Control of Helminth Parasites in Cow Calf Operations in the Southern United States

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To control parasitic disease in cattle one has to first determine which parasite(s) are present and if they are or could cause harm. The idea of "I don"t care what they have, I just want to kill „um all" is not likely to be a successful approach over time. The class of cattle, geography, time of year and management may help determine if the numbers of parasites are sufficient to cause clinical or economic disease. Each producer is likely to have a different array of parasites that infect his/her cattle and this will determine if the cost of parasitic infection will reach the economic threshold. Occasionally clinical disease may be seen but the primary loss is economic. Lower milk production, decreased calf weaning weight, delayed return to estrus or repeated breeding are the major economic effects of helminth (worm) parasites.

During the past 30 years with the advent of broad spectrum, safe, effective drugs there has been a shift in the kinds of parasites a cattle producer is likely to encounter. Some parasites such as cattle grub, scab mites, and lung worm are gone or so rare they cause a stir when identified. Others that were and still are of lesser significance are still here and are increasing in prominence. The remainders are important now as they were then and, in some instances, much more important because of animals"" failure to develop immune resistance due to low levels of exposure or increased exposure due to higher stocking density.

Control programs should be predicated on the idea that we prevent disease not that we rid the herd of parasites. The surviving parasite species either have strategies that enable some of them to evade treatment by not being where they will be adversely affected when treatment is done or evolved populations that are resistant to the drugs used.

Parasitic nematodes are the most important profit-limiting group of parasites in the cattle industry at this time. They are found from the true stomach to the large intestine with each parasite species occupying a specific portion of the digestive tract and earning its living in different ways. All of the important parasite species have the same life cycle in the environment but vary in where they go and what they do inside cattle. Eggs are passed by female worms living in the digestive tract and the egg must develop in the dung pat for a few days to weeks before hatching. The larva that emerges from the egg feeds on bacteria and after a week or two, molts to the infective stage larva. This infective stage moves away from the dung pat in a film of moisture and if there has been sufficient rainfall and/or dew ascends in a film of water onto the vegetation where it is grazed by cattle.

Overall, the stomach parasite *Ostertagia ostertagi* is the most important parasite of cattle in North America. Most of the suggestions associated with control programs are directed toward this parasite. However, *Ostertagia* is a temperate climate parasite and cannot persist in tropical climates such as far South Florida or Texas. In the remainder of the southern United States (most of Texas or Florida) this parasite is transmitted to cattle during the cool season, October to May. It is important because it is one of the few parasites that do well in older cattle. Cattle become resistant to *Ostertagia* after being exposed multiple times over several years. If

there are several years not conducive to the transmission of the parasite older cattle react similar to calves, whereas exposed cattle will develop resistance to the parasite by 3 to 4 years of age. Cattle with Braham ancestry may never develop resistance to *Ostertagia*.

The disease caused by *Ostertagia* is associated with the emergence of the larval stages from deep in the crypts of the glands lining the stomach. The emergence is part of the maturation process and occurs 10 to 14 days after they were acquired by grazing unless the worm goes into hypobiosis. The cells lining the stomach glands are either destroyed or put in fast forward and do not produce the hydrochloric acid needed to convert the enzymes in the stomach into a form necessary for digestion. In addition there will be leakage of protein into the lumen of the gastrointestinal tract and a failure to absorb nutrients in the intestine. A result of this is an animal that loses its appetite even though it may appear normal. When there are large numbers of larvae emerging concurrently, as happens following hypobiosis, diarrhea and fluid loss is commonly seen. The disease following hypobiosis is called type II ostertagiosis and treating cattle during the hypobiotic period with an effective drug against these resting larvae is essential to control programs.

Conversely *Haemonchus placei* is a tropical parasite that may only be transmitted during a short time period in the summer in the north but April through October in the south.

Because of a limited time frame during which successful transmission occurs many nematodes undergo hypobiosis or arrested development. Hypobiosis is a cessation of metabolic activity within the host whereby the parasite survives by evading unfavorable conditions in the host or environment. If worms mature and reproduce at an unfavorable time the eggs or larvae encounter conditions too hot, too dry, or too cold for their successful gaining of a host. Hypobiosis occurs with *Haemonchus* over winter and *Ostertagia* over the summer in the south. Hypobiotic larvae are not recognized by the host immune system so even if the host has the ability to expel that species of worm it is unable to do so. However, when the host is stressed, during parturition or lactation, when the worms resume development and they will successfully reproduce in the host.

Resistance by the host to parasites is largely under the control of the immune system and requires exposure to the parasite in order to stimulate a protective response. Host resistance is relative as animals become resistant to disease but not free of parasites. Within populations we see an extreme variability in parasite numbers even in resistant hosts. Approximately 20% of the herd will have 80% of the parasites. Some of the variation may be accounted for by factors such as stress, or lactation. For instance, first calf heifers are still trying to grow, feed a calf, and compete with older and wiser cows for quality nutrients and tend to have a much higher parasite load than the remainder of the herd.

Early infection by gastrointestinal nematodes by single suckled beef calves is usually limited and the provision of high quality feed (milk) assures that the suckling calf will probably not show signs of disease. The exposure to parasites is much less than when calves are run as a group. Likewise, with adequate nutrition they are able to compensate for numbers of parasites that may otherwise cause disease. However, if replacement calves are not sufficiently exposed they may fail to gain sufficient immunity to give them a level of protection during their productive lives. Immunity to some species of parasitic nematodes may be established during the first grazing year as there is sufficient exposure to stimulate some protective response. One common calf nematode *Cooperia* is a small intestinal parasite with one species more likely to be transmitted in the cool season along with *Ostertagia* and others in the warm season with *Haemonchus*.

One species of *Cooperia, C. punctata*, a warm season parasite, may cause excessive inflammation of the small intestine, apparently due to the attempts of the body to rid itself of the parasite. This is the major species implicated with anthelmintic resistance in cattle. The genus can cause diarrhea if it is present in large numbers and can be a problem in dairy or light

weight stocker calves. In time, *Cooperia* stimulates a strong protective response and is unlikely to be seen in older cattle in sufficient numbers to be of any concern.

Haemonchus in young cattle can be a primary pathogen. Most problems with *Haemonchus* have been associated with stocker or dairy calves on high density summer pastures. Suckling calves are infected but the resistance to the parasite by adult cattle is such that they act as a biological vacuum sweeper removing most of the larvae from the pasture so that the calves are not seriously affected. However calves grazing pastures with forages such as Coastal bermudagrass may encounter sufficient worms that they die of exsanguination. *Haemonchus* is a voracious blood sucker and calves may look normal until just prior to death. Some calves will be very sluggish and may develop bottlejaw but do not lose much weight or usually have diarrhea associated with this parasite. The losses caused by this parasite are clinical not just economic.

The large intestinal parasite *Oesophagostomum radiatum* is the only parasite of this group of nematodes that can be transmitted by larvae penetrating the skin as well as by grazing. The larval stages of the parasite cause the formation of nodules in the intestinal wall which are a cause for condemnation of intestines at slaughter. The adult worms are also blood suckers but pale in comparison with *Haemonchus*. A major problem is that the eggs of *Cooperia, Haemonchus, Ostertagia Oesophagostomum*, and a few others all look the same. So diagnosis of the worm species based only on egg morphology may miss the correct diagnosis.

The large roundworm of cattle, *Toxocara vitulorum*, has been seen in Florida and south Texas. This worm is a tropical parasite that is transmitted to calves while suckling. The worm becomes an adult in the calves‟ intestine where it produces a thick shelled egg that is very resistant to the environment. Each female worm will produce hundreds of thousands of eggs each day. The egg is the infective stage. A larva develops in the egg after a few weeks and remains encased in the egg until eaten by a bovine. In the cow the egg hatches and the larval worm migrates into the tissues where it ceases migration until the cow calves and the larva resumes its migration to the mammary glands and the calf becomes infected while suckling. Where calves are infected by a great number of these large worms (nearly a foot in length) the calf suffers from ill thrift. However finding eggs in most calves is an interesting finding that veterinarians congratulate themselves in being able to diagnose the worm species because of its unique egg.

Liver flukes in cattle are either present or absent from a premises. Maps showing their distribution are only an approximation, and do not indicate the microhabitat of the snail intermediate host; which are low laying areas with clay soils that are periodically flooded, or seeps that keep the soil moist. The range of the intermediate host snails appears to be expanding. Seasonal transmission varies with geographic region and available moisture. Only a few cows infected with flukes will directly benefit from treatment. Treating adult cattle for *Fasciola hepatica* will have only minimal effect in their production. However, treating these cattle may lower the infection rate in the snail population sufficiently that calves on the pastures will acquire levels of parasitism below the threshold of disease. Environmental manipulation of snail habitat may be feasible in some circumstances but is unlikely to have long-term effects. The effective drugs against liver flukes in the U.S., Clorsulon and albendazole, are only effective against adult *Fasciola*. By the time the drugs are used the damage caused during fluke migration has already occurred but treating cattle before the snail activity season may lower transmission the following season. Treating cattle in the autumn appears to be the best approach in the south.

Approximately 50 years ago a revolution occurred in the development of anthelmintics. The products had a much broader spectrum of activity than anything previously used and the safety margin was sufficient that over dosage was unlikely to cause toxicity. With the development of these safe, effective products the

approach to the control of parasitic disease shifted from one of treating "wormy" cattle to strategic or tactical approaches which resulted in cattle that ate more, grew faster, produced more milk and were able to exploit pastures, which formerly had been too dangerous to graze by young cattle. Economic parasitism, not clinical disease has become the gauge of the success for anthelmintics. The primary economic effects of anthelmintics (drugs that kill worms) are those of increased milk production in grazing cattle and the increased growth rate in beef calves. Not only do cows produce more milk there is evidence they breed back more quickly after calving. To be certain not all experiments indicated these findings, as there is variability among herds, geographic localities and years. Different anthelmintics have features that may give them a competitive advantage: cost, ease of administration, spectrum of activity (both in species stages of the life cycle stages controlled), or the persistence of activity. Four classes of anthelmintics currently on the market in North America are effective against gastrointestinal nematodes: Benzimidazoles (albendazole, fenbendazole, and oxfendazole), imidazothiazoles (levamisole), tetrahydropyrimidines (morantel), and macrocyclic lactones (doramectin, epronomectin, ivermectin and moxidectin). The drugs are administered orally, topically, or by injection depending on the solubility and metabolism within cattle and the effects depend on the species and developmental stage of parasites of major concern.

Benzimidazoles prevent the polymerization of rapidly dividing cells, such as those lining the intestine of the worm, and the cells are not able to absorb nutrients essentially starving the worms. This family of drugs is extremely safe with possible exception of Albendazole in the first month of pregnancy. Imidazothiazoles and tetrahydropyrimidines act on receptors in nematode muscle cells which causes a spastic paralysis. Adequate doses of these drugs are important because at low doses some worms recover and are able to reestablish. Overdoses of these drugs may cause calves adverse reactions such as staggering, slobbering, and increased urination. Apparently the effects of these drugs on the worm are due not only the ability to cause the paralysis but the worm must be situated so that normal peristalsis removes it from its normal habitat before recovery. Macrocyclic lactones (macrolides) paralyze parasites but not all muscles are affected equally. The pharyngeal muscles are especially vulnerable to these compounds and the worm is unable to feed. The worms may be lost through normal peristalsis. The macrolides have residual effects against newly acquired larvae that are dispatched as they enter the host.

The specific drug, method of administration, and species of worm will determine the duration of residual activity. At first glance a product with residual activity has considerable advantages as cattle can remain in highly infested pastures without danger of disease. However, as the efficacy tails off it can be a powerful selection mechanism for anthelmintic resistance. Doramectin and moxidectin have longer residual activity than ivermectin. Moxidectin has the longest duration with 99% efficacy against *Ostertagia* at 35 days post injection.

Strategic use of anthelmintics to control parasites clear the host of hypobiotic larvae before the worms do damage and lessen pasture contamination by preventing the number of larvae in the environment that reach levels that cause disease later. The seasonality of arrested development varies geographically. *Ostertagia* undergoes summer arrest in the south and this becomes the strategic period. Tactical use of anthelmintics is the application of the drugs when worm levels are increasing in numbers or likely to do so. Such as two weeks following rains after a period of drought or a few weeks after green up of a pasture in the spring. Treatment based on egg counts, serum protein, hematocrit, or pepsinogen levels can also be considered.

From the time of development of the anthelmintics we have on the market there have been a few worms out there that decided that they did not believe the label and were tolerant of the drug‟s effect. If the drug was used on that population of worms, after a while they were the only worms present, as the believers were killed

and the population changed from a few worms tolerant to, to a lot of worms resistant to a drug. Because cattle ranchers do not tend to use anthelmintics as often as small ruminant or horse owners, the advent of detectable numbers of resistant worms took longer to become apparent in cattle than elsewhere. Is anthelmintic resistance a problem in cattle? It can be! Not everywhere or all the time. *Cooperia* and *Nematodirus* (a small intestinal parasite more important in dry and cold climates) were tolerant of the macrocyclic lactones from the get go. They are still resistant to this class of drugs but both genera stimulate an early robust resistance to disease. When light weight stocker or replacement calves are grazed on heavily contaminated pastures the use of a benzimidazole or combined with a macrocyclic lactone is certainly warranted. A major concern is populations of *Haemonchus* that are resistant to both benzimidazoles and macrocyclic lactones just as in small ruminants. Did the resistant worms come from small ruminants? Probably not but, the resistant worms are found in the highest prevalence in the Gulf Coast and have been transported elsewhere. Properties where calves are backgrounded or permanent summer pasture are used by one group of naïve calves after another are the major source of the resistant worms at this time. Resistant *Haemonchus* in cow-calf operations is not common and if it were, the cows cleaning up the pasture is likely to keep the level below that of any significance.

Evidence has been reported that the use of pour-on macrocyclic lactones to control flies especially often at low doses is a powerful selection mechanism for resistant worms. You might get fly control for a while then loose the effectiveness of the dewormers. Because most of the resistance thus far reported has been in calves. The cow may act as a biologic vacuum sweeper and unless under dosed for the other worm species once or twice a year the drugs are likely to be effective in cows but not in calves.

The bottom line is to treat the Southern cow herd with a drug effective against arrested *Ostertagia* in the late spring i.e. May or June. If you want to save money; treat bulls, first and second calf heifers and not the older cows. If suckling calves are at least 200 lbs body weight, treating them at the same time as mommy will usually result in heaver calves at weaning. Lighter calves, if nursing, will not likely be benefited by treatment. In calves an injectable or oral product is essential. If you are setting the dose gun for the calves set the dial at the weight for the heaviest calves not the mean. Deworm calves at or near weaning again using an injectable or oral product and there is considerable evidence that using both concurrently may improve parasite control. Treat for flukes in the early autumn to protect snails. Evaluate the effectiveness of anthelmintics in replacement cattle on the farm every several years. The internet will not tell you which dewormer is best for your ranch, the cattle will.