

# The Effect of Dexamethasone on Bovine Coccidiosis

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## SUMMARY

When dexamethasone was administered intramuscularly to Hereford calves at the time of inoculation with *Eimeria zurnii* and *E. bovis*, there was no apparent effect on the resulting infection. Medication at the time of appearance of the clinical signs caused sufficient aggravation of the disease to result in the death of the animals. Administration of the drug after subsidence of the clinical signs did not cause a clinical relapse, but resulted in prolonged oocyst discharge in the feces. Treatment of uninoculated normal calves with dexamethasone caused a brief period of increased oocyst discharge without clinical signs.

## RÉSUMÉ

Si on administre de la dexaméthasone par voie intra-musculaire à des veaux Hereford, en même temps qu'on les inocule avec *Eimeria zurnii* et *E. bovis*, on n'observe aucun changement dans l'infection consécutive. L'administration du médicament au moment de l'apparition des symptômes de la maladie entraîne une aggravation telle, que la mort des animaux peut s'ensuivre. Son administration après une atténuation sérieuse des symptômes n'entraîne pas de rechute clinique, mais provoque un allongement de la période pendant laquelle les animaux évacuent des oocystes dans leurs fèces. Des veaux normaux traités à la dexaméthasone, sans subir d'inoculation, présentèrent une brève période au cours de laquelle ils éliminèrent plus d'oocystes, sans toutefois manifester de symptômes.

## INTRODUCTION

Corticosteroids appear to be useful in the study of various parasitic diseases since they suppress the host's immune responses and thus modify the course of a disease. Numerous investigators have studied the effects of corticosteroids on parasitic infections in several animal species. The influence of these substances has been shown in helminth infections (2, 5, 6, 7) and in natural and experimental infections caused by various protozoan parasites (1, 4, 10, 11). There are only a few reports on the use of corticosteroids in experimental coccidial infections. McLoughlin (4) successfully transmitted the turkey parasite *Eimeria meleagrimitis* to chickens treated with dexamethasone and Long (3) reported the occurrence of schizogony of *E. tenella* in the liver of chickens under the influence of this drug.

Apparently there are no references on the use of corticosteroids in the study of coccidial infections of cattle.

This report describes experimental and natural coccidial infections in calves treated with a synthetic corticosteroid, dexamethasone.

## MATERIALS AND METHODS

Hereford calves, five to ten months old, were used in the three experiments. They were stanchioned in a barn on an unbedded wooden platform raised about 15 cm above the cement floor, but not individually isolated. Manure was removed daily, but no special precautions were taken to maintain cleanliness. Bodily contact between adjacent animals was possible. The animals

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were fed good quality alfalfa hay, salt-mineral mix and water *ad lib*.

For experimental infection, the calves were inoculated by ruminal intubation with 400,000 sporulated oocysts of *Eimeria*, containing the following species: *E. zurnii*, 75%; *E. bovis*, 20%; *E. ellipsoidalis*, *E. canadensis*, *E. auburnensis* and *E. subspherica*, 5%. The oocysts originated from natural clinical cases of coccidiosis in cattle and were prepared as described previously (9).

Fecal samples were collected daily and the coccidian oocysts were counted with a McMaster chamber.

Dexamethasone (9-alpha-fluoro, 16-alpha-methyl prednisolone)<sup>1</sup> solution was injected by the intramuscular route at 20 mg per animal at predetermined intervals.

#### EXPERIMENT I

Eight calves, each of which since birth had been kept isolated with its dam in separate boxstalls, were weaned and stanchioned at the age of five months. They weighed approximately 125 kg. Periodically, these calves were found to discharge a few coccidian oocysts in the feces, but were considered to have had minimal exposure to coccidia.

Each calf was inoculated with oocysts and dexamethasone was administered on the following dates: (i) two animals (Nos. 14 and 15) received the drug on the day of inoculation (day 0) and day 2; (ii) three animals (Nos. 5, 6 and 12) were injected on day 20 and day 22 when the clinical signs of coccidiosis had already appeared; (iii) three calves (Nos. 2, 4 and 7) were given the treatment on day 34 and day 36 when they were recovering from the infection. The oocyst shedding was monitored from day 0 to day 57.

#### EXPERIMENT II

Five calves which survived Experiment I (Nos. 2, 4, 7, 14 and 15) and were considered to be immune to clinical coccidiosis were given a single dose of dexamethasone at the age of seven months. This time they were not inoculated with coccidia. The oocyst counting began ten days prior to treatment and continued until 30 days post-treatment.

<sup>1</sup>Azium, by Schering Corporation Ltd., Pointe Claire, Quebec.

#### EXPERIMENT III

Eight "normal" steer calves, nine to ten months old, and weighing approximately 230 kg, were used for repeated administration of dexamethasone in the absence of inoculation with coccidia. These calves were born and kept on dry pasture in the summer, weaned in the fall and kept in a dry corral for three months prior to confinement for experiment. These animals were considered to have had moderate natural exposure to coccidia but had not suffered from clinical coccidiosis.

Two of these animals (Nos. 23 and 45) were treated with dexamethasone once, two (Nos. 43 and 73) were treated on four consecutive days, and another two animals (Nos. 52 and 69) were injected on three consecutive days and the same schedule was repeated five days later. Two additional animals were treated with dexamethasone three times weekly for a total of four weeks. Coccidian oocyst checking was conducted as in Experiment II.

### RESULTS

#### EXPERIMENT I

Figure 1 shows the oocyst counts per gram of feces in logarithmic units for animals treated with dexamethasone at the time of inoculation and shortly thereafter. Fig. 2 shows similar graphs for animals treated at later stages of the infection. *E. zurnii* was the predominant species found in all peaks of the oocyst discharge patterns.

All eight calves were clinically affected and developed diarrhea. Blood appeared in the feces of calves 13, 5, 6, 7 and 4. The three calves (13, 5 and 6) which were treat-

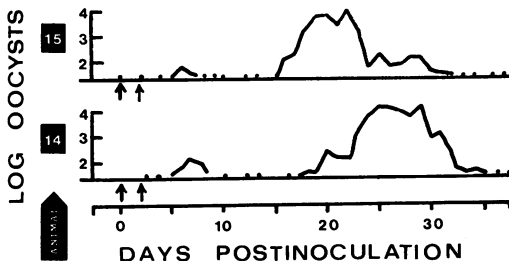


Fig. 1. Experiment I. The coccidian oocyst counts per gram of feces of two calves treated with dexamethasone on days 0 and 2 after inoculation with 400,000 sporulated oocysts. Arrows indicate dexamethasone treatment. Days 38 to 57 are not shown on the graph.

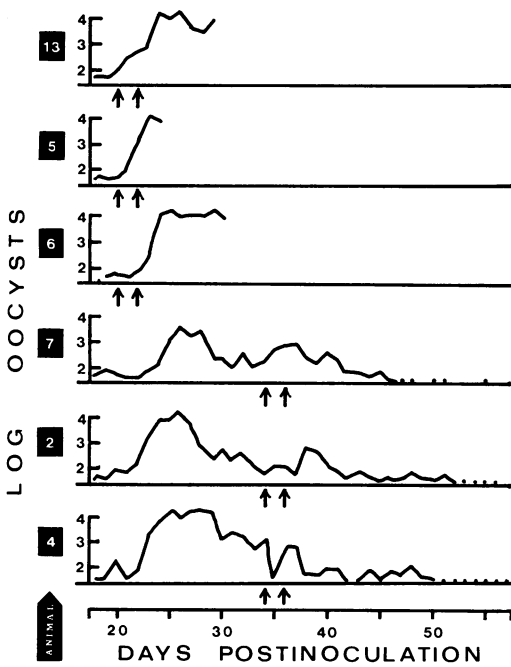


Fig. 2. Experiment I. Oocyst counts of three calves treated with dexamethasone on days 20 and 22, and of three calves treated on days 34 and 36 after inoculation with 400,000 sporulated oocysts. Arrows indicate dexamethasone treatment. Days 0 to 18 are not shown on the graph. The end of the curve for animals 13, 5 and 6 signifies their death.

ed at the time of the clinical disease had the most severe enteric signs, depression, anorexia and died during days 24 to 30. The five remaining calves recovered from the clinical disease within five to seven days, but animals 7, 2 and 4 continued to discharge large numbers of oocysts for a longer time than animals 14 and 15.

### EXPERIMENT II

The oocyst discharge patterns are shown in Fig. 3. There were no coccidian oocysts found in the feces of these calves when tested for ten days prior to dexamethasone administration. None of the animals developed any detectable clinical signs of disease. The predominant coccidial species were, in the order of frequency; *E. bovis*, *E. zurnii* and *E. ellipsoidalis*.

### EXPERIMENT III

Figure 4 shows the oocyst discharge patterns for six of the eight "normal" uninoculated animals treated with dexamethasone. The other two animals treated for four

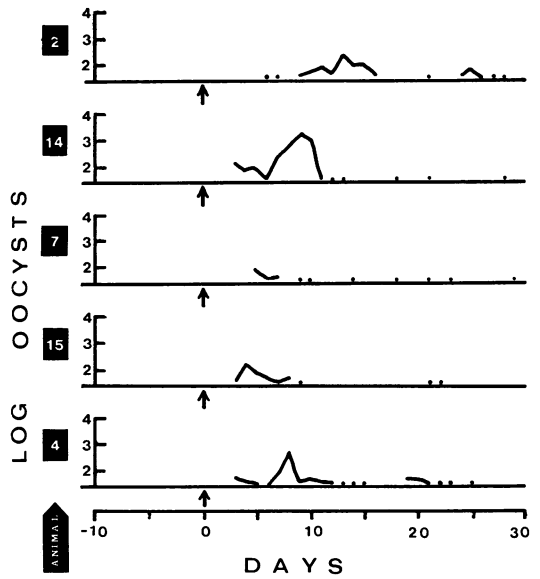


Fig. 3. Experiment II. Oocyst counts of five calves which survived Experiment I and were given a single dose of dexamethasone two months later. Arrows indicate dexamethasone treatment.

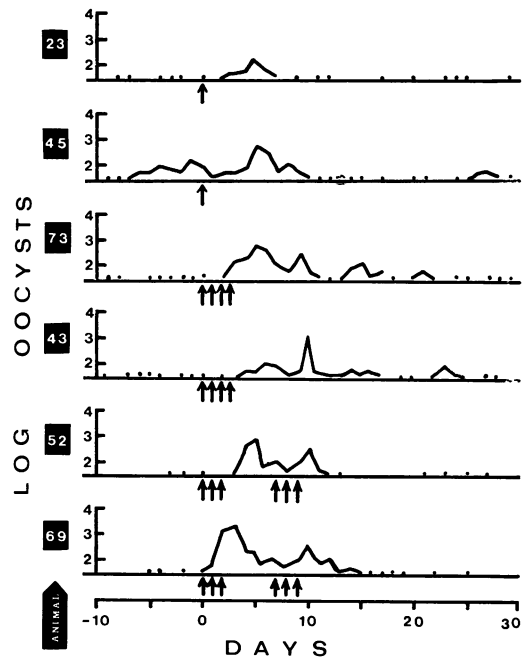


Fig. 4. Experiment III. Oocyst counts of six "normal" (uninoculated) calves following single or repeated injections of dexamethasone. Arrows indicate dexamethasone treatment.

weeks showed only initial peaks of oocyst discharge, similar to those of animals 52 and 69, followed by small numbers of oocysts irregularly as in animal 23; these two are not shown in Fig. 4. Calf 45 discharged many oocysts prior to treatment with dexamethasone. None of these animals developed any obvious clinical signs. The predominant coccidial species in these calves was *E. bovis*. *E. zurnii* predominated on only two occasions and *E. ellipsoidalis* once (in counts of more than 100 oocysts per gram of feces).

## DISCUSSION

The results of these experiments imply that dexamethasone may exert a significant effect on coccidial infections in cattle. As dexamethasone is known to suppress immune mechanisms, particularly at the cellular level (8), the exacerbation of the clinical infection and increased oocyst discharge following treatment with this drug may indicate that the cellular factors of the host are important in resistance to coccidiosis caused by *E. zurnii* and *E. bovis*.

Although small numbers of oocysts were shed often, particularly by the naturally infected animals, the largest numbers of oocysts were discharged two to ten days after the treatment. Even animal 45 (Fig. 4) which began discharging many oocysts prior to dexamethasone administration, showed a marked discharge peak (nearly ten-fold or one log unit increase) four days post-treatment, followed by decline to negligible level at day 9. This indicates that the infected host's response to show a markedly increased oocyst discharge appears to occur between two to ten days after administration of dexamethasone. Animals given dexamethasone at the time of inoculation with the coccidia showed a brief increase in oocyst discharge on days 6 and 7 (assumed to originate from a previous low-level infection, and influenced by dexamethasone), but the clinical infection and the peak oocyst discharge pattern did not differ appreciably from those of previously unmedicated animals (compare appropriate portions of graphs on animals 14 and 15 vs. 2, 4, and 7 of Figs. 1 and 2).

In the naturally infected ("normal") calves the dexamethasone-induced oocyst discharge peaks failed to be sustained upon prolonged treatment with this drug, al-

though physical conditions existed for continual natural reinfection. This suggests accumulation of coccidial developmental stages in the intestinal epithelium of animals with some degree of immunity. When the host's defences are modified under the influence of dexamethasone, such accumulated forms would then mature and be discharged as a brief shower of oocysts.

A clinical implication of dexamethasone administration, which likely applies to all corticosteroids, would be the enhancement of the disease if the drug is given during a critical period.

As an experimental aid, dexamethasone could be used to detect inapparent coccidia carriers. Such an approach had been recommended and successfully used to detect bovine carriers of the blood protozoa, *Babesia argentina* (1).

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