once strongyle eggs turn into infective larvae, the only factors that can diminish the risk of future infections are hot weather, time, and keeping horses off the pasture.

The true objective of parasite control is to prevent contamination of the environment with the eggs of the target parasites. Once strongyle eggs turn into infective larvae, the only factors that can diminish the risk of future infections are hot weather, time, and keeping horses off the pasture. The single practical way to decrease future infection is to limit the passage of worm eggs by killing female worms before they reproduce. So that's what we aim for with cyathostome control recommendations: Limiting the passage of large numbers of strongyle eggs onto pasture.

2. Environmental Factors. Because larvae encysted in gut tissues are not consistently susceptible to dewormers, all horses pass strongyle eggs in their manure at a predictable interval after treatment. Regardless of the anthelmintic used, it is impossible to "clean out" a horse's parasite burden by deworming.

When strongyle eggs pass into the environment, their ultimate infectivity is controlled by environmental conditions. In the southern United States, the climate is most conducive for hatching of strongyle eggs and their development into infective larvae during autumn and spring. In winter months, larvae can survive on pasture for long periods of time, but in the summer, larval development and survival are poor. In the north, environmental conditions are most conducive for hatching of strongyle eggs and their development into infective larvae during the spring, summer, and autumn. Contrary to popular opinion, conditions from autumn through winter are very favorable for the persistence of infective larvae on pasture.

During certain seasons (i.e., summer in the south) or in particular management conditions (i.e., stabling during winter in the North), strongyle eggs can't develop into future parasites. Under these circumstances, it's harmless for horses to pass large numbers of strongyle eggs in their manure, because those eggs don't turn into future parasites. At these times, anthelmintic treatments can be reduced or even discontinued.

3. Host Factors. Individual horses vary widely in their susceptibility to cyathostome infections, and fecal egg counts (FEC) will reflect those differences. The majority of the parasites in any group of animals are concentrated in a minority of the animals. Traditionally, however, all horses in a herd have been treated exactly the same when it came to parasite control. When one considers this practice critically, it should be obvious that routine deworming is unnecessary for some members of the herd; at best this practice is cost-inefficient for the animals that receive more treatment than necessary, and at worst it promotes drug resistance. It is also likely that the same programs that are excessive for some horses will prove to be suboptimal for highly susceptible members of the herd.

Fortunately, it is possible to categorize the potential of members of a herd to contaminate a pasture with strongyles. The timely determination of quantitative fecal egg counts can identify the troublemakers as well as the easy keepers (see "Determining a Horse's Strongyle Contaminative Potential" on page 55).

Quantitative fecal examination is *absolutely essential* if one intends to approach parasite control in a logical, medically based fashion. However, most equine practices probably don't offer this procedure for their clients at the present time. Let your veterinarian know that you want this service and are willing to support it (pay for it). Diagnostic testing isn't free, but the expense might be offset by money saved when eliminating some unnecessary or ineffective anthelmintic treatments.

4. Anthelmintic Issues. The first step is to determine which anthelmintics are effective in your herd. This can be accomplished by Fecal Egg Count Reduction Testing, or FECRT (see "Diagnosis: Examining the Evidence" in the June 2004 issue, www.TheHorse.com/emag.aspx?ID=5193, for

this diagnostic technique) with all of the major drug classes that are effective against cyathostomes (see "Anthelmintics with Adulticidal or Larvicidal Activity Against Cyathostomes" on page 57).

You also need to become familiar with the expected egg reappearance periods (ERPs) of the various anthelmintic products (see "Interval After Deworming with Various Anthelmintics to Perform Fecal Egg Counts for Determining the Strongyle Contaminative Potential of Individual Horses" on page 53). ERP is an extremely important concept in small strongyle control. As we've emphasized previously, most dewormers don't kill the encysted larval stages, and even those dewormers that are approved for this purpose do not completely eradicate the larvae. Therefore, when you treat a horse with an effective dewormer that removes adult worms from the intestinal cavity (lumen), encysted larvae already lurking in the intestinal wall are primed and ready to repopulate the gut lumen.

ANNE EBERHARDT; INSET–SARAH JACKSON

If we can learn to deworm horses according to the fecal egg count evidence rather than the calendar, the end result might just be less reliance on anthelmintics, which will help preserve their effectiveness and stave off those ever-looming resistance issues.

It takes several weeks for emergent worms to become sexually mature and begin egg laying. The time between treatment and when eggs reappear in feces is the ERP (egg reappearance period), and the ERP differs depending on the drug you use. If the time between treatments exceeds the ERP for the drug last given, then egg shedding onto pasture will occur, guaranteeing that horses will always be infected.

Since ERPs differ for the different dewormers, using standard intervals between treatments in a rotation will likely fail to adequately control egg shedding in many horses, and this is your primary objective. It is also important to appreciate that if there is no egg disappearance, then egg reappearance becomes a moot point. High levels of resistance to many common dewormers means that you won't achieve egg disappearance with your treatments, and thus will again fail to adequately control egg shedding.

5. Management Issues. Standard parasite control measures are always helpful. These include providing safe pastures. What's a "safe" pasture? That's one that has been left vacant for at least two months during the warm season of the year, one used recently to produce hay, or a pasture that was grazed by an alternate livestock species, such as cattle or sheep.

If possible, horses should not be fed directly off the ground. Manure collected from stalls or from paddocks should be composted for at least several weeks before it is spread on occupied pastures. Pastures should never be harrowed (dragged) while they are occupied by grazing horses, and harrowing should only be performed during the warmest months of the year.

Take-Home Message

In the past 12 months, we've taken a thorough and exhaustive look at the current state of parasitism in North American horses. If there is only one message you take home from all of the information we've presented, it should be that there is a pressing need to change our thinking about parasite control methods and recommendations.

We've gotten used to deworming our horses by rote and without much veterinary intervention; now it's time for veterinarians to become more involved again, with informed consultation and monitoring services. Horse owners also need to become more proactive about parasite control and to be willing to pay for that consultation and monitoring instead of just buying another tube of anthelmintic paste and trusting it to do the job.

Time-honored health care routines can be hard to shake. But if we look at the data and recognize the shortcomings in the traditional approach, we can become more open to the idea of deworming according to the FEC evidence rather than the calendar. The end result might just be less reliance on anthelmintics, which will help preserve their effectiveness and stave off those ever-looming resistance issues. That's a goal few could argue—especially when it also results in healthier, less parasitized horses.