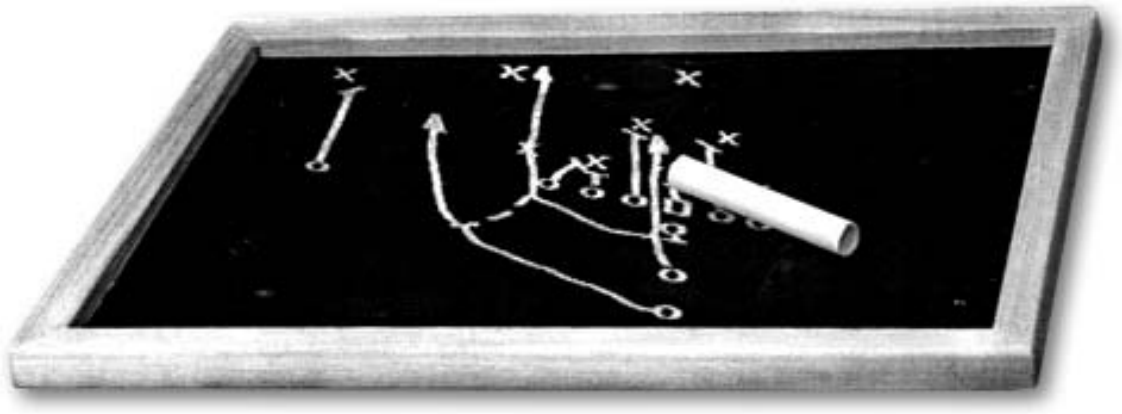


Don't Get Played

Understanding the brown stomach worm's playbook is key to beating this parasite at its own game.

Story by
CORINNE PATTERSON

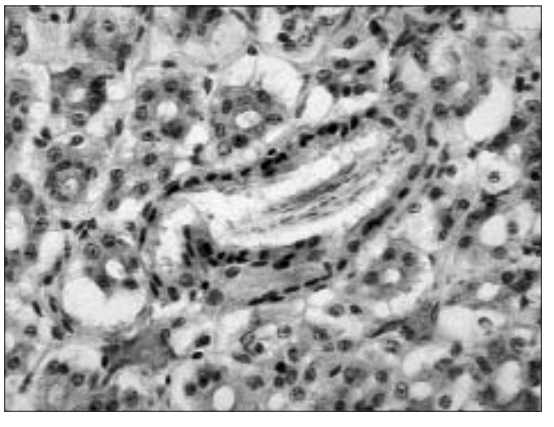
Ostertagia ostertagi is a tough opponent of the cattle industry. It's capable of running up the score against the nation's beef and dairy producers, costing them $10-$20 per animal when factoring in treatment costs and production losses, says Louis Gasbarre, a research scientist with the Agricultural Research Service (ARS), based in Beltsville, Md.

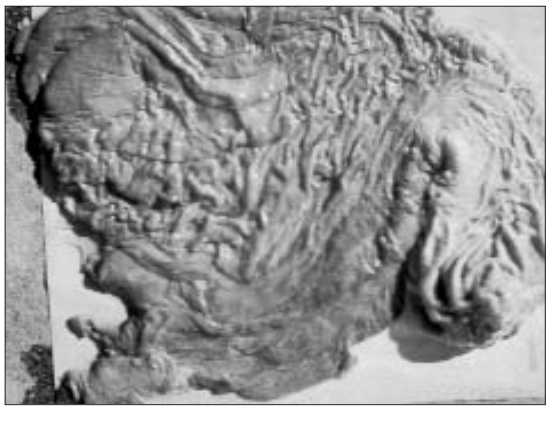
O. ostertagi, commonly called the brown stomach worm, thrives in temperate environments found throughout the United States. Thomas Craig, a professor of pathobiology at Texas A&M University, College Station, says there are a few rules, however, this roundworm must obey before any points can be scored.

"It plays the game a little bit differently in different geographic localities," Craig says, which makes understanding this parasite's life cycle within a specific locale key to controlling it. Producers will generally find that the most effective time to deworm for this parasite is during its developmental stage, while it is inside the animal and before it gets the chance to reproduce.

Gasbarre says, however, producers too often rely on the deworming process alone, which is costly and could contribute to problems in the future.

"One of the problems we have is that there are some extremely good products out there for treating for the worms, but in some cases they are being used improperly, and the potential for selecting for drug resistance is very high," Gasbarre notes. "So, it is important that the producer gets maximum effect with minimal numbers of treatments."





The adult worm (above) destroys the gastric gland when it exits. If the environment isn't ideal for eggs to survive, the worms will go into arrested development (right) and remain in the gastric glands until conditions improve. [PHOTOS COURTESY OF THOMAS CRAIG]

The playbook

This parasite is a nematode with a life cycle similar to an insect, Gasbarre says. It undergoes a metamorphosis, or a developmental process.

"Nematodes have five life cycle stages — the fifth being adult. The other four are larval stages," he adds.

Eggs enter the environment through fecal material via cattle that shed the eggs from adult worms living within their stomachs. An adult worm can reproduce for about 30 days. Craig says the first-stage larva (L1) develops within the egg, hatches and feeds on fecal bacteria through the second-stage larva (L2). It then molts and becomes a stage-three larva (L3). At this point the parasite is infectious to cattle, Craig says, but it has to use some strategy from its playbook to continue the game.

"At this point the larvae have to get away from that fecal pat and out onto the vegetation. This requires water because they don't have legs, so they can't crawl away from things. They can just move in a film of moisture," Craig says, noting damp spring and fall weather are ideal. "So, if we've had some rainfall, after that larva is developed it then moves away from the fecal pat and up on to the vegetation where cattle graze the parasite. One drop of dew can have hundreds, if not thousands, of larvae."

Once ingested, the worm then undergoes change while it is passing through the rumen and entering into the abomasum — the true stomach — as an immature larva. The L3 stage worm is very tiny, which allows it to easily enter the gastric gland, where it will continue to develop. Older literature regarding the subject, Gasbarre notes, indicates this point in time to be the most dangerous, but he warns this isn't the only stage that causes significant problems for cattle.

What is significant about that gastric gland, however, is its role in aiding digestion.

"The gastric gland produces two really important things," he says. "It produces a proteolytic enzyme called pepsinogen; then it produces hydrochloric acid."

For pepsinogen to become the active enzyme pepsin, it requires an acidic environment. Proteins enter the digestive tract as very big, complex structures, and pepsin is the first enzyme that begins breaking them down. Without pepsin those big proteins enter the small intestine, where the enzymes are less efficient in breaking them down.

"When the larva is in the gastric gland, it's destroying that gland, so it is interfering with the animal's ability to begin the digestion of protein," Gasbarre says. The animal's health is generally compromised, and it can lose weight and performance.

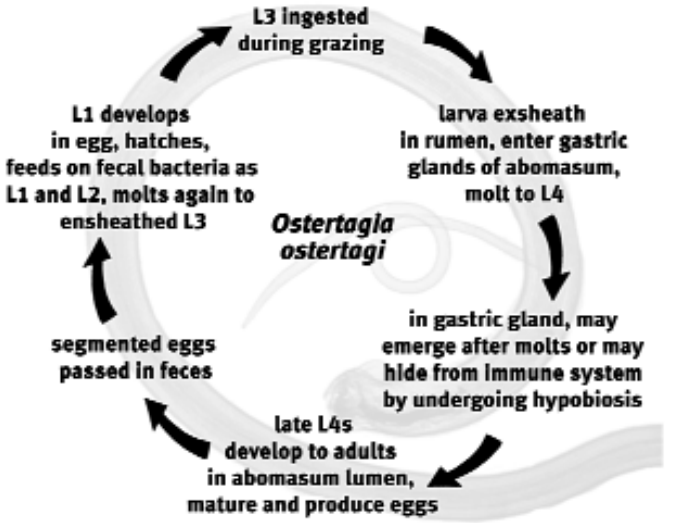
Once in the gland, the larva undergoes two molts (one to L4 and one to a young adult) to become an adult worm before its departure approximately 15 days later. "They come out just shortly before they are sexually mature and begin to reproduce," he says.

"The larva that went in was relatively small and penetrated the gland without much damage," Gasbarre says. "But when the adult worm now goes to leave, it's much, much larger, and it can't come back out through that little neck of the gland, so it tears its way out. If you look at the abomasum of an animal when the larvae are emerging, it looks a little bit like raw hamburger."

Once the adults are reproducing within the stomach, they are also causing trouble because they are tissue feeders, Gasbarre continues. "They are basically grazing on the surface of the mucosa. They are eating away the lining of the stomach, so the stomach becomes extraordinarily ulcerated." The stomach may become so damaged that it becomes nonfunctional.

This continuous cycle takes place when the *O. ostertagi* sense the environment is right for survival when temperatures are moderate and moisture is plentiful.

However, Craig points out, *O. ostertagi* also know when to take a



Source: *Thomas Craig*

time-out and delay the life cycle until conditions are better.

"Here's where we start seeing differences depending on where we are geographically and the age and immune status of the host," Craig says.

Another play

O. ostertagi can go into arrested development, a sort of hibernation known as hypobiosis that takes place within the gastric gland, Craig says. "Arrested development of early L4 larvae in the gastric glands enables the parasite to evade unfavorable conditions in the environment — extremes of hot, dry or cold — or immune response by the host."

"This is where the games that this parasite plays become important," he says. In the South, if the parasite emerges from the gastric glands during midsummer, when temperatures are scorching hot, the eggs won't survive long enough to further develop within the fecal pats. In the North, the middle of winter will jeopardize survival due to extreme cold. In areas in between, Craig says, the worms may suffer an "identity crisis" and go into arrested development at different times.

The real danger is that numbers of larvae can accumulate during a period of time within the gastric glands.

"When the conditions in that geographic locality are better for them, then as a group they start their development, and instead of destroying a few hundred gastric glands, they start destroying thousands of gastric glands," Craig explains. "Animals quit eating; they develop diarrhea; they lose protein."

Other game strategies he says producers should be aware of include:

(Continued on page 32)

Clinical disease

Thomas Craig, a professor of pathobiology at Texas A&M University, College Station, says there are three stages of disease caused by *Ostertagia ostertagi,* commonly called the brown stomach worm.

Type I ostertagiosis occurs in young animals during the early phases of exposure to larvae on pasture. Larvae are on the fast track, entering the gastric glands a few days postinfection and emerging two to three weeks later. Most of the animals in a population will have some disease, from anorexia to diarrhea and weight loss, he says. Watery diarrhea is observed with Type I, and if the forage is rapidly growing, a greenish diarrhea is seen.

Pretype II infection occurs later in the grazing season as larvae acquired on pasture are programmed to enter hypobiosis. These larvae enter the gastric glands and cease development at the early fourth stage. They do not feed or develop, and no disease is seen.

Type II ostertagiosis occurs when the previously arrested (hypobiotic) larvae resume development, usually in the spring in the North and autumn in the South. The larvae emerge from the gastric glands more or less en mass; those acquired over several months may emerge in weeks. The number of animals showing signs may be fewer, but the disease is usually more serious from the numbers of gastric glands being destroyed. Type II signs vary with the forage — a brownish diarrhea if hay or silage is consumed or green if lush grass is consumed.

Submandibular edema (bottle jaw), anemia and dehydration with a dull hair coat are also often seen.

Don't Get Played *(from page 31)*

- ►Heat and desiccation are damaging to larvae in pastures, but survival under snow is common.
- ►Larvae can survive in pastures if entrapped in dung pats for up to a year during periods of drought.
- ►Emergence from the arrested state often coincides with reproduction, and the periparturient relaxation of resistance by the host allows the survival of worms for a sufficient period to ensure the next generation of worms is present in the pastures for acquisition by the offspring.

Defense tactics

William Jolley, a parasitologist with the Wyoming State University Department of Veterinary Sciences, says while complete elimination of roundworm parasites in cattle isn't likely, there are valuable strategies to minimize their effects.

In his region, he says calves experience the most detrimental effects of worms in late summer or early fall due to adult, egg-laying worms in the lumen of the abomasum. Yearling cattle to 2-year-olds experience the most problems with worms during midwinter months to early spring. Winter disease in the older group is a result of the L4 larvae's inhibiting the gastric glands from fully functioning, preventing proper protein absorption.

"Late winter/early spring 'wrecks' are due to high numbers of inhibited larvae simultaneously breaking out of hypobiosis, becoming adult worms in lumen of the abomasum," Jolley says, which usually occurs within two weeks of a warm weather episode in late February, March or April in the North.

Jolley recommends producers in states with climates similar to that of Wyoming to:

1) use an effective enteric dewormer to target adult worms in calves coming off range at weaning;
2) apply effective systemic anthelmintic to target inhibited larvae to animals during late fall/early winter, after grazing is minimal; and
3) if possible, apply enteric dewormer to adult animals prior to spring/summer turnout to minimize and delay buildup of worms in pasture during grazing season.

In southern states, Craig says, the basis of *O. ostertagi* control involves treating to remove hypobiotic larvae and/or the use of anthelmintics (dewormers) that have residual effects so incoming larvae are destroyed before they can establish themselves in the animal. Summer treatment in the South may accomplish this.

"Broad-spectrum, safe anthelmintics are the primary means of treatment and control. Virtually all of the available anthelmintics are effective against adult and metabolically active larvae," he says. "Some benzimidazoles and all macrolides are effective against arrested larvae. Macrolides have a residual effect against incoming larvae that will kill them before they can establish. This residual period (several weeks) varies with the chemical and may be a powerful selection mechanism for resistance."

Craig says states in the middle regions of the United States may benefit most from deworming in the winter and summer; but, regardless of location, he recommends producers discuss a game plan with their veterinarians for the best strategy.

Different anthelmintics have features that may give them a competitive advantage, Craig points out, from cost, ease of administration, spectrum of activity (both in species or in life cycle stages controlled), or the persistence of activity. Classes of anthelmintics currently on the market in North America that are effective against *O. ostertagi* are benzimidazoles (albendazole, fenbendazole and oxfendazole), imidazothiazoles (levamisole, which is only effective against adult worms), and macrolides (doramectin, epronomectin, ivermectin and moxidectin).