Coccidiosis in Dairy Cows

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This paper is not designed to delve into the scientific aspects of coccidiosis, but rather to make observations and even speculations about the disease. I might even add that there are some questions asked that experienced veterinarians may be able to shed some light on or start researchers thinking.

Coccidiosis is a disease caused by a single celled parasite of the *Eimeria* genus. It is primarily a problem seen in neonates and related to filth. The similarity of disease between species is similar and I would like to do some comparisons to aid in understanding prevention and control of the disease.

Much that we see in the disease relates to its life cycle. The disease starts by injesting a sporulated oocyst. These invade intestinal mucosa cells and undergo an asexual phase of reproduction call schizogony. After a few days, the next phase of reproduction is sexual called gametogony. This is the time the scouring is reported to take place; around 17 days in cattle. The oocyst then passes in the stools and under the right conditions of warm, moist weather sporulate to restart the cycle.

Diagnosis is fairly easy. Run fecal flotations! Necropsy and histopath are also diagnostic, but if they do get to that point, the coccidia should be quite apparent in terminal small intestinal contents and readily seen on flotation. The disease must also be differentiated from other scours in calves. These include: *E. coli, Salmonella,* Rota, Corona, and BVD as well as Cryptosporidium.

The epidemiology of the disease is the most interesting aspect of the disease and where we can make some of our comparisons to other species of animals to aid in prevention and control. The first item to consider is exposure. The sporulated oocysts are tough, sticky little buggers. Washing and scrubbing with disinfectants will help but cannot eliminate the coccidia. The only means to kill them is high concentrations of ammonia which cannot safely be done. The next means to reduce exposure is a physical barrier. An idea stolen from swine producers is to paint over the contaminated walls. The floor must initially be clean and a good layer of straw is usually adequate to reduce exposure.

The disease is also self limiting. This means that severity depends on how many coccidia are ingested. If the animal is removed from the contaminated environment, the disease should continue to finish its course and problems subside.

Paper presented at the Dairy Herd Health Programming Conference (1986), University of Minnesota, St. Paul, MN, Dr. James Hanson, Coordinator.

Considering immunity, I would like to think chicken. There is definitely an immune response made to coccidia. When raising layers, the poultry producer generally puts them on amprolium. As adults, they usually go into cages and are essentially never exposed to coccidia. If they could potentially end up scratching around a farm yard, the raiser does not use amprolium. Instead another product such as nitrofurazone is used. This allows the birds to contract a limited amount of coccidiosis and produce an immune response. Thus as they move on to farm yards with greater risk, the immunity is already present.

I am sure some of the calves in our area are treated similarly. When coccidia are a problem, the farmers sometimes get overzealous with amprolium (CoRid®). This can be frustrating when calves are turned in to growing pens and in a short time succumb to coccidiosis. This is probably the same problem that feedlot calves have coming off clean, dry pasture onto a crowded lot. I would think some efforts to balance a limited exposure and immunity would help alleviate this problem.

Prevention and control as we have covered is primarily cleanliness and physical barrier. Again, in review, I would also like some answers from researchers to allow limited exposure with concurrent immunity.

Treatment is not effective in clinical cases. That's what the textbooks say. I'm convinced that amprolium works to stop the disease. Do **NOT** follow label directions! The double dilution with full and half strength concentrations are too confusing and compliance is extremely difficult. Amprolium 9.6% solution calculates to 5 cc/calf treatment (5 days) and 2½ cc/calf as prevention (21 days). This can easily be added directly to the milk or water.

Unfortunately, this may not present itself as a simple, uncomplicated disease. In our practice, we feel that coccidia works hand-in-hand many times with *Clostridium* enterotoxemia. It is reported that the epsilon toxin cannot penetrate the intestinal mucosa. However, with a severely compromised mucosa from coccodia, I question that. When we are seeing calves and feedlot cattle with nervous signs, we always include C & D antitoxin (Dybelon® Bio-Ceutic) along with thiamine and antibiotics in our treatment. This does give quite encouraging results.

I don't think I have shed a lot of new light on the problems that you are having in your practice. If possible, though, there may be some new ideas raised that could be considered by those in research to continue investigation.