Efficacy of Monensin Fed to Cattle Inoculated with Coccidia Oocysts

L. E. Watkins, *Ph.D.*
M. I. Wray, *Ph.D.*
R. P. Basson, *Ph.D.*
D. L. Feller, *D.V.M.*
R. D. Olson, *D.V.M.*
Lilly Research Laboratories
Greenfield, IN 46140
P. R. Fitzgerald, *Ph.D.*
University of Illinois
B. E. Stromberg, *Ph.D.*
University of Minnesota
G. W. Davis, *D.V.M.*
Greenbriar Veterinary Services, Inc.
Dublin, Ohio

Introduction

Monensin has been used to prevent coccidiosis in broilers since 1971.[1,2] In 1975, this same compound was approved as a feed additive in beef cattle.[3,4] Monensin has also been reported to be effective in treating and preventing coccidiosis in lambs and cattle.[5,8]

Coccidiosis is the third most prevalent health problem of cattle[9], and the clinical signs which include poor performance, bloody diarrhea, and mortality are readily identifiable. Therapeutic treatment of animals when signs are clinically visible does not always ensure survival.[10] Coccidiosis infection can damage the absorptive surface of the intestine which leads to lower rates of gain and feed efficiency by the infected animal.[10] Therefore, it is important that intestinal damage be prevented.

The purpose of this investigation was to determine the prophylactic dose range of monensin which is effective against cattle coccidiosis as measured by coccidial oocyst shedding, weight gain, feed intake, clinical signs, and mortality of ruminating cattle challenged with *Eimeria bovis* and/or *E. zuernii* oocysts.

Materials and Methods

One hundred thirty-nine ruminating Holstein-Friesian bull calves weighing initially an average of 150 to 188 lbs were used in four trials at three locations to determine the prophylactic effects of monensin fed at 0, 10, 20, or 30 g/ton of air-dry feed on cattle coccidiosis.

All animals were obtained and reared for a period of time in an isolated environment prior to the initiation of the trial. This age of calf and the isolation procedure were used to ensure that cattle free of previous exposure to coccidiosis were used in the trials. All animals were ruminating at the time the trials were initiated. Each animal was restrained in a separate location within a barn and subjected to an oral gavage of at least 200,000 sporulated coccidia oocysts on the third day after monensin feeding was begun. In three of the four trials, cattle with a "coccidia type" block were given a challenge consisting predominately of *E. bovis* or *E. zuernii,* although other species were present at a lower level in the inoculum. In the fourth trial, all cattle were in one block and were given a challenge which contained large numbers of sporulated oocysts of both *E. bovis* and *E. zuernii.*

The diets used in these trials consisted primarily of corn, soybean meal, oats, and hay, and were fed on an *ad libitum* basis.

The experimental design used at each location was a randomized block with several observations per cell. Coccidia challenge type (i.e., predominantly *E. bovis,* or predominantly *E. zuernii* or both) and/or initial weight were the blocking factors within each location. The monensin treatments were 0, 10, 20, or 30 g/ton. There were four to seven animals, treated alike with respect to challenge type, on each monensin treatment. Four animals were used as uninoculated controls to monitor the severity of the coccidia challenges at each location. These animals were not included in the statistical analysis.

The experimental variables measured in these four trials were as follows:

1. Body weight gain, lbs/head/day
2. Feed intake, lbs/head/day
3. *E. bovis* oocyst counts (1000/g of feces)
4. *E. zuernii* oocyst counts (1000/g of feces)

This paper was presented by Dr. R. D. Olson.

5. Fecal scores (1 = normal, 2 = slight diarrhea, 3 = diarrhea, 4 = diarrhea/blood, and 5 = diarrhea/mucus)
6. Other coccidial species oocyst counts (1000/g of feces), i.e., any oocysts other than *E. bovis* or *E. zuernii*
7. Mortality

Each individual animal was considered an experimental unit since all animals in all trials were individually infected, restrained, and fed in a separate location within a barn.

Trial lengths ranged from 33 to 38 days. Oocyst counts and fecal score data were collected on days -14, -7, 0, 7, 14 through 30, and 32. These data were viewed as sub-sampling units and were analyzed accordingly. Animal weights and feed intakes were measured weekly, but cumulative values analyzed over the entire length of the trial were analyzed. Mortality within each treatment group was recorded upon occurrence.

Results and Discussion

An unweighted least squares analysis was used for all variables except for mortality. Least square treatment means are given in Table 1.

Average daily gain was increased ($P<.07$) above that of inoculated controls by 20 and 30 g/ton monensin. Average daily feed was increased ($P<.05$) above that of inoculated controls by 30 g/ton monensin. The average monensin intake for the 0, 10, 20, and 30 g/ton treatment groups averaged 0, 0.4, 0.8, and 1.2 mg/kg of body weight, and 0, 32, 69, and 104 mg/head/day, respectively, over the treatment period.

TABLE 1. Effect of Monensin on the Weight Gain, Feed Intake, Fecal Occyst Numbers, Fecal Condition Scores, and Mortality of Ruminating Calves Challenged with Coccidia.

	Monensin Level (g/ton)			
Variable	**0**	**10**	**20**	**30**
Ave. daily weight gain, lbs/head/day	1.310	1.592 .36[a]	1.941 .07	1.960 .06
Ave. daily dry matter intake, lbs/head/day	5.488	5.578 .80	6.033 .13	6.168 .04
E. bovis oocysts, sq. root, 1000/g feces	49.936	29.077 .07	15.894 .01	11.163 .005
E. zuernii oocysts, sq. root, 1000/g feces	18.472	7.032 .04	2.313 .01	2.214 .009
Other oocysts, sq. root, 1000/g feces	19.275	9.233 .04	8.666 .002	4.710 .001
Feces scores, sq. root, scale 1-5[b]	1.40	1.28 .07	1.12 .04	1.09 .007
Mortality, % of initial number[c]	16	0	0	0
Daily monensin dose, mg/kg bodyweight	0	0.4	0.8	1.2
Daily monensin intake, mg	0	32	69	104

[a] The superscripts are P levels comparisons of the monensin level to the inoculated control treatment (0 g/ton).
[b] 1 = normal, 2 = slight diarrhea, 3 = diarrhea, 4 = diarrhea/blood, 5 = diarrhea/mucus.
[c] Calculated percent (No. died/No. initially) × 100.

From curve-fitting techniques, it was determined that average daily gain was significantly improved for cattle fed monensin at 15-30 g/ton monensin. Increases were progressive with increasing levels of monensin. The average daily gain response was calculated to reach a maximum level above 30 g/ton.

Average daily feed intake was improved for cattle fed 30 g/ton monensin. Average daily feed intake progressively increased with increasing levels of monensin which is the reverse of what has been historically seen in feedlot cattle fed monensin.[4] The monensin intakes for the 0, 10, 20, and 30 g/ton treatment groups averaged 0, 0.4, 0.8, and 1.2 mg/kg of body weight, and 0, 32, 69, and 104 mg/head/day, respectively.

E. bovis, E. zuernii, and other oocyst counts were decreased ($P<.04$ to .07) below those exhibited by the inoculated control animals by the 10, 20, and 30 g/ton monensin treatments. *E. bovis* and *E. zuernii* oocysts counts, feces scores, and other species oocysts counts were all significantly reduced below inoculated controls when monensin was fed at levels of 10-30 g/ton. While there is moderation of the response with increasing dose, reductions were progressive with increasing levels of monensin. Fecal scores were decreased ($P<.07$) below inoculated controls by 10, 20, and 30 g/ton monensin.

Mortality occurred in two of the four trials. All five cattle that died after receiving the coccidial challenges were in the inoculated control treatment. Monensin at 10-30 g/ton prevented the mortality seen (16%) in the inoculated controls.

Conclusions

Monensin at a range of 10-30 g/ton effectively controls coccidiosis in cattle exposed to severe oocyst challenges.

References

1. Shumard, R.F. and M.E. Callender. 1967. Monensin, a new biologically active compound. VI. Anticoccidial activity. *Antimicrob. Ag. Chemother.*, 369. 2. Reid, W.M., *et al.* 1972. Anticoccidial activity of monensin in floor pen experiments. *Poultry Sci.*, 51:139-146. 3. Potter, E.L., *et al.* 1976. Effect of monensin on performance of cattle fed forage. *J. Anim. Sci.*, 43:665-669. 4. Raun, A.P., *et al.* 1976. Effect of monensin on feed efficiency of feedlot cattle, *J. Anim. Sci.*, 43:670-677. 5. Bergstrom, R.C. and L.R. Maki. 1974. Effect of monensin in young crossbred lambs with naturally occurring coccidiosis. *JAVMA*, 164:288-289. 6. McDougald, L.R. and W.J. Dunn. 1978. Efficacy of monensin against coccidiosis in lambs. *Am. J. Vet. Res.*, 39:1459-1462. 7. Fitzgerald, P. and M.E. Mansfield. 1973. Efficacy of monensin against bovine coccidiosis in young Holstein-Friesian calves. *J. Protozool*, 20:121-126. 8. McDougald, L.R. 1978. Monensin for the prevention of coccidiosis in calves. *Am. J. Vet. Res.*, 39:1748-1749. 9. Bertelsen, A. 1982. Vets rank coccidiosis as major health concern. *Feedstuffs*, April 19, p. 16. 10. Edwards, A.J. 1984. Digestive diseases in feedlot cattle. *Animal Nutrition and Health*, Jan-Feb.

Questions & Answers:

Question: Have you done CNS analyses?

Answer: Not on these animals. We've done that in the past. CSF analyses are typically normal with a possible exception of CSF glucose. There have been a couple papers in the literature stating that you will have a hyperglycemia and glucosuria and you will get a high CSF glucose level, but otherwise CSF, as far as I know, should be normal.

Question: Is there any toxin in the CSF?

Answer: We have not looked at that. I would think that toxin in the CSF would be exceedingly difficult to find, probably not there. And I should make a point that in a clinical cases and in research animals where botulism has been produced, it is very difficult to find toxin in the peripheral circulation, in the serum. In people, yes, in pigs, yes, because they are much more resistant. It takes a lot more toxin to produce clinical signs. But it is almost impossible to find toxin in the serum, and I would think in the CSF of infected animals.

Question: Can you continue to ship the milk?

Answer: Yes, there probably is not enough toxin to be detectable there. Most of the toxins are bound to the motor-end plates and not excreted, as far as we know, or at least excreted at extremely low levels.

Question: What are the sources?

Answer: The sources of that have been in the past hydraulic oils used in the aircraft industry. Lubricants which are contaminated with this. Somebody brings a barrel home and it has some of this oil in the bottom of it. They mix other things with it, pour-ons, and something like that, insecticides, and it is absorbed through the skin. So in the literature and in the couple of cases that I've dealt with, it has been people working with airplanes, and so on, bring some of this stuff home and then get it mixed in with some dips and something like that.

Question: What were your cases?

Answer: Two were rye silage which was abnormally fermented. The pH was not low enough and if you don't get the pH below 5 then the *Clostridium botulinum* spores can continue to grow and produce toxin.

Questions & Answers:

Question: From the studies in calves, were most of them derived locally or were some of them from other areas outside the state?

Answer: I think a lot of them are brought in from Canada, from northern Pennsylvania, New Jersey, and surrounding states. But, of course, a lot of them are locally grown. Something I didn't really have a chance to talk about was, the Canadians have done a lot of phage biotyping studies and they are finding the same *Salmonella typhimurium* phage biotypes in their cattle as we are, so there probably is a dissemination back and forth across the border. And as far as I know, no other studies have been done in cattle populations in the United States doing phage and biotyping, so we really have nothing to compare it with. Many times a lot of these herds, especially the veal calves, are getting growth promoting levels of antibodies. A lot of the cattle, most of them, are treated clinically before we actually make a diagnosis by isolation. We're probably not getting antibodies. I think that's reflected in what we saw in molecular epidemiology. The calf strains had many plasmids which were selective for antibiotics and the adult cattle do not have plasmids. That's also true in the equine population. They have many plasmids and they traditionally get more antibiotics.

Question: If an adult cow is infected, once a carrier, is it always a carrier?

Answer: I think the problem there is doing controlled studies with *Salmonella* dublin infected adult animals which usually carry for life. But with *Salmonella typhimurium* I've mentioned cases that have been shedding over a year but you can't rule out the possibility of recontamination from the environment, so you really don't know without good controls that it hasn't been reinfected. But I'd say probably 6-8 months is maximum for *typhimurium.* Some of the Group E's, they're shedding, just as long.

Question: What happens?

Answer: Many times they elect to have an autogenous bacterin made and I think the problem is, once they recognize they have a *Salmonella* problem, by the time you get a bacterin made and administered to the herd, the outbreak is winding down any way. So the bacterin looks efficacious. We've recommended to these owners that vaccination isn't the only thing that can be done. It's mostly a management problem at that point. But certainly vaccination of dry cows for passive immunity of calves is highly recommended.

Question: Are humans at risk?

Answer: I think definitely since it is a zoonotic disease humans are at risk, especially small children. We've had a number of these outbreaks with human involvement. The herdsmen, small children involved around the barn. Certainly restriction is probably a role. It's a management problem I feel. It's very hard to keep manure off your person when you're working with the animals but hygiene is the best thing you can do. As far as drinking raw milk, I would say no. It should be pasteurized, at least for the immediate period of the outbreak.

Questions & Answers:

Question: When did you conduct the trial?

Answer: This trial started in the fall of the year. I would guess that if we ran it in the summer with the heat it would have been more excentuated. But it started about September, October, and went through about January/February. In a short term trial like this, 8 or 10 weeks post-calving, reproductive performance isn't really that useful to us. I should mention low-chloride cows, whenever we decided they were going to die tomorrow if we didn't treat them today would be treated with 16 liters of a commercial solution and within one to two weeks we significantly brought back all the blood parameters within 3 to 4 of their body weights and milk was beginning to come back, never to where they would have been. But we were able to do some reversals.

Question: What did you feed them?

Answer: Corn silage, shelled corn, soybean meal.

Question: How did you maintain necessary levels?

Answer: TMR. Everything's balanced. And the way we get sodium in without chloride was by using sodium bicarbonate. The other ones had salt. That's how we varied and kept sodium equal in all rations but varied chloride.

Question: What did you check?

Answer: We checked urinary pH's. Some of those cows were quite high, quite alkaline. I can't remember all the numbers, but they were checked.

Question: How did you sample them?

Answer: There were spot samples two or three times a

day on all cows. They're just an index of chloride content of the urine, not total amount of chloride excreted per day. But in the case of low chloride cows, you couldn't even measure the amount of chloride in the urine, so the total collection wouldn't have done much for us. They were down to almost basically zero concentration so that the volume times concentration still wouldn't have yielded as many grams on the low cows. We did not do total collection.

Question: Does a cow have a selective appetite for these?

Answer: We think there's pretty good evidence that a cow has a selective appetite for sodium and probably for chloride. Beyond that we don't feel there's very good evidence. So probably if these cows would have had access to free-choice salt, they probably would have presented the problem. If we had extended it to six months we would have had six dead cows and I wasn't, as an assistant professor going for tenure, willing to take that chance! I don't know what we would have seen postmortem if we had slaughtered and looked at them. We were not brave enough to go six months. Four or five weeks were enough to get the job done for what we were after.