Epidemiology and Control of Gastrointestinal Parasitism in Grazing Cattle in Missouri

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Introduction

The state of Missouri ranks second in the U.S., both in the number of cattle on farms, and in the number of calves sold each year. Gastrointestinal parasitism is almost universal in grazing cattle in the state, but until recently, it was unclear whether the loss it causes is economically significant or what form programs for its control should take. Those producers who do practice some form of control frequently rely on anthelmintic therapy alone, and deworming is often done only when it is convenient for the producer, as at weaning. Futhermore, the choice of compounds is usually based on cost rather than the applicability of the compound to the parasites to be controlled.

For these reasons, an on-going project was initiated at the University of Missouri in 1980 to define the epidemiology of gastrointestinal parasitism in grazing cattle in Missouri, and to formulate effective programs for its control. The purpose of this presentation is to summarize the findings of this project to date, and to discuss current recommendations for parasite control in grazing cattle in Missouri, which are based on those findings.

But first a brief review of the parasites.

The Parasites

Six nematode parasites, five trichostrongylids and one strongylid, commonly occur in grazing cattle in Missouri. In the abomasum are found *Haemonchus placei,* the large stomach worm or "barber pole" worm; *Ostertagia ostertagi,* the medium stomach worm; and *Trichostrongylus axei,* the small stomach worm. Parasites in the small intestine include at least three species of *Cooperia,* the "Cooper's" worms, and *Nematodirus helvetianus,* the "watch-spring" worm. The only significant nematode found in the large intestine is *Oesophagostomum radiatum,* the nodular worm.

Haemonchus is a large worm, up to 34 mm in length, and a voracious blood-feeder. In sheep, *H. contortus* burdens may become quite large and cause clinical signs such as submandibular edema ("bottle jaw"), anemia, constipation and death. Fortunately *H. placei* burdens in Missouri cattle rarely reach these proportions and clinical haemonchiosis is seldom seen.

Such is not the case with *Ostertagia.* As its common name implies, *O. ostertagi* is smaller than *H. placei,* 7-12 mm in length. When immature adult *O. ostertagi* emerge from their nodules in the abomasal wall, severe functional and structural changes result which disrupt protein digestion and cause the mucosa to become permeable to macromolecules, particularly serum proteins. The result is severe hypoproteinemia, coupled with severe hypoalbuminemia, and the affected animal presents with diarrhea, severe edema and anemia. Weight loss may be great, up to 20 percent in the first seven to 10 days after onset, and mortality is not uncommon.

A particularly significant aspect of ostertagiosis is the parasite's ability to arrest its development during the early-fourth larval stage while in the abomasal wall. Since these arrested larvae cause almost no pathology, ostertagiosis is said to occur as two distinct clinical entities: Type I, which arises from the acquisition of large numbers of normally-developing parasites, and Type II, which arises from the resumption of development of large numbers of previously-arrested parasites.

The traditional view of this phenomenon, which is based on observations made in Great Britain and western Europe, holds that arrest is an over-wintering mechanism of the parasite. Because of this, the Type I disease was thought to occur only in the fall, while the Type II disease was thought to occur only in the spring. It will be seen, however, that in Missouri the parasite arrests during the summer, apparently to escape high temperatures and/or dessication, and that Type II occurs in the late summer or fall.

The third common abomasal worm is *Trichostrongylus.* It is a small parasite, 4-7 mm in length, and it feeds on tissue fluids. Because of their small size, large numbers of *T. axei* are necessary to produce clinical pathology. When this happens, the glandular epithelium of the abomasum, and occasionally the duodenal epithelium, take on a "frosted" appearance, much like "freezer-burned" tissue. Fortunately, like *H. placei, T. axei* populations rarely reach these proportions in Missouri cattle.

Cooperia are very common worms in Missouri, and at least three species are found: *C. onchophora, C. mcmasteri* and *C. punctata.* As a group, they range in size from 5 mm to 12 mm in length. Although they imbibe blood, they are apparently well tolerated by cattle and clinical cooperiosis has not been reported in the state. Nevertheless, because of their abundance, they cannot be overlooked as a cause of economic loss due to subclinical parasitism.

Nematodirus is another large parasite, reaching 28 mm in length. Both the larvae and the adults suck blood and large populations can cause anemia and diarrhea in young calves. *Nematodirus helvetianus* is unique among bovine parasites in that its larvae are very resistant to environmental stress, and peak populations of *Nematodirus* often arise in the early spring or late fall in Missouri.

The only important nematode parasite residing in the large intestine is *Oesophagostomum*, a strongylid. The fourth-stage larvae of the parasite develop in nodules in the wall of the cecum and colon and after they emerge, the nodules form small abscesses that persist for extended periods. Unlike most gastrointestinal parasites, *O. radiatum* burdens tend to be largest in mature animals, and since older animals are better able to tolerate gastrointestinal parasitism, clinical oesophagostomiosis is not often seen.

While it is only natural to emphasize the clinical aspects of gastrointestinal parasitism, it must be kept in mind that chronic subclinical parasitism is usually much more economically significant than readily identifiable morbidity or mortality. This is a particularly difficult concept for many producers to grasp since they rarely have a "worm-free" standard against which they can compare their own herds.

The Missouri Project

During the summer grazing season of 1980, a preliminary survey was conducted in central Missouri to determine the nature of the parasite burdens in grazing cattle. Three farms, representing high, medium and low levels of management, were selected and the beef herds on them were followed throughout the grazing season with random bi-weekly fecal parasite egg counts and bi-weekly counts of infective parasite larvae on pasture herbage.

Clear epidemiological patterns failed to emerge that year due to a prolonged drought, but one finding was evident: *Ostertagia ostertagi* was by far the most predominant species of parasite present. This was particularly significant in view of *Ostertagia's* ability to arrest its development, and the fact that over 90 percent of Missouri's annual calf crop is born in the spring each year.

Because of this finding, it was determined that *O. ostertagi* should receive the major emphasis in the project and a more or less standard protocol was adopted to help define the bionomics of that parasite. In each of the studies that followed, a group of "resident" animals was selected that consisted of 20-40 beef cows with their respective spring or fall calves. The resident animals were then equivalently allotted to replicated 8.1 ha (20 A) fescue/orchard grass pastures that previous observation had shown to be contaminated with infective parasite larvae. Stocking rates varied from study to study but were always in the range of 0.4-0.8 ha (1-2 A) per cow/calf pair.

In addition to the resident animals, "tracer" or "sentinel" calves were also employed. These were young weanling calves that had been rendered parasite-free through repeated anthelmintic treatment. Pairs of these animals were co-grazed with the resident animals and then sacrificed for worm counting. This allowed for monitoring of the parasite population without altering the integrity of the resident group.

As part of the worm counting procedure, the tracer/sentinel calves were held in box stalls for 21-28 days after their removal from pasture. This holding period precluded any further parasitic infection and allowed the parasite burdens acquired by the animals during grazing to mature. Then at necropsy, the worm burdens were counted and the *ostertagia* present were subdivided into three classes: early fourth-stage larvae, developing forms (late-fourth stage and immature fifth-stage), and mature adults.

Using these data, a model was developed which stated that if after the holding period, a statistically ($P<0.05$) significant proportion of the *Ostertagia* population had remained in the early-fourth larval stage, and if the population distribution over the three classes assumed a bi-modal configuration, then it would be concluded that the parasite had arrested its development.

In addition to the worm counts, a number of other observations were made during the studies. These included: continuous monitoring of climatological conditions, bi-weekly counts of infective parasite larvae on pasture herbage, monthly fecal parasite egg counts in the resident animals and weight gains by the resident calves.

The first major study in the project was conducted during the winter grazing season of 1980-81 to determine if *O. ostertagi* in Missouri would arrest at the traditional time. Consecutive pairs of tracer calves were co-grazed with the resident animals for 28-day intervals and then held and sacrificed as described.

The parasite burdens acquired by the tracers were small, and overall burdens declined steadily during the study, but *Ostertagia* was acquired in all but one of the grazing periods (mid-winter). From September through early March, only small, non-significant proportions of the population remained in the early-fourth stage after the holding period. In contrast, a significant ($P<0.05$) proportion remained in the early-fourth stage following the last period from late March through early April.

Based on these findings, an in-depth study was conducted during the summer grazing season of 1981 to confirm that arrest was occurring, and to attempt to estimate the impact of parasitism on the performance of grazing cattle. The resident animals were equivalently allotted to either fescue pasture or orchard grass pasture on the basis of cow age, calf weight and calf sire-type. Sentinel animals were added to the pastures at the start of the study, and at 28-day intervals, a pair of them were removed for worm counts. Parasite larvae counts were made on the pasture herbage, fecal parasite egg counts were made in the resident animals and the resident calves were weighed.

The herbage larvae counts revealed that extremely large numbers of infective parasite larvae, the majority of which were *O. ostertagi*, were present on the pasture at the start of grazing in late April. This was particularly significant since these pastures had been idle since the previous December, indicating that *O. ostertagi* readily over-winters on pasture in Missouri. The grazing animals acquired their initial parasitic infections from these over-wintered larvae, and their parasite burdens increased steadily until mid-summer. During this same period, the number of larvae on the pastures declined steadily until at mid-summer, the pastures were essentially free of infection. At that point, the parasite populations in the grazing animals began to decline until at weaning, they were relatively small. Then in early fall, the level of pasture contamination began to increase once again.

Peak numbers of parasites, again principally *Ostertagia*, were found in the sentinel animals in June and July that year and *Ostertagia* arrested its development from the start of the grazing season (late April) until early August. At that time, one of the sentinels died of Type II ostertagiosis.

Statistical analysis of fecal parasite egg counts and weight gains in the resident calves revealed that egg counts increased significantly ($P<0.05$) during the grazing season while weight gains decreased significantly ($P<0.05$), and that the greatest decrease in gains coincided with the resumption of development of arrested *O. ostertagia*. These two factors were strongly correlated (r significant $P<0.001$) and a linear regression of calf gains on fecal parasite egg counts revealed that the calves gained on average 0.2 kg less per day than they theoretically could have had they not been parasitized. This amounted to a theoretical loss at weaning of nearly 30 kg per calf.

The results of the 1981 summer study provided a clear picture of the pattern of parasite transmission during grazing and an estimate of the economic impact of that parasitism. Nevertheless, doubts remained that those findings may have been abnormal for Missouri, resulting from the typical weather pattern of the previous year. Therefore it was decided to repeat the previous winter study (winter 1980-81) using the same pastures that were grazed during the summer study.

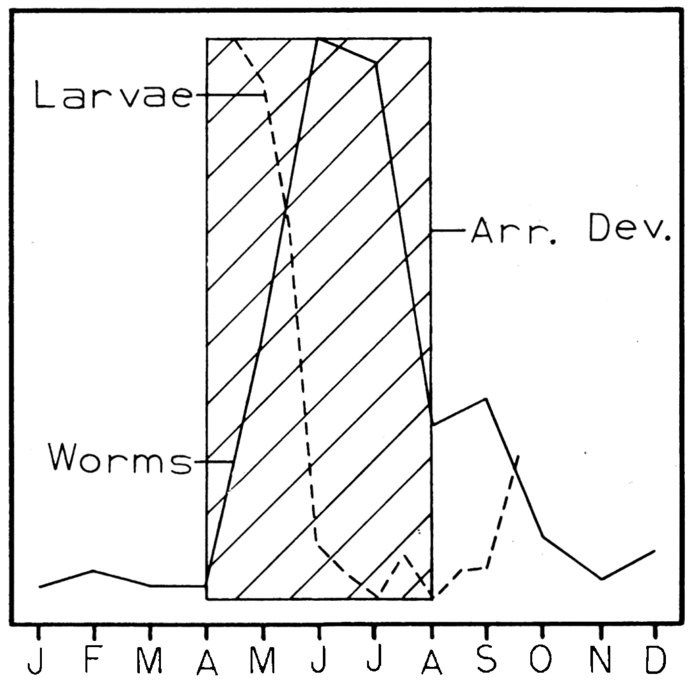
The numbers of parasites acquired by the tracer calves were somewhat higher during the second winter study, but the pattern of transmission was nearly the same. Overall parasite burdens declined to a low in mid-winter and *Ostertagia* was once again the predominant species. Similarly, significant developmental arrest of that parasite was not seen.

It seemed only logical at that point in the project to conduct another summer study to confirm the findings of the 1981 summer study. Plans had been made to evaluate the efficacy of a strategic deworming program during the summer of 1982 (the results of which are presented below) and a typical tracer calf protocol, similar to the 1980-81 and 1981-82 winter studies, was "piggy-backed" onto it.

Initiation of the study was delayed by another "atypical" weather pattern, an abnormally wet spring, but the results were very similar to those from 1981. Peak numbers of parasites were acquired by the tracers in August that year and *Ostertagia* was once again the predominant species. Although the onset of developmental arrest of *O. ostertagi* was missed due to the delay in starting the study, significant ($P<0.05$) levels of arrest were seen in May, June and July. These findings parallel quite closely those of the previous summer.

Figure 1 summarizes the findings of the project to date.

Figure 1. Pattern of transmission of gastrointestinal parasitism in grazing beef cattle in Missouri, 1980-1982. The solid line represents the relative size of the parasite burdens in grazing tracer or sentinel calves. The broken line represents the relative level of pasture contamination by infective parasite larvae. The shaded area is the interval during which **Ostertagia ostertagi** is arrested in the early-fourth larval stage.



A Strategic Deworming Program

Based on the project findings through the spring of 1982, at least three things were clear regarding the formulation of effective control programs for grazing cattle in Missouri. First, the program would need to be directed primarily against *O. ostertagi*. Over the term of the studies, that one species comprised nearly 90 percent of all the parasite seen, both as infective larvae on pasture herbage and in parasite burdens in the tracer/sentinel animals. And the greatest decrease in the performance of the grazing calves in the 1981 summer study coincided with the resumption of development of previously arrested *Ostertagia* larvae.

Second, any anthelmintic compound employed would need to have acceptable efficacy against arrested *O. ostertagi.* In both of the summer studies (1981 and 1982), the parasite arrested during the first one-half of the grazing season and it was during these periods that the majority of the parasite burdens were acquired.

And third, the application of specific control measures would need to be strategically timed to critical periods in the epidemiological cycle. It can be seen from Figure 1 that at least one of these critical periods is mid-summer. If control measures, particularly deworming, were to be applied earlier than this, the number of over-wintered infective larvae remaining on the pasture would still be sufficient to re-infect the animals. If applied later, arrested *O. ostertagi* would have already resumed development and most of the pathological insult to the animals would have already occurred.

With these three criteria in mind, a study was conducted during the summer of 1982 to evaluate the efficacy of strategic deworming with fenbendazole (PANACUR, SAFE-GUARD: American Hoechst). Forty cow/calf pairs were equivalently allotted to four pastures on the basis of cow age, calf sex and calf weight. Two of the pastures had a base forage of fescue and orchard grass. One group on each pasture type was then chosen to receive the treatment program, while the other group served as a non-medicated control. The cows in the medicated groups received fenbendazole 10 percent oral suspension, at the rate of 10 mg/kg body weight, just after calving (March 1). Then at midsummer (July 19), both cows and calves in the medicated groups received the compound at 5 mg/kg body weight.

As expected, both the medicated cows and the medicated calves had significantly ($P<0.05$) lower fecal parasite egg counts over the term of the study than did their non-medicated counterparts. The latter difference was particularly noticeable in August and September when the non-medicated calves had extremely high egg counts. Body weights of the medicated calves were also significantly ($P<0.05$) greater over the term of the study than those of the non-medicated calves. Interestingly, this difference was statistically significant as early as July 1, which was before the calves themselves were dewormed, indicating that spring treatment of the cows had played a role in the improved calf weights.

When the results were summarized, the calves in the medicated group were found to have gained on average 0.11 kg more per day (significant, $P<0.05$) than the non-medicated calves. When adjusted 205-day weaning weights were computed for each group, this difference translated to a 22.5 kg advantage (significant, $P<0.05$) for the medicated group. In short, application of strategic deworming alone, without other control measures such as pasture rotation, resulted in the recovery of nearly 75 percent of the theoretical 30 kg loss that was estimated in the 1981 summer study.

Recommendations for Control

As mentioned at the outset of this presentation, the Missouri Project is ongoing, and there is still much to learn about gastrointestinal parasitism in grazing cattle in Missouri. With the information currently in hand, however, it has been possible to formulate preliminary control recommendations for Missouri producers and practitioners. These are presented below according to type of production system.

Spring-calving cow/calf herds. Deworm cows and any calves more than 45 days of age before moving to spring/summer pasture. At mid-summer, deworm both cows and calves with a compound effective against arrested early-fourth stage *Ostertagia ostertagi* and move the animals to fresh late summer/fall pasture. Deworm both cows and calves again at weaning and move the animals to separate winter pastures or dry lots.

Maintain proper stocking rates on all pastures and if possible, graze a pasture for only one period in a calendar year. Harrow pastures during hot, dry periods only and never follow weaned calves with suckling calves.

Fall-calving cow/calf herds. Deworm cows after calving and move the animals to fresh autumn pasture. Deworm both cows and calves at weaning and move the animals to separate pastures or dry lots for the remainder of the winter. Deworm both cows and previously-weaned calves again before moving to the spring/summer pasture.

Maintain proper stocking rates and provide adequate supplemental feed during winter. Never harrow pastures during cold weather.

Weaned calves and stocker cattle. Deworm all cattle at entrance time. If severe parasitism is suspected, or when clinical parasitism is already present, repeat deworming 21 days after the initial treatment. Deworm all cattle at midsummer with a compound effective against arrested *Ostertagia* larvae and move the animals to clean fall pasture. Maintain proper stocking rates and provide adequate supplemental feed when necessary.

References

1. Armour J: Parasitic gastroenteritis in cattle. Vet Rec 95:391-395, 1974. 2. Armour J: The epidemiology of helminth disease in farm animals. Vet Parasit 6:7-46, 1980. 3. Armour J, Bruce RG: Inhibited development in Ostertagia ostertagi infections - a diapause phenomenon in a nematode. Parasitol 69:161-174, 1974. 4. Armour J, Ogbourne CP: Bovine ostertagiosis: A review and annotated bibliography. Misc Pub No 7 of the Commonwealth Agriculture Bur, Farnam Royal, England, 1982. 5. Brauer MA: The epidemiology and bionomics of Ostertagia ostertagi in beef cattle in Missouri. PhD Dissertation, Univ Missouri-Columbia, Columbia, Mo, 1983. 6. Brauer MA, Corwin RM: The control of internal parasites of cattle. Hoechst Mon, Am Hoechst Corp, Somerville, NJ, 1983. 7. Bumgarner SC: Epidemiology of beef cattle parasitism in Missouri: A strategic deworming program for spring-calving cow/calf herds. MS Thesis, Univ Missouri-Columbia, Columbia, Mo, 1983. 8

Corwin RM, Talent NK, McDowell AE: A guide to anthelmintic compounds in livestock practice. Bov Pract 1:20-32, 1980. 9. Borgsteede FHM (ed): Facts and reflections III. Workshop on arrested development of nematodes of sheep and cattle. Central Vet Instit, Lelystad, The Netherlands, 1978. 10. Michel JF: The epidemiology and control of some nematode infections of grazing animals. Adv Parasit 7:211-282, 1969. 11. Michel JF: The epidemiology and control of some nematode infections in grazing animals. Adv Parasit 14:355-397, 1976. 12. Pritchard RK, Donald AD, Dash KM et al: Factors involved in the relative anthelmintic tolerance of arrested 4th stage larvae of Ostertagia ostertagi. Vet Rec 102:282, 1978. 13. Snider TG, Williams JC, Sheehan, DS et al: Plasma pepsinogen, inhibited larval development, and abomasal lesions in experimental infections of calves with Ostertagia ostertagi. Vet Parasit 8:173-183, 1981. 14. Williams JC: Bovine ostertagiosis. Anim Nut Health 36:6-9, 1981. 15. Williams JC: Ecology and control of gastrointestinal nematodes of beef cattle. Vet Clinics North Amer: Large Anim Pract 5:183-205, 1983.

District News

District III — Dr. David McClary

Membership from District III now totals approximately 357 with 66 new members added in the 1983 supplement, and 34 added in the 1984 supplement. In an attempt to increase membership all veterinarians in District III who are not AABP members and are listed as large or mixed animal veterinarians in the AVMA directory will be sent a letter and a membership application inviting them to join AABP. All members promote our organization and encourage non member bovine practitioners to join AABP.

All 7 veterinary schools in District III now have AABP student chapters. The newest chapter is at North Carolina State where approximately 40 students enrolled. The total student enrollment for District III is approximately 165 students.

Continuing education courses sponsored by AABP in District III during the last 6 months include:

1. Performance Evaluation of cattle using DHIA Records and other monitors. March 29 and 30 Gainsville, FL. Presented by Drs. Jan Shearer, Art Donovan, Ken Braun and Mr. Dan Webb of DHIA.
2. TI59 Dairy Nutrition Seminar. May 18, 19, 20. Raleigh, NC. Presented by Drs. Ben Harrington, Tim Lesch, and Arden Nelson.

Future planned meetings include a one day in-depth seminar at the Eastern States Veterinary Conference January 14, 1985. Eastern States planners want to make this an annual seminar during the first day of their conference.

(District III included Alabama, Florida, Georgia, Mississippi, North Carolina, South Carolina, Tennessee, Puerto Rico, Cuba, South America and West Indies.)

Practice Tip

Q. & A.: Drying up the mammary gland

Question: What product, dosage, and duration of treatment may be used *safely*, with little systemic side effect, to cause a mammary gland to dry up during heavy lactation of the other glands i.e. in the case of self inflicted treatment amputation?

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Answer: Use of corrosive agents in the udder may help reduce the amount of lactating tissue or destroy all of the tissue. Strong iodine, Lugols solution and silver nitrate have all been used with some success.

I suggest 40 to 60 cc's of strong tincture of iodine. Expect local tissue reaction and several days to a month's time to destroy the tissue. There should be limited systemic effect other than the body's response to the foreign substance in the udder.

Jenks S. Britt, D.V.M.
Russellville, KY