Beef Session II

Cow-Calf/Feedlot Combined

Louie Perino, *Presiding*

Cow/Calf Parasitology: How Critical is the Time of Deworming? and, Ostertagiosis Update

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Based upon our studies in Missouri from 1979 to the present, we established that gastrointestinal parasitism is a year round problem in beef cattle herds. These parasites include nematodes, cestodes and protozoans (coccidia, *Giardia*). The assemblage of nematodes is sizeable, both in terms of numbers and species. There may be thousands of adults and larvae present, even in clinically inapparent infections. In the abomasum, *Haemonchus, Ostertagia,* and *Trichostrongylus* are found; in the small intestine, *Cooperia, Bunostomum, Toxocara* and *Nematodirus;* in the cecum and colon, *Trichuris* and *Oesophagostomum;* and in the respiratory tract, *Dictyocaulus.* Each of these does have its own seasonality, though there is certainly an overlap in time and place for these nematode species. In attempting to formulate a strategic parasite control then, we looked for the one parasite species which caused most of the parasite problems causing clinical disease and economic loss. That one species is *Ostertagia ostertagi.* So based upon its seasonality and the peculiarities of its life cycle, a strategic deworming program including pasture management was developed.

Ostertagia ostertagi, the medium brown worm, resides in the abomasum. As an adult, it grazes on the luminal epithelium, but as a larva (L_4) it is found in the gastric gland with little activity occurring. Thousands to hundreds of thousands of both stages may be present, although in Missouri, counts of 5-10,000 are more typical. It is as an emerging larva that most pathology occurs. This stage (EL_5) causes a disruption of glandular epithelium causing a breakage of intercellular bridges, sloughing of columnar epithelium, loss of HCl production, decrease in pepsin formation, leakage of albumin into the abomasum resulting in a hypoalbuminemia, absorption of pepsinogen with a subsequent increase in plasma pepsinogen, replacement of glandular epithelium with nonglandular cuboidal cells, the gross appearance of a pebbled ("Morocco leather") luminal surface rather than a glossy smooth one, edema of the abomasal folds as well as of most tissues, a normochromic normocytic anemia, and, with an increasing pH, the likelihood of an influx of bacteria from the small intestine that may cause a septicemia. (Remember that this animal at necropsy will have the heavy, thick abomasum with the pebbled appearance; use litmus paper to test the pH and sieve the contents and abomasal scrapings for species identification). Affected animals are going to be dyspeptic, off-feed, appear rough, and show a liquid scouring. Weight loss is common; it may require weeks for restoration to normalcy or there may be no recovery with the victim being a poor-doer forevermore.

The problem of ostertagiosis is a complex one, with seasonal exacerbation due to differing intensity of larva emergence. There are two types of ostertagiosis, viz., Types I and II, which have different lengths of cycles, are seasonal, may overlap in time and place, and appear at different times in our northern and southern temperate climates (even within the same state). Type I represents the condition resulting from the strain with a cycle of 21-28 days. The L_4 spend only a few days in the gastric glands, emerge, and rapidly mature to the egg-laying adult stage. We usually see one or two generations of this type *Ostertagia* in the late spring-early summer and again in the late summer-early fall. This results in contamination of pastures with eggs and larvae in the early summer and in the fall. Potentially infective larvae die off during the hot dry months but survive fairly well overwinter. Larvae may escape the hazards of adverse weather conditions by being carried several inches into the soil, perhaps serving as a successful reservoir for future infections. Type 1 results from emergence of the El_5 and feeding of the adults; pathologically and clinically it may be indistinguishable from Type II, but remember that this is intensity-related and may show a range of affect from mild to severe. Type I is the condition then resulting from a population which is adult dominant.

Type II represents the condition caused by a strain of

Ostertagia with a prolonged cycle, this due to the EL_4 which persists in the gastric glands for 14-18 weeks. This arrest-prone strain overwinters in cattle in northern temperate areas and oversummers in southern zones. For example in Missouri we observe the period of arrest or hypobiosis or pre Type II from late March through July whereas to the north it is seen from late October through February. Interestingly, occurrence of pre Type II may differ within a given state. From reports I have read or heard, there may be over-summering in Illinois south of I-70 and overwintering north, oversummering in the Virginia coastal plains and overwintering in the Shenandoah Valley, and oversummering in Kentucky west of Kentucky Lake but overwintering east of the twin lakes. (Certainly many more epidemiologic studies in all of our beef areas need to be made). This period is a pathologically-clinically quiescent one, but with the sudden massive emergence of EL_5, a severe clinical picture usually develops, especially in a few animals within each herd (much as we see lousiness especially in certain individuals). Thus severe ostertagiosis occurs in August-September in the south and February-March in the north. Certainly these times may vary a bit with climatic-geographic gradations. Pre-Type II then is a larva-dominant population.

In a spring calving cow herd, cows usually carry a population of adult *Ostertagia* overwinter as well as hypobiotic larvae in the north. If Type II occurs at calving, there may be increased loss in condition: difficulty in calving, reduced lactation and slowness in regaining weight. At calving, there is usually an increased egg output ("periparturient") because of immune depression. If cows and calves are later moved to summer pasture, this increased contamination at calving may be of little consequence. As calves begin to graze, continuing contamination of grass and soil by their infected dams leads to infection of the calves especially with Type I. For example, calves in northern areas would only be exposed to Type I *Ostertagia* during early summer grazing and those in southern climes possibly to the Type II strain as well as to Type I. (It should be emphasized here that the terms Types I and II really refer to conditions but their use here is being stretched to include the strains producing these conditions). Thus spring calves in either area are being infected and affected predominately by Type I. Hence, an adulticide anthelmintic compound in mid-summer would be the drug type and dosage level of choice, e.g., fenbendazole, levamisole, ivermectin (although at the recommended level of dosage, ivermectin is also larvicidal). If yearling calves are being backgrounded on the same pastures, those in southern regions would have been exposed to arrest-prone larvae from March to early summer and this age group would be at greatest risk for the late summer occurrence of Type II ostertagiosis. Administration of an anthelmintic drug with larvicidal properties needs to be performed by midsummer in these areas to avert this potential disaster, e.g., ivermectin at 200 mcg/kg body weight, fenbendazole at 10 mg/kg, whereas in the north in midsummer, an adulticide anthelmintic should be recommended. Hopefully, pasture rotation would occur soon after each deworming.

In the fall, when deworming should again be performed, we must again look at the geographical region. In the north, larvicidal anthelmintics must be administered to calves (and justifiably to bred cows) to diminish the arrest-prone larva population, whereas, in the south, adulticide anthelmintics are appropriate.

Fall calves should certainly be treated by midsummer of the next year and again in the following fall, the type and dosage level of course being dependent on the regional occurrence of pre Type II. Replacement heifers should be dewormed by age and by regional requirements.

The production benefits of a strategic deworming program in our Missouri studies at midsummer have been 15 to 25 lbs advantage for calves from dewormed cows vs those from cows not dewormed, and 45 to 50 lbs advantage for stocker calves dewormed in the spring vs those not dewormed. In the fall, spring calves dewormed had a 49 lb advantage over those not dewormed, and stocker calves dewormed midsummer had a 67 lb advantage in September and 80 lb advantage by November. Those weight gain differences were consistently shown over a 3-year period of observation. Weight gain advantages in the Gulf States are even greater.

As regards the tapeworm *Moniezia,* there is certainly much more to be learned. Field observations indicate that problems do arise in calves, but this has yet to be proved on a scientific basis. Fenbendazole does remove some of these tapeworms and perhaps could negate their effect. Coccidia are present as 9 species, all of which we observe in Missouri herds, and some of which are seasonally pathogenic, e.g., during the droughts of late summer, at fall weaning, and during winter stress. Coccidiostats should be available for periods of several weeks prior to these times of stress. But we have also found that if deworming is strategically performed, coccidiosis is minimized.

In conclusion, there have been several significant advances in the understanding of ostertagiosis in the past several years:

Perhaps the most important has been a better understanding of the epidemiology of ostertagiosis in each of our geographical-climatic regions. This has permitted development of control (pasture rotation) and treatment schedules which effectively reduce the impact of this parasitism.

Anthelmintic development has been very impressive, too, both in terms of chemical class and vehicle of administration: Ivermectin and levamisole as injectables, fenbendazole as a paste and suspension and morantel in a sustained-release cartridge. These have allowed ease of administration and safety for you and the animal being treated. Look for additional advances in the near future.

Diagnostically, we have relied on the fecal egg count, which I must state is not reliable because of the common

morphology of all trichostrongylids, the uneven fecundity of each species, that low levels may not reflect a large population of adult worms and do not reflect the larval population, and that pathology is difficult to demonstrate, although plasma pepsinogen levels may measure ongoing damage. Unfortunately pp does not diagnose the early phase of infection. Intradermal injection with secretory-excretory antigens (S/E) of L_3 do show a positive correlation with early infection. How this test may be used practically is still open for debate. I feel that at this time such a diagnostic tool is still an experimental one. Studies on the immunology of ostertagiosis has shown that the S/E of L_3 induce an oesinophilic chemotactic response; the Ag has a MW of 14,000 and its Ab counterpart is a receptor on the eosinophil surface, so that eosinophils mobilize and move toward the gastric glands. Unfortunately, L_4 are relatively inactive and inert with little effect of the eosinophils upon them. Thus there is an initial inflammatory response and then quiescence.

So back to square one. Practically, a strategic control program must be employed. Development of better anthelmintics, modes of administration to reduce labor intensity, use of "safe" pastures, and better diagnostic tools should be of benefit. Vaccines are not likely to be developed in the foreseeable future.

Questions & Answers:

Question: Gill, you were at a coccidiosis seminar. Was there anything stated about the aftermath of subclinical coccidiosis?

Dr. Corwin: The question relates to the use of an anthelmintic before winter stress to suppress that particular stress. And we're advocates of the use of anthelmintics at this time of the year so that the worm burden is suppressed. Suppressed because of the effects during the winter and not so much because of the subsequent contamination of pasture. I guarantee that there is contamination of pasture, but as I said earlier I'm really not sure how effective or active that contamination is going to be. It certainly does have an effect on the growth performance of cattle over winter. It certainly has an effect upon the coccidia which are present. If we use an effective anthelmintic at this time of year, I think it will also suppress the effect of coccidiosis. The second part of your question was about resistance by the long term use of a compound like morentel. I believe at this time that a compound such as morentel is going to have a large part of its effect as a larvacide as the larvae gain entry into the host we are going to be killing them off. There won't be an opportunity for the development of resistance. I think morantel can be used effectively in that way. But to continue on with that, I would suggest that none of you use compounds that are at less than advised recommended level. The use of benzeminazols at 2.5 to 5.0, but say 2.5 miligrams per kilogram body weight, I think if we're going to be using compounds at a lower than advised dose level that we certainly are going to be asking for problems. There certainly is an economic advantage for that now. But I think that in a year or two or three from now, if you use compounds like oxfendazol and oxybendazol which salesmen have suggested their use at those levels because of the cost benefit, that what we are dealing with is removal of some parasites with the persistence of other parasites that may develop a resistance to that class of compounds.