Strongylus vulgaris, S. equinus, S. edentatus

Large strongyles. *Strongylus vulgaris* is more commonly called the bloodworm

**Description:** These worms are stout-bodied. They range in length from 2 centimeters (*S. vulgaris*) to 5 centimeters (*S. equinus*). Fresh specimens are reddish-brown due to the ingestion of blood from the host.

**Predilection sites:** Colon and cecum.

**Geographic distribution:** Very common throughout the US.

**Life cycle:** The eggs of all three species develop to infective L₃ larvae on pasture. Moisture is critical for larval survival, and warmth speeds development to as little as 3 days. The host ingests L₃ larvae, which shed their protective sheaths in the small intestine. From this point, each of the large strongyle species develops according to a different pattern.

*S. vulgaris:* L₃ larvae of *S. vulgaris* penetrate the intestinal mucosa, where they molt to L₄ larvae in several days. These fourth-stage larvae penetrate nearby blood vessels and migrate through the arteries for about two weeks before reaching the anterior mesenteric artery, a major blood vessel that carries oxygenated blood to the intestine. The prepatent period is 6 to 8 months. However, *S. vulgaris* eggs have been seen in the feces of young foals as early as 20 weeks.

*S. equinus:* L₃ larvae of *S. equinus* burrow into the submucosa and molt to the L₄ stage, then migrate to the liver where they wander for 6 to 7 weeks. Emerging from the liver, they molt to become immature adults in various abdominal organs, eventually returning to the colon. The prepatent period is about 9 months.

*S. edentatus:* Larvae penetrate the intestine and migrate through a vein to the liver, where molting to the L₄ larval stage occurs. After about 9 weeks, the L₄ larvae begin to migrate in the peritoneum, causing the formation of nodules. Next, they migrate back to the colon, form nodules in the gut wall, and then rupture the nodules to enter the lumen of the colon.

**Significance:** The large strongyles are the most important parasites of horses, exerting a significant economic impact wherever horses are raised. Of the three species, *S. vulgaris* is the most pathogenic. In some areas, *S. vulgaris* affects 85 to 90 percent of horses, particularly yearlings. For these reasons, it is considered to be the most important of all equine endoparasites.

**Clinical effects on host:** Larvae irritate the walls of the small intestine and the arteries. Adult worms are “plug feeders,” meaning that they feed by ingesting plugs of mucosal tissue and capillaries. Heavy feeding of this type produces intestinal damage, anemia, anorexia, depression, weight loss and dehydration.

Each of the three species also produces other specific clinical effects. *S. equinus* and *S. edentatus* cause liver damage and peritonitis.

As noted, *S. vulgaris* larvae (the L₃ stage) live within the lumen of the horse’s colon, causing damage as they feed on the host’s blood. However, the most serious damage is caused by the L₄ larvae, which penetrate the network of mesenteric arteries and begin a 4-month cycle of migration through these blood vessels. As they travel through the mesenteric arterial network, the larvae produce irritation of the blood vessel walls, inducing the formation of lesions.

These arterial lesions can become severe enough to cause a thickening of the arterial walls (verminous arteritis), a condition that may result in blood clotting and, subsequently, thrombosis. In addition, portions of these clots may break off, travel to, and lodge in other blood vessels, cutting off the blood supply in various other parts of the body.

Should the blood supply in the intestines become impaired, the result is a serious condition called intestinal colic (verminous colic). Death can occur when this series of events progresses to include disintegration of intestinal tissue and the development of gangrene of the bowel. Weanlings and yearlings are particularly susceptible to verminous colic.

**Diagnosis:** Larvae are needed for species identification. After adult worms have developed, eggs can be found in feces. Diagnosis is difficult during the pre-patent, migratory phase.

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