

MU Guide

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Common Internal Parasites of Cattle

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Although there are many species of worm parasites harbored in the gastrointestinal and respiratory tracts of cattle, only a few target species are clinically and economically important. These include the brown stomach worm *Ostertagia*, the coccidia *Eimeria bovis* and the lungworm *Dictyocaulus*. These are of great significance throughout the United States and worldwide, with *Ostertagia* considered the single most important parasite of cattle. And this is true for native Missouri cattle as well as for cattle imported into Missouri.

Clinically, the parasites of the stomach and intestine cause anemia, scouring, depression and even death, but clinical parasitism occurs infrequently. The effects of parasitism usually are insidious and subclinical, such as indigestion and poor feed conversion, less than expected weight gain and (for brood cows) decreased milk production. Lungworms cause verminous pneumonia and provide an environment conducive for viral and bacterial pneumonia, with labored breathing and anxiety evident. Depressed performance then may be a consequence of internal parasitism.

Ostertagiosis

The principal worm parasite of beef and dairy cattle is *Ostertagia*, a very small (1/2 inch) brown worm found on the lining and in the gastric glands of the abomasum (the true stomach). Adult worms graze the lining or mucosa and cause irritation and fluid loss, interfering with the digestive function of the stomach. Dyspepsia or "heartburn" results so that feed, especially hot rations such as grain, is not tolerated well. Feed conversion for weight gain, body maintenance, reproductive fitness and milk production is then compromised. Larvae or immature worms invade the gastric glands, which are pits in the lining of the stomach, disrupting normal structure and function of these glands. This compounds the effects of parasitism.

Adults lay microscopic eggs, which are passed in the feces and hatch in dung pats. Microscopic larvae are then dispersed, with some trickling deep into the soil and others carried onto surrounding grass. The free-living larvae feed on fungi and other soil and grass microorganisms and represent "pasture contamination."

When these larvae have developed to an infective stage, they may be found on blades of grass in dew drops early in the morning and seasonally in late spring and late summer/early fall. Cattle become infected while grazing. Upon passage of grass to the stomach, infective larvae penetrate into gastric glands. Larvae usually emerge from the glands in relatively small numbers after one to two weeks, then mature so that glandular disruption is not so dramatic and therefore is of less clinical consequence.

However, those infective larvae that are picked up in March and April in southern climates (most of Missouri) will remain in "arrested development" or as "inhibited" larvae for four to five months, effectively overwintering until late August to October. These larvae then rapidly increase in size, emerge in large numbers and cause massive destruction to the gastric glands, bringing stomach function to a virtual halt. The host cow or calf becomes clinically ill with anemia, accumulation of body fluid (edema) often seen as "bottle jaw," and loss of appetite. Some cattle are overwhelmed and die; others are set back for several months before adequate stomach repair can occur. In northern climates (Iowa, northern Missouri), overwintering of inhibited larvae occurs with fall pickup of "arrest-prone" infective larvae followed by late winter (February-March) clinical ostertagiosis.

If death or debility results from this infection, your veterinarian can necropsy the affected animal. Cobblestone lesions will be found on the lining of the stomach and the normally thin folds of the stomach

will be waterlogged or edematous. The cobblestone appearance is caused by irreparably damaged gastric glands. Affected stomachs may be submitted to the Veterinary Medical Diagnostic Lab (VMDL), College of Veterinary Medicine, for confirmative diagnosis.

To prevent occurrence of ostertagiosis (parasitic gastritis), a **strategic** deworming schedule has been developed by University veterinarians and adopted by the industry. The strategic program attacks the parasite cycle on a seasonal basis prior to occurrence of disruptive damage to the host. During the spring calving period and before summer pasture turnout, cows should be dewormed so that their parasite burden is virtually eliminated and pasture contamination is stopped. Fall calves and yearlings should be dewormed at this time also.

With inhibited larvae present, a dewormer with adulticidal and larvicidal effects needs to be administered. Dewormers with this property include the benzimidazoles or “white” dewormers such as albendazole (Valbazen®), fenbendazole (Safeguard®, Panacur®), and oxfendazole (Synantic®) and ivermectin (Ivomec®).

Midsummer deworming may be necessary for late spring calves or for other cattle missed in the spring. In the fall, cows and calves should be dewormed because of fall pickup of larvae. If you are in an area where overwintering of inhibited larvae occurs, such as in northern Missouri, one of the dewormers mentioned should be given. Thus cattle should be dewormed twice a year for best protection and to provide for optimal performance.

Adulticide-only dewormers such as levamisole (Levasole®, Tramisol®) and morantel (Rumatel®) may be given in the spring in a region with overwintering inhibited larvae and in the fall where oversummering inhibited larvae occur. Pour-ons and medicated feed blocks may be used to provide alternative and supplemental deworming formulations. Remember that incoming stocker calves to be backgrounded or cattle to be finished in the feedlot may originate from many places and should be dewormed upon arrival with an adulticide-larvicide dewormer.

As for pasture contamination, percolation of larvae into the soil provides for their protection, especially in the winter, and they will appear on grass following thaws. Missouri’s hot summers will usually kill off most larvae over summer. Regardless, cattle serve as reservoirs of infection for these periods of the year and mature parasite populations will contaminate forage.

Other stomach and intestinal worms also parasitize cattle. These too are relatively small in size, like *Ostertagia*. They include the barberpole worm *Haemonchus* in the stomach and the hookworm *Bunostomum* in the small intestine, both of which cause loss of blood and are considered warm-weather

parasites. Another worm parasite that could disrupt intestinal function is the twisted wireworm *Nematodirus* and the whipworm *Trichuris*.

A segmented, fleshy tapeworm, *Moniezia*, is transmitted by free-living pasture (grass) mites and is found in the intestinal tract, but in spite of its large size is probably of little consequence. As has been discussed, the lungworm *Dictyocaulus* parasitizes the respiratory tract, causing inflammation, emphysema and mucus buildup.

The benzimidazole dewormers are effective against these parasites so that a strategic deworming schedule for *Ostertagia* would also include the other worm parasites. Ivermectin would have less effect against *Nematodirus* and *Trichuris* and none against *Moniezia*, but the clinical effects of these parasites are debatable. However, ivermectin is very effective against the other worm parasites and as an ectoparasiticide. Levamisole would be effective against the adult gastrointestinal worms and *Dictyocaulus*, but not against larval stages or the tapeworm, and morantel only against adult gastrointestinal worms and their infective larvae.

Liver flukes (*Fasciola*) are occasionally brought into the state, but they are not native here and as far as is known are not transmitted here. Albendazole and ivermectin with clorsulon (Ivomec F®) are effective against immature flukes only.

Your veterinarian can examine fecal samples for eggs of these worm parasites and can quantify them on a per gram feces basis. By examining pre- and post-treatment samples from 10 to 20 percent of the herd, the dewormer’s effectiveness can be judged. To date, no worm resistance to dewormers has been reported for cattle in the United States, so annual rotation of drugs is not necessary. Few or no eggs does not mean that there is no infection; instead an immature, non egg-producing population of worms could be present and clinical or subclinical problems might still occur. Identification of worm species based on appearance and size of eggs has limitations in that all stomach and some intestinal worm eggs look alike.

Coccidiosis

These microscopic parasites infect the interior of cells lining the lymphatic blood vessels (lacteals) in the distal small intestine (ileum); later in their cycle they also infect the cells lining the ileum, cecum and proximal portion of the colon. These coccidia are collectively referred to as *Eimeria*, with *Eimeria bovis* as the target because of its common occurrence and potential for causing enteritis, including blood-tinged scouring.

The cycle begins with ingestion of a microscopic cyst (oocyst) that contains eight infective organisms which are released in the digestive process. These infective organisms rapidly penetrate the gut wall

and infect the lining of the lacteals, where each organism multiplies into a population of 100,000-plus so that the infected cell becomes dilated (hypertrophied) and functionless. Breakdown of infected cells occurs at 1-1/2 to 2 weeks following infection, releasing invasive parasites which then move to cells lining the gut. Fewer numbers of microorganisms are produced per cell, but millions of host cells may now have become infected. By 2-1/2 to 3 weeks, oocysts are produced and passed in the feces. By this time, scouring and blood loss could have occurred as there is breakdown in the integrity of the gut lining with dehydration and blood loss anemia.

Clinical disease intensifies with an increase in the number of oocysts ingested (dose dependency) and is enhanced by failure of the immune response in very young calves or calves not previously exposed to coccidia. Disease also is enhanced by the stress of weaning, feed and water deprivation, shipping and worm parasitism. Some light exposure allows for a mild infection and prepares an immune response to coccidia.

Anticoccidial drugs may prevent parasite invasion of cells and impair development. These are designed for the treatment of coccidiosis in the face of an outbreak or for prevention when conducive conditions prevail. Amprolium (Corid[®]) is administered in water or in medicated feed for five days for treatment and 21 days for prevention. Decoquinate (Deccox[®]) is for the prevention of coccidiosis and is fed for at least 28 days during an exposure or if one is anticipated. Lasalocid (Bovatec[®] Premix) is used for the control of coccidiosis. Sulfaquinoxaline (Sulfa-Nox[®], S.Q. Solution[®]) products may also be used for the treatment and control of coccidiosis outbreaks).

Because oocyst production is not directly affected by anticoccidial drugs, oocysts may continue to be passed for a short period following treatment. Because anticoccidial drugs are primarily coccidiostatic rather than coccidiocidal, resumption of cycling may occur for a short time but with fewer oocysts produced and passed. If contamination is minimized, reinfections will be lighter, of less clinical consequence and will allow for immune response.

