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Bovine Coccidiosis – A Review

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Abstract

Coccidians are protozoal parasites that are host specific, transmitted by the fecal-oral route and cause enteritis. Economically significant species discussed in this paper that cause disease in cattle belong to the genus *Eimeria.* Young animals are more susceptible to clinical disease than older cattle. Coccidiosis is frequently observed in stressed, overcrowded and confined conditions, however, the disease can occur on pasture. The disease can occur any time of the year but is more prevalent during winter months, even in confinement operations. Animals may pass oocysts in their feces without clinical disease, therefore, a diagnosis of coccidiosis is based on clinical signs and ruling out other diseases. Identification of coccidian postmortem specimens supports the diagnosis.

Management procedures that reduce stress and prevent contamination of feed and water are important in preventing coccidiosis. Effective control programs also commonly incorporate the use of medicated feed or water. Drugs commonly used in the US for coccidiosis control and prevention programs include monensin, lasalocid, decoquinate and amprolium. When clinical disease is present, amprolium, sulfonamides and management changes are used to control the disease.

Résumé

Les coccidies sont des protozoaires parasites, transmissibles par la voie fécale-orale, qui sont spécifiques à leur hôte et qui entraînent des entérites. Les espèces traitées dans cet article appartiennent au genre *Eimeria*. Ces espèces ont un impact économique et causent des maladies chez le bétail. Les jeunes animaux sont plus sensibles aux maladies cliniques que les animaux plus âgés. La coccidiose est fréquemment observée chez les animaux stressés, entassés et confinés dans des espaces restreints. Toutefois, la maladie peut aussi prendre place au pâturage. La maladie peut survenir en tout temps de l'année mais elle est plus commune en hiver même dans les cas de confinement. Les fèces peuvent contenir des oocystes même lorsque les animaux ne montrent pas de signes cliniques de sorte qu'un diagnostic de coccidiose doit se baser sur les signes cliniques et exclure les autres maladies. L'identification post-mortem de coccidies aide à supporter le diagnostic.

Les éléments de régie qui réduisent le stress et préviennent la contamination de l'eau et de la nourriture sont importants pour prévenir la coccidiose. Les programmes de contrôle efficaces incluent souvent l'utilisation de nourriture et d'eau médicamentés. Les agents antimicrobiens couramment utilisés aux États-Unis pour le contrôle et la prévention de coccidiose incluent entre autres le monensin, le lasalocide, le décoquinate et l'amprolium. Lorsque des signes cliniques se manifestent, l'amprolium, les sulfamides et des changements dans la régie sont utilisés pour contrôler les maladies.

Introduction

Production animal agriculture has changed considerably during the past few decades. Many production animal units have increased in size to improve efficiency. Transmission of infectious diseases is frequently enhanced in these larger units. Coccidiosis commonly becomes more economically significant as management systems become more intensive. A good understanding of the coccidian life cycle, pathogenesis of the disease and control, prevention and treatment options is essential to optimize animal health in cattle operations.

The Organism

Coccidians are protozoal parasites. Those discussed in this paper belong to the genus *Eimeria. Eimeria* spp are host specific, and *E. bovis* and *E. zuernii* are considered the most common cause of coccidiosis in cattle.[7,11,33] Coccidia are transmitted by the fecal-oral route.

The life cycle of coccidians is characterized by both asexual and sexual stages. The asexual phase of the coc-

cidia life cycle includes sporozoite, schizont (also called meront), merozoite and meront stages, while the sexual phase includes macrogametes (female sex cells) and microgametes (male sex cells). The macrogametes and microgametes are collectively referred to as gamonts.

Infected animals pass unsporulated oocysts in their feces, which contaminate the environment. Once oocysts sporulate they become infective and are protected from the environment by double cyst walls.[31] Sporulation, which can occur in a few days or a few weeks, is dependent upon time, temperature and moisture.[59] Cattle ingest sporulated oocysts by consuming contaminated feed and water or by grooming themselves.

Once in the intestinal lumen, sporozoites are released from the oocysts and penetrate the epithelial cells of the host. The invasion process is facilitated by microneme proteins of the parasite which form close interactions with actin-myosin motility-based complexes.[4] Inside the host cell, asexual reproduction occurs, producing merozoites in the schizonts. As the organism continues to reproduce, host cells rupture and release more merozoites into the intestinal lumen, which infect additional intestinal epithelial cells. After a specific number of asexual generations, merozoites produce macrogametes and microgametes. Through sexual reproduction the macrogamete and microgamete come together to form a zygote, producing new oocysts. The oocyst matures, the host cell ruptures and unsporulated oocysts are released. The unsporulated oocysts are passed in the feces, thus completing the cycle. Different coccidian species have different predilection sites in the gastrointestinal (GI) tract. *E. bovis* and *E. zuernii* primarily infect the distal ileum, cecum and colon.[62,79]

Pathogenesis

Clinical disease is usually the result of infection with more than one species.[47] During the first generation of asexual reproduction, schizonts are formed in relatively low numbers, generally producing minimal damage to the intestine and minimal or no clinical signs are observed.[79] If the colon is unaffected at this stage, it can compensate and maintain the animal's hydration status. As the cycle continues and more meronts and gamonts are formed, more tissue damage occurs and blood and plasma leak into the intestinal lumen. As reproduction continues, the large intestine is unable to compensate and diarrhea is observed. As the disease progresses and more tissue is damaged, intestinal mucosa is sloughed and hemorrhage occurs.[62] Segments of mucosa, fibrin and blood clots are often visible in the feces. Because the intestine is no longer able to efficiently absorb nutrients and electrolytes, various imbalances occur.

Investigators have evaluated water, sodium and potassium metabolism in calves experimentally infected with *E. bovis*. Although water and electrolyte metabolism was altered, physiologic adaptation occurred.[9] Cattle become depressed, reduce feed and water intake and lose weight. In severe infections, animals may die. Cattle that survive begin eating within a few days. In some individual cases, the intestine may not fully repair itself for several weeks, hence, performance in these individuals will not be optimal during the repair process.[21,79]

Another clinical syndrome attributed to infection with coccidia is nervous coccidiosis (also called nervous enteritis). Affected animals may present with muscular twitching, strabismus, nystagmus, opisthotonos, paddling, recumbency, seizures and death. The exact mechanism that causes these neurological signs is unknown, but a neurotoxin or an imbalance is suspected.[33,69]

By experimentally challenging calves with coccidia, researchers have correlated clinical signs with time of infection. Twelve Holstein-Friesian calves 2-4 months of age were divided into two groups; the challenged group was infected with *E. zuernii* sporocysts while the other group served as non-infected controls. Diarrhea with mucus was observed in infected calves on days 17 and 18 following inoculation, and dysentery and fibrinous casts were observed on day 19. Two calves died on day 20. Infected calves gained weight through day 21, but lost weight during days 22 - 26. Calves appeared to regain the weight until day 30, which was the end of the trial.[71] Coccidiosis is generally not considered to cause diarrhea in dairy calves less than 30 days of age.[43]

When susceptible young animals ingest large numbers of sporulated oocysts, they either clear the infection, becoming immune, or the disease becomes severe enough to require treatment. Another hypothesis proposes that when calves are stressed, such as at weaning or when exposed to severe weather, arrested endogenous stages of the organism in the calves may be activated. This hypothesis is supported by the fact that sporozoites do not develop immediately in all instances.[47]

Cattle frequently exhibit more severe clinical signs of coccidiosis following stressful conditions. The severity of clinical signs is dependent upon an individual calf's resistance to infection and/or the challenge dose of the organism. Even immune animals can succumb to overwhelming challenges. A study investigating the effects of glucocorticoids (dexamethasone) on oocyst output was conducted in 16-week-old Holstein bull calves using sporulated *E. zuernii* oocysts. The oocyst output of dexamethasone-treated calves increased approximately tenfold, suggesting that stress and glucocorticoids appear to play a significant role in oocyst output.[72]

Excessive amounts of corticosteroids can suppress the immune system. In another study, dexamethasone (20 mg) was administered at different times to 5-to-10-month old Hereford calves that had been inoculated with sporulated *Eimeria* spp oocysts. Three of eight calves developed severe clinical disease and died. The surviving five calves were then given a single 20 mg dose of dexamethasone. No clinical disease was detected, however, all five calves shed oocysts. The researchers then investigated the response of naturally exposed calves to dexamethasone. Eight steers 9–10 months of age were administered dexamethasone at various times. Few oocysts were detected in the feces prior to dexamethasone administration. None of the steers developed clinical disease after receiving dexamethasone, but increased numbers of oocysts were detected in the feces. This increased shedding could be a source of infection to other cattle.[53] As the environment becomes heavily contaminated with oocysts, cattle are more likely to acquire infections and develop serious disease.

Immunity plays a role in subsequent infections. In an experiment using six-week-old Holstein-Friesian calves, five calves were assigned to five different groups, including an uninoculated, non-medicated control group. Inoculated, non-medicated calves that survived had decreased water consumption for 4 or 5 weeks and decreased feed intake for 13 weeks. Calves that were inoculated but did not receive any medication weighed 10 lb (22 kg) less than uninoculated, non-medicated controls, and 12.3 lb (27 kg) less than inoculated treated calves.[29] Calves were challenged a second time 3.5 months later. Only a few oocysts were identified in the feces, and only a few calves exhibited loose stools. The researchers concluded that protective immunity existed.[29]

Neonates have a functional but naive immune system. Canadian researchers investigated the immune response of newborn calves exposed to 100 oocysts at 3 to 40 hours of age, and subsequently challenged at five, seven and nine months of age. Four calves were exposed as neonates and three served as non-exposed controls. All calves were housed in an isolation facility to prevent natural exposure. Control calves developed clinical coccidiosis after the first challenge, but no signs were observed following subsequent challenges. Calves exposed to coccidia as neonates developed diarrhea after the challenge at five months of age, but it was of shorter duration than controls. These calves also developed mild diarrhea for up to four days following subsequent challenges. It was concluded that the challenge at five months of age was an immunizing dose for control calves, which developed immunity to subsequent challenges. On the other hand, the level of immunity stimulated by exposure to oocysts as neonates was insufficient to provide protection. Calves infected at a very young age may shed oocysts following subsequent challenges, and thus be a source of infection to other animals.[52]

Immunity resulting from previous exposure has been demonstrated in several other experiments. Holstein-Friesian calves 3 to 4 weeks of age were administered 10, 100, 500, 1,000, 5,000 or 15,000 sporulated *E. bovis* oocysts in milk for 47 to 62 days. Calves that received the highest challenge dose of oocysts exhibited more severe clinical signs than the other calves. The previously infected calves were then administered a single large challenge dose of sporulated oocysts. Most of the calves resisted re-infection as evidenced by few or no clinical signs, or discharge of oocysts. Control calves were not given daily doses of oocysts but did receive a single challenge dose of sporulated oocysts. Control calves exhibited severe clinical signs and discharged numerous oocysts. Calves that received 500 or more sporulated oocysts daily had better immunity than the calves that received 10 or 100 oocysts per day.[22]

Immunity to one species of coccidian does not cross protect against another species. The level of immunity following exposure may vary. Animals with partial immunity may not exhibit clinical disease, however, infections can still occur. These individuals pass oocysts and, therefore, continually contaminate the environment.[67]

Significance/Prevalence

In a Canadian review, coccidiosis was common in beef cattle, primarily affecting six-to-12-month-old calves in confinement during fall and winter months. Up to 20 to 30% of cattle in herd epidemics exhibited nervous signs including convulsions, opisthotonus, muscle tremors, nystagmus and blindness. The case fatality rate of cattle with nervous signs was approximately 50%. Of the cattle that died with nervous signs, 91% also had evidence of colitis and typhlitis.[62]

Outbreaks of coccidiosis in the summer have been reported. In a summer outbreak in 1976 in Wyoming, five ranches experienced an approximate 3% death loss in calves due to coccidiosis. Neurologic signs were predominant in affected animals. *E. zuernii* was the most common coccidian identified in fecal samples. Heavy contamination around water sources and grazing short contaminated pastures during the dry season likely contributed to the outbreak.[41]

Although coccidiosis is primarily recognized in confined overcrowded conditions, oocysts can be identified in feces from cattle grazing rangeland. Fresh fecal samples were collected at 60-day intervals from April to December from six ranches in South Dakota. Calves had higher numbers of oocysts than replacement heifers or cows; cows had lower oocyst counts than heifers. *E. bovis* was the most frequent species identified throughout the study. In fact, all species of *Eimeria*

identified were more prevalent during the spring, but were present throughout the study. Results indicated that persistence or reinfection occurred in the herds and continuous infection was maintained.[65]

Weaning is a stressful event for calves. Researchers in Australia investigated an outbreak of coccidiosis and the effects of weaning stress on oocyst production. An outbreak of diarrhea occurred three to four weeks post-weaning. *E. zuernii* was the predominant coccidian species identified, and maximum oocyst counts occurred 29 days after weaning. The researchers concluded that the stress of weaning precipitated the clinical disease.[55]

An outbreak of coccidiosis in a herd of Friesian dairy cows has been reported. Conditions that contributed to the outbreak included severe drought during the summer and housing the cows in a covered yard with straw bedding that became heavily soiled with feces. Initially, a six-year-old cow in late lactation developed bloody diarrhea and tenesmus. Three more adult cows six to eight weeks into lactation also developed clinical signs. No *Salmonella* spp were isolated from feces, however, 2800 and 3000 oocysts (primarily *E. zuernii*) per gram of feces were identified. Adult cows frequently shed less than 50 oocysts per gram of feces.[34]

Diagnosis

Initial diagnosis is generally based on clinical signs of diarrhea, dysentery, tenesmus, fibrinous casts and pale mucous membranes in at-risk animals. Differential diagnoses for these clinical signs would include bovine viral diarrhea virus (BVDV), salmonellosis, winter dysentery, cryptosporidiosis, internal parasitism, acidosis and arsenic toxicity. Identification of pathogenic coccidian species, along with appropriate clinical signs and ruling out other diseases, supports a diagnosis of coccidiosis. Although not all species are pathogenic, disease can result under the right conditions if high numbers of low pathogenic species are present.

Oocysts can be identified microscopically either by direct smear, flotation or centrifugation methods. Oocyst counts of 5,000 per gram of feces or greater are considered significant in cattle.[61] The mere identification of oocysts in feces does not confirm a diagnosis of clinical coccidiosis.[31] In a Georgia study, investigators analyzed 1,090 fecal samples from calves for coccidia oocysts. None of the calves exhibited clinical signs of coccidiosis, although one calf had 45,800 oocysts per gram of feces.[12]

During necropsy examination, the colon and cecum should be examined for the presence of suggestive lesions. Appropriate tissues should be submitted for microscopic examination. Histologically, edema of the intestines and identification of coccidia can help support the diagnosis.[34] Ruling out other enteric diseases is paramount, since oocysts can be identified in cattle without clinical coccidiosis.[42]

Prevention/Control/Treatment

Stress, challenge dose and immune status influence the calf's response to challenge. Several management issues should be addressed whenever treatment, control or preventive programs are instituted. Minimizing stress through herd health preventive programs including prevention of other diseases, proper nutrition, and internal and external parasite control will improve the overall health of individual animals. Waterers, feeders and equipment should be cleaned regularly to reduce fecal contamination. Dietary changes should be as gradual as economically possible, and overcrowding should be avoided.

Several drugs are available to control and treat coccidiosis (Table 1). In large production units, drugs that can be mixed in the feed or water for mass medication are desirable.

Prevention / Control. Ionophores are commonly used in cattle production. Ionophores, such as monensin and lasalocid, are used to improve the efficiency of rumen fermentation, increase daily gain and are effective coccidiostats. The exact mechanism of action of these compounds is unclear, however scientific information indicates it is related to the transport of various ions across cell membranes.[45] Ionophores appear to have an effect on the cell membrane of sporozoites and merozoites, changing the membrane integrity and intracellular osmolality.

In the US, monensin is a feed additive for cattle indicated "for improved feed efficiency, for increased rate of weight gain, and for the prevention and control of coccidiosis caused by *Eimeria bovis* and *Eimeria zuernii*".[19] Optimal dosing of monensin to calves for the control of coccidiosis has been investigated. Ten-week-old Holstein-Friesian calves were fed diets containing no monensin (controls) or diets formulated to provide 0.11 mg/lb (0.25 mg/kg) of body weight (BW), 0.45 mg/lb (1.0mg/kg) BW or 0.91 mg/lb (2.0 mg/kg) BW. The medicated feed was fed beginning three days prior to challenge with sporulated *E. bovis* oocysts. Control calves experienced significant weight loss and did not regain the lost weight during the 30-day observation period. Oocyst discharge was low or zero for the medicated calves, and no clinical signs of coccidiosis were observed, indicating that monensin was effective at those dosages. Additionally, researchers observed that weight gain was best in calves that received the diet containing 0.45 mg/lb (1.0 mg/kg) BW of monensin.[28]

It is difficult to consistently reproduce clinical disease when calves are experimentally challenged with

Table 1. Drugs labeled for coccidiosis treatment and control in the US.

Active Ingredient	Use	Dosage	Indications
Amprolium	calves (beef & dairy)	2.27 mg/lb (5 mg/kg) BW/d x21d	prevention
		4.55 mg/lb (10 mg/kg) BW x5d	aid in treatment
Decoquinate	cattle	13.6-27.2 g/T (liquid in dry feed)	prevention
		13.6-535.7 g/T (from dry feed)	prevention
Lasalocid	cattle	0.45 mg/lb (1.0 mg/kg) BW/d up to 800 lb. max 360 mg/hd/d	control
Monensin	cattle fed in confinement for slaughter	0.14-0.42 mg/lb BW max 360 mg/hd/d	prevention/control
	pasture cattle	0.14-0.42 mg/lb BW max 200 mg/hd/d	prevention/control
	mature reproducing beef cows	0.14-0.42 mg/lb BW max 200 mg/hd/d	prevention/control
	calves (excluding veal calves)	0.14-1.0 mg/lb BW max 200 mg/hd/d	prevention/control

*Always read and follow label directions
**Information from 2002 Feed Additive Compendium[14-20]

BW = body weight; T = ton; d = day; lb = pound; kg = kilogram; mg = milligram; hd = head

E. zuernii. Different studies have compared feeding rations containing 10g, 20g or 30g of monensin per ton of feed to young calves inoculated with either *E. bovis* or *E. zuernii.* Calves inoculated with *E. zuernii* exhibited less severe clinical signs compared to calves challenged with *E. bovis.* The investigators concluded that the low dose of 10g /ton (906 kg) of feed reduced infections but results were inconsistent. Effective control of coccidiosis was achieved when monensin was fed at 20 or 30g / ton of feed.[26,76] When formulating diets, the calf needs to consume 0.45 mg/lb (1 mg/kg) BW of monensin to prevent clinical breaks.

Canadian scientists fed monensin at 0.45 mg/lb BW/d (1mg/kg/d) to calves inoculated with *E. bovis* oocysts. Calves fed monensin for 10 days (days 10-20 of the experiment) shed fewer oocysts and had better daily gain than non-medicated control calves. When challenged with *E. zuernii,* cattle treated with monensin at the same dosage and for the same time period had less severe clinical signs and better weight gain than controls.[70]

In another study, six-week-old Holstein-Friesian male calves were fed diets containing 16.5g or 33g of monensin/metric ton of feed. Medicated feed was offered beginning three days prior to challenge with *E. bovis* oocysts. Non-medicated control calves shed large numbers of oocysts on days 19-28 and developed diarrhea. Calves fed monensin did not exhibit clinical signs of coccidiosis, and weight gain and feed efficiency were equal to or better than the controls.[49]

Treatment with coccidiostats following experimental challenge has also been evaluated. Four-week-old Holstein-Friesian calves were administered sporulated *E. bovis* oocysts. On days 10-20 post-inoculation, calves received monensin at 0.45 mg/lb (1.0 mg/kg) BW, amprolium at 4.55 mg/lb (10 mg/kg) or served as non-medicated controls. On post-infection days 13, 14 and 15, calves were given 40 mg of dexamethasone. Results indicated that both monensin and amprolium were effective in decreasing oocyst shedding and weight loss. Clinical signs in the amprolium-treated groups were less

severe than those in the monensin-treated calves.[32] A study with similar design used a challenge inoculum of *E. zuernii;* both drugs were effective in preventing clinical signs and in reducing oocysts.[73,74]

A novel method to administer monensin for long periods of time has been evaluated. A slow release intra-ruminal capsule containing monensin was administered to early weaned beef calves. Dosages delivered ranged from 10-60 mg/hd/d for 30 or 60 days. Results suggested that monensin released from the intra-ruminal device was effective for the one or two-month time period. However, calves that received a dose of 60 mg/d had a significant rise in fecal oocyst counts and experienced severe clinical disease following cessation of treatment. The investigators concluded that "it is possible that high doses of monensin prevented the infection from undergoing primary schizogony and becoming active while lower doses allowed slow development."[56]

In the US, lasalocid is an ionophore commonly used in feed "for improved feed efficiency and increased rate of weight gain in cattle fed in confinement for slaughter, and for control of coccidiosis caused by *Eimeria bovis* and *Eimeria zuernii*".[17] In dose titration studies, lasalocid was fed to 10-week-old Holstein steers 0.23 mg/lb (0.5 mg/kg), 0.34 mg/lb (0.75 mg/kg) and 0.45 mg/lb (1.0 mg/kg) BW that were inoculated with a mixture of *E. bovis* (88%) and *E. zuernii* (12%) sporulated oocysts. All three dosages of lasolocid were effective, but a dose of 0.45 mg/lb (1.0 mg/kg) BW was most effective in decreasing oocyst shedding, thus less contamination of the environment.[30]

Higher doses of lasalocid have been evaluated to determine the effect on efficacy and tolerance. Dosages up to 1.36 mg/lb (3.0 mg/kg) BW were administered to four-week-old Holstein-Friesian calves. The medicated feed was offered to the calves three days prior to challenge with a mixture of *E. bovis* (70%), *E. zuernii* (25%) and *E.* spp (5%) sporulated oocysts. Shedding of oocysts was virtually eliminated in calves that received 1.36 mg/lb (3.0 mg/kg) BW of lasalocid; the higher dose was tolerated with no observed ill effects.[27] In a similar study, calves weighing approximately 169 lb (77 kg) were challenged with *E. bovis* and *E. zuernii* oocysts. Lasalocid was first fed to cattle on the day of inoculation rather than three days pre-inoculation. Dosages of 0.23 mg/lb (0.5 mg/kg), 0.34 mg/lb (0.75 mg/kg), 0.45 mg/lb (1.0 mg/kg) or 1.36 mg/lb (3.0 mg/kg) BW were evaluated. The 0.34 mg/lb (0.75 mg/kg) dose was the lowest level of lasalocid that reduced infection with coccidia.[75]

Lasalocid and monensin have been individually incorporated into protein supplement blocks to deliver approximately 100 mg of drug daily to growing beef steers. Daily gain, feed efficiency and feed intake were the same for steers receiving medicated blocks as for steers provided non-medicated control blocks. Of significant benefit was the percentage of steers shedding oocysts, 4.2% for treated steers versus 41.5% for control steers.[6] Thus, less environmental contamination occurred when steers were offered medicated blocks. This type of delivery system is practical in many types of cattle operations.

Coccidial infections can be acquired early in life. Lasalocid was incorporated into the milk replacer and calf starter ration of three-to-seven-day-old Holstein bull calves. Lasalocid was in the milk replacer to provide 36.36 mg/lb (80 mg/kg) BW and added to the calf starter at either 1.36 mg/lb (3 mg/kg) or 20 mg/lb (44 mg/kg) dry matter (DM). Calves were challenged orally with *Eimeria* oocysts on day 10 of the trial. Calves fed lasalocid in milk replacer had higher gain, shed fewer oocysts and exhibited less severe clinical signs than calves fed lasalocid in the calf starter or the controls. The performance of the calves was similar when lasalocid was fed in milk replacer only, or in both milk replacer and calf starter.[60] Similar results were observed in an Australian trial using two-to-four-day-old Friesian bull calves. Treatment groups included lasalocid in milk at 0.45 mg/lb (1 mg/kg) BW per day, in calf starter at 23.86 mg/lb (52.5 mg/kg) DM, in both milk and calf starter, or no medication. Calves were challenged at approximately two weeks of age. No clinical disease was observed in calves that received medicated milk, medicated starter ration or both. Calves fed lasalocid in both milk and calf starter shed fewer oocysts in their feces than the other groups.[50]

In another study, young dairy calves were either fed calf starter containing lasalocid at 18.18 mg/lb (40mg/kg) of starter or non-medicated feed (controls). Calves fed lasalocid had less severe clinical signs and weight gain was improved by 50%. Additionally, control calves that were challenged had significantly reduced feed efficiency.[66]

In the US, decoquinate is labeled "for the prevention of coccidiosis in ruminating and nonruminating calves (including veal calves) and cattle caused by *Eimeria bovis* and *Eimeria zuernii*".[16] Decoquinate interferes with the mitochondrial electron transport mechanism of the coccidian organism.[46] To evaluate efficacy, six-week-old Holstein calves were administered decoquinate at 0.68 mg/lb (1.5 mg/kg) BW at various times after inoculation with sporulated *E. bovis* oocysts. Calves were necropsied 7, 12, 18, 22 and 28 days post-inoculation. Various stages of the coccidia were identified in control calves, depending on when they were necropsied, while only schizonts were present in calves treated for 7, 12, 18 or 22 days. No schizonts, gamonts or oocysts were identified in tissues from calves treated for 28 days. Results of the study indicated that "decoquinate apparently kills sporozoites or arrests development and release of merozoites from the schizonts when fed at 0.68 mg/lb (1.5mg/kg) of body weight in the feed."[25]

Challenge experiments have been performed comparing different dosages of decoquinate. Six-to-seven-week-old Holstein-Friesian calves were dosed with decoquinate at 0.045 to 0.36 mg/lb (0.1 to 0.8 mg/kg) BW in their daily grain ration. Three days after the medicated ration was first administered, calves were inoculated with either *E. bovis* oocysts or a mixture of *E. bovis* and *E. zuernii* oocysts. Efficacy was measured by suppression of oocyst production, bloody diarrhea and death. Dosages of 0.23 to 0.36 mg/lb (0.5 to 0.8 mg/kg) BW for 28 days effectively controlled experimental coccidiosis, while dosages of 0.16 mg/lb (0.36 mg/kg) BW or less were only partially effective.[51] In a similar trial, dosages ranging from 0.23 to 0.68 mg/lb (0.5 to 1.5 mg/kg) BW were used; dosing intervals ranged from continuous to every second or third day. All the calves, including controls, were challenged with sporulated *E. bovis* oocysts. A dose of 0.23 mg/lb (0.5 mg/kg) fed continuously was most effective in preventing infections. When calves were fed 0.45 to 0.68 mg/lb (1 or 1.5 mg/kg) of decoquinate every second day or 0.68 mg/lb (1.5 mg/kg) every third day, oocyst output was decreased and clinical signs were prevented.[23]

Scientists have investigated the effect of dexamethasone administration to calves challenged with coccidia oocysts and medicated with decoquinate. Calves treated with dexamethasone alone developed clinical disease, while dexamethasone treated calves fed decoquinate did not.[63] In another study, both control calves and those treated or not treated with dexamethasone had large numbers of the endogenous stages of the organism in the intestines after challenge with *E. zuernii*. Calves treated with decoquinate or decoquinate and dexamethasone had few of the endogenous stages of the organism when the intestines were examined microscopically. Results of this study suggest that administering dexamethasone to calves fed decoquinate did not increase the risk of coccidiosis.[24]

Studies have been performed to determine the length of time a coccidiostat should be administered. Twenty Holstein calves were premunized with 2,000 *Eimeria* spp oocysts/d for five days and fed a ration containing either lasalocid or decoquinate. Both drugs were included in the diet to provide approximately 0.55 mg/lb (1.2 mg/kg) BW. Both drugs were effective coccidiostats while being fed to the calves. Following withdrawal of the medicated feed, the calves were challenged. Oocysts were identified in fecal samples and some calves developed diarrhea. This suggests that coccidiostats should be fed continuously to prevent coccidiosis.[8]

Studies have been performed comparing the efficacy of decoquinate and lasalocid as coccidiostats under field conditions. In three studies, non-medicated control calves shed more oocysts than treated animals.[39,40,80] In two of the studies, little or no difference in performance was found between the non-medicated calves (controls) and calves that received either lasalocid, decoquinate or a combination of both drugs. The natural challenge in these studies was considered "low level".[40,80]

Salinomycin is a coccidiostat used in poultry, but is not labeled for use in cattle in the US.[20] In research trials, scientists administered this drug to calves at various times before or after challenge inoculation with sporulated *E. bovis* oocysts. Results from this study indicated that salinomycin administered daily at 0.23 mg/lb (0.5 mg/kg) BW or higher prevented experimental bovine coccidiosis. Oocyst production was markedly reduced in calves fed salinomycin at 0.91 mg/lb (2.0 mg/kg) BW (highest dose administered in this study). The drug was apparently active against the first asexual generation.[5]

Treatment. Amprolium is approved for treatment and control of coccidiosis in cattle and can be administered in feed or water. In the US, the product is labeled "as an aid in the prevention of coccidiosis caused by *Eimeria bovis* and *Eimeria zuernii* during periods of exposure or when experience indicates that coccidiosis is likely to be a hazard, and as an aid in treatment of coccidiosis caused by *Eimeria bovis* and *Eimeria zuernii*".[14] In a review of the efficacy and safety of the drug, the optimum dose for preventing coccidiosis was reported as 2.27 mg/lb (5 mg/kg) BW for 21 days, while the dose for treatment of clinical coccidiosis was 4.55 mg/lb (10 mg/kg) BW for five days.[54] Amprolium is a thiamine antagonist[44] and has been used at very high doses to experimentally induce polioencephalomalacia. Other researchers administered four times the recommended dose to calves every day for 12 weeks, and no adverse effects such as polioencephalomalacia were observed.[54]

Amprolium has been used to control coccidiosis in young dairy calves by adding it to milk. Holstein-Friesian calves approximately two weeks of age were inoculated with a mixture containing approximately 90% *E. bovis* oocysts. Calves were divided into different treatment groups: 1) 19.55, 16.36, or 10 mg/lb (43, 36 or 22 mg/kg) BW for 21 days (treatment initiated the day of inoculation), 2) 65 mg/lb (143 mg/kg) BW for five days (treatment initiated 13 days after inoculation), or 3) 65 mg/lb (143 mg/kg) BW of drug one time (treatment administered 13 days after inoculation). Untreated controls were included in the experiment. Results of the study indicated that amprolium was effective for control of experimentally induced coccidiosis at all dose regimens except when used at 65 mg/lb (143 mg/kg) BW one time 13 days after inoculation. Because there was no difference in outcome between the treatment group that received amprolium for five days beginning 13 days after incoculation and the group that received the drug

for 21 days, investigators concluded that amprolium affected the later stages of *E. bovis*.[36]

Other drugs have been investigated for control or treatment of coccidiosis. In a broad-scale-study, Holstein-Friesian calves were challenged with *Eimeria* spp sporulated oocysts and administered different compounds, including amprolium (1.5 gm PO), lincomycin (0.5 gm PO), sulfamethazine (1.5 gr/lb body weight IV), chloroquine sulfate (1.0 gm PO), and di-phenthane -70 (45 ml of a 15% suspension PO). Calves were assigned to groups, and within the assigned groups individual calves were administered the drugs at different time intervals. Non-treated, inoculated controls were included in the experiment. Calf survivability, severity of disease and oocyst production were measured. Amprolium administered 14-18 days after challenge prevented coccidiosis and decreased oocyst production; lincomycin suppressed oocyst production; treatment with sulfamethazine prevented morbidity and mortality and suppressed oocyte production; chloroquine sulfate was of little value; and diphenthane-70 given on days 15-18 decreased oocyst production compared to controls.[58]

Additional experimental studies evaluating the efficacy of various drugs, including lincomycin HCl, chlorhydroxy-quinolin, and sulfonamides, to control or treat coccidiosis have been reported in the literature. Several are not available in the US or are not labeled for the control, prevention or treatment of this disease. This paper will briefly mention these studies and refer the reader to the respective references for more details.

Lincomycin HCl is an antibiotic, but in the US it is not labeled for use in cattle.[18] However, it has been used experimentally for the prevention and control of bovine coccidiosis and found to be effective when administered either parenterally (IM) or orally in drinking water.[1,2,3] A report in the *Indian Veterinary Journal* showed that chlorhydroxy-guinolin administered orally for three days to naturally infected calves and adults reduced oocyst counts in the feces by an average of 87.3%.[64]

A commonly used feed additive containing chlortetracycline and sulfamethazine[a] has been evaluated for use as a coccidiostat. In the US, combination chlortetracycline-sulfamethazine is labeled for use in beef cattle "as an aid in maintenance of weight gains in the presence of respiratory disease such as shipping fever."[15] In a research trial, calves weighing from 260 to 450 lb (118 to 205 kg) were divided into non-medicated controls or treated groups and challenged with infective oocysts from a mixed culture. Treated calves received 350 mg chlortetracycline and 350 mg sulfamethazine daily for five days before challenge. Medicated animals passed fewer or no oocysts, exhibited minimal or no clinical signs of coccidiosis and had less inflammation and fewer schizonts than did control calves.[78]

Sulfonamides are common antimicrobial drugs used throughout the world, and exert their antimicrobial action by interfering with folic acid metabolism.[68] Sulfonamides have been evaluated for treatment and control of coccidiosis. Sulphadimidine was used to treat calves with coccidiosis. Treated calves received therapy at different times and dosages, and experienced a delay in infection as compared to controls.[57] In a different study using sulfaquinoxaline and sulfamerazine, both drugs were most effective for control of *E. bovis* in young calves (8-25 days of age) when treatment was instituted 13 to 17 days after inoculation. When drugs were administered at other times, efficacy was less than desirable. In addition, researchers reported that sulfaquinoxaline was less effective than sulfamerazine.[37] Sulfabromomethazine, a sulfonamide that persists in the blood stream for long periods, was effective against *E. bovis* when administered 13 - 17 days after inoculation.[35]

Eimeria alabamensis is an important cause of diarrhea and weight loss in grazing calves in Sweden and Great Britian.[48,77] In a Swedish study, investigators administered long-acting boluses containing baquiloprim (1.6 g) and sulphadimidine (14.4 g) to 6-16-month-old calves grazing contaminated pastures. Treatment with baquiloprim and sulphadimidine prevented clinical coccidiosis and reduced the weight loss as compared to control calves.[77]

Toltrazuril, an anti-coccidial compound, and sulfonamides were evaluated as treatments for coccidiosis in an Italian study. Calves were infected with massive amounts of *E. bovis* and *E. zuernii;* treated calves were considered clinically cured by the second or third day of treatment with toltrazuril.[10] This compound is being investigated in Europe and has potential to be another treatment option for cattle.

Other Control Options. Some cattle operations incorporate manure into rations as a means of waste disposal. Logically, transmission of coccidia is a concern. A study was conducted to investigate the survival time of coccidia in ensiled bovine manure-containing diets. In this trial, sporulation of oocysts was considered synonymous with oocyst survival. Oocysts did not sporulate in properly ensiled diets. The degree of inhibition is dependent upon the ensiling temperature. A temperature range of 77 to 95°F (25 to 35°C) was most effective when evaluated during a 3-week storage period.[13]

Researchers have investigated whether growth promoting implants affect the response to experimental challenge with *E. bovis*. Twenty-seven Holstein bull calves were administered an estradiol-progesterone (EP) growth promoting implant. Calves challenged with *E. bovis* had significantly lower feed intake and weight gain than noninfected calves. Implanted calves inoculated with *E. bovis* had higher feed intake and gained more

weight than inoculated, nonimplanted calves. The inoculated-EP implanted calves also had diarrhea for fewer days and lower mean rectal temperature than inoculated nonimplanted calves. Results of this study suggested that EP implants had a sparing effect against some of the debilitating effects of *E. bovis*.[38]

Commercial vaccines are available for control of coccidiosis in poultry. Currently, no anti-coccidial vaccines are available for cattle.[4]

Conclusions

Management programs that minimize stress, reduce environmental contamination and improve the overall health of each individual animal through preventive health programs help minimize the impact of coccididiosis. Several products are routinely used for the control, prevention and treatment of bovine coccidiosis in the US, including monensin, lasalocid, decoquinate and amprolium. Sulfaquinoxaline and sulfamethazine are labeled for the treatment of coccidiosis, however, these drugs are used less frequently for this purpose than in the past. Approved products are efficacious. If animals exhibit clinical signs of coccidiosis, options for treatment are available, depending upon cost-benefit, facilities and labor availability. Veterinarians and producers should be familiar with this disease and take appropriate measures for prevention and treatment.

Footnote

[a]Aureo-S-700®, Alpharma Inc., Fort Lee, NJ 07024

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