ASCARIDS:

A Growing Problem

PARASITE PRIMER—PART 3

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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.

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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.

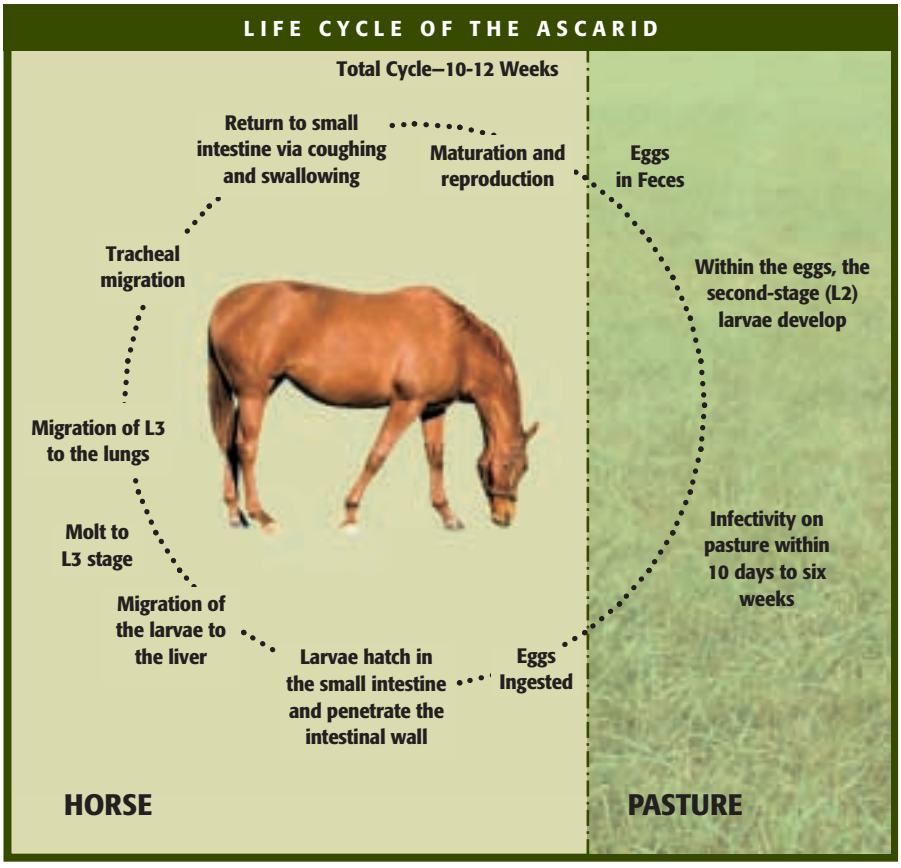
Equine Ascarids

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

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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 yearling horses, 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 4), 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.

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