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).
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 L3 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...
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.

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**What Kills Larval Strongyles?**

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*Source: Craig Reinemeyer, DVM, PhD*