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Effects of lasalocid on coccidial infection and growth in young dairy calves

Glendon Douglas Sinks

To the Graduate Council:

I am submitting herewith a thesis written by Glendon Douglas Sinks entitled "Effects of lasalocid on coccidial infection and growth in young dairy calves." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Animal Science.

James D. Quigley III, Major Professor

We have read this thesis and recommend its acceptance:

Craig R. Reinemeyer, Richard N. Heitmann

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

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Date November 29, 1990

EFFECTS OF LASALOCID ON COCCIDIAL INFECTION AND GROWTH IN YOUNG DAIRY CALVES

A Thesis

Presented for the

Master of Science

Degree

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Glendon Douglas Sinks

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ABSTRACT

Effects of lasalocid on coccidial infection and growth were examined using 16 Holstein bull calves. Animals were assigned randomly to a 2 x 2 factorial arrangement of starter containing 0 or 40 ppm lasalocid beginning at 3 d of age (SE = .46) and single oral infection with 0 or 30,000 oocysts (*Eimeria bovis*) at 28 d. Pelleted calf starter was fed for *ad libitum* consumption from d 1; milk replacer was fed at 3.6 kg/d to 28 d. Average daily gain, dry matter intake, and body weight were increased in calves fed lasalocid and decreased in calves dosed with coccidia. Addition of lasalocid to the feed improved gains by 8% in uninfected calves and by 50% in infected calves. Fecal oocyst numbers were increased by administration of coccidia and decreased by lasalocid. Calves infected with coccidia had greater fecal scores, indicating abnormal or diarrheic feces. Respiration, rectal temperature, hematocrit and serum sodium and potassium concentrations were unaffected by treatment. Data indicated that lasalocid minimized effects of coccidial challenge and increased growth in this study.

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CHAPTER I

INTRODUCTION

Coccidiosis in young calves is a source of major economic loss in many parts of the United States. In 1984, it was estimated that 62 million dollars were lost annually due to this disease (53). Coccidial infection is ubiquitous, and morbidity approaches 53% (17) Calves less than a year old are the most severely affected (15), and mortality is as high as 24% in some severe outbreaks (17). Effects of coccidiosis may be attenuated by the natural resistance of the calf (17,41), and transmission hampered by an unfavorable ecosystem for the protozoa.

Intestinal coccidiosis in cattle is caused by protozoan parasites of the genus Eimeria (21). This genus is characterized by an oocyst with four sporocysts, each with two sporozoites (15). Coccidia are transmitted by ingestion of sporulated oocysts in contaminated water and feed and soiled pastures, or by hair licking. After infection, calves may develop diarrhea with or without blood present, dehydration, anorexia, anemia, weakness, and loss of weight (39).

Inclusion of ionophores such as lasalocid in early weaning programs improved growth and researchers (19,51) have reported control and/or prevention of coccidiosis in

young (4 to 12 wk of age) calves when lasalocid was fed at .5, .75, 1.0, and 3.0 mg/kg of body weight. There are no data available, however, that evaluate lasalocid on growth and prevention/control of coccidiosis in early weaned dairy calves (4 wk of age). Additive or synergistic effects of lasalocid in growth responses of young calves infected with coccidia would be of economic benefit to dairy producers raising replacement animals. Therefore, the objectives of the current study were to evaluate efficacy of lasalocid on growth and coccidial challenge in young dairy calves.

CHAPTER II

LITERATURE REVIEW

Coccidiosis of Cattle

Coccidiosis, caused by protozoa of the genus Eimeria, results in health and economic problems to several classes of livestock. The disease reduces feed consumption, body weight, and feed efficiency and may cause mortality of 24% in some cases (17). An estimated 77 million young cattle in the United States could be infected by coccidia during the first year of their life. Of these, 4 million will be treated for coccidiosis, and 80,000 cattle could die from the disease (17). Annual economic losses due to coccidiosis have been estimated at $62 million (53).

Coccidiosis commonly affects young cattle up to 2 years of age. Animals that are housed in proximity are more likely to contract the disease. Therefore, feedlot and dairy cattle are most susceptible. Coccidiosis in feedlot cattle is associated with stress caused by shipping, changes in ration and in weather, and overcrowding (15). Stress caused by weaning makes dairy calves very susceptible to coccidiosis.

Etiology

All domestic animal species are susceptible to

coccidial infections. Although coccidia are host-specific, each host may be infected with several species of coccidia at the same time. At least 13 different coccidial species are known to infect cattle in the United States (15), but not all are pathogenic. The two most pathogenic species are Eimeria bovis and Eimeria zuernii (15). Incubation periods for E. zuernii and E. bovis are usually 15 to 20 days (21). Immunity to coccidiosis perists only 3 to 4 months, and reinfection may occur in the absence of continuous challenge (17).

Life Cycle

A coccidian life cycle (Figure I) is initiated when a host ingests sporulated oocysts containing 8 banana or cigar-shaped sporozoites (21). In the host cell, sporozoites grow, undergo nuclear division, and meronts are formed (15). This kind of multiple internal fission is called schizogony (21). With the growth of the meronts, nuclear fission continues until merozoites (small ameboid sporozoan trophozoites produced by schizogony that are capable of initiating a new sexual or asexual cycle of development) develop within the schizonts. Each merozoite contains one nucleus. After the merozoites mature, they escape from the schizont, invade a new host cell, and begin another generation of schizogony. Schizogony continues for

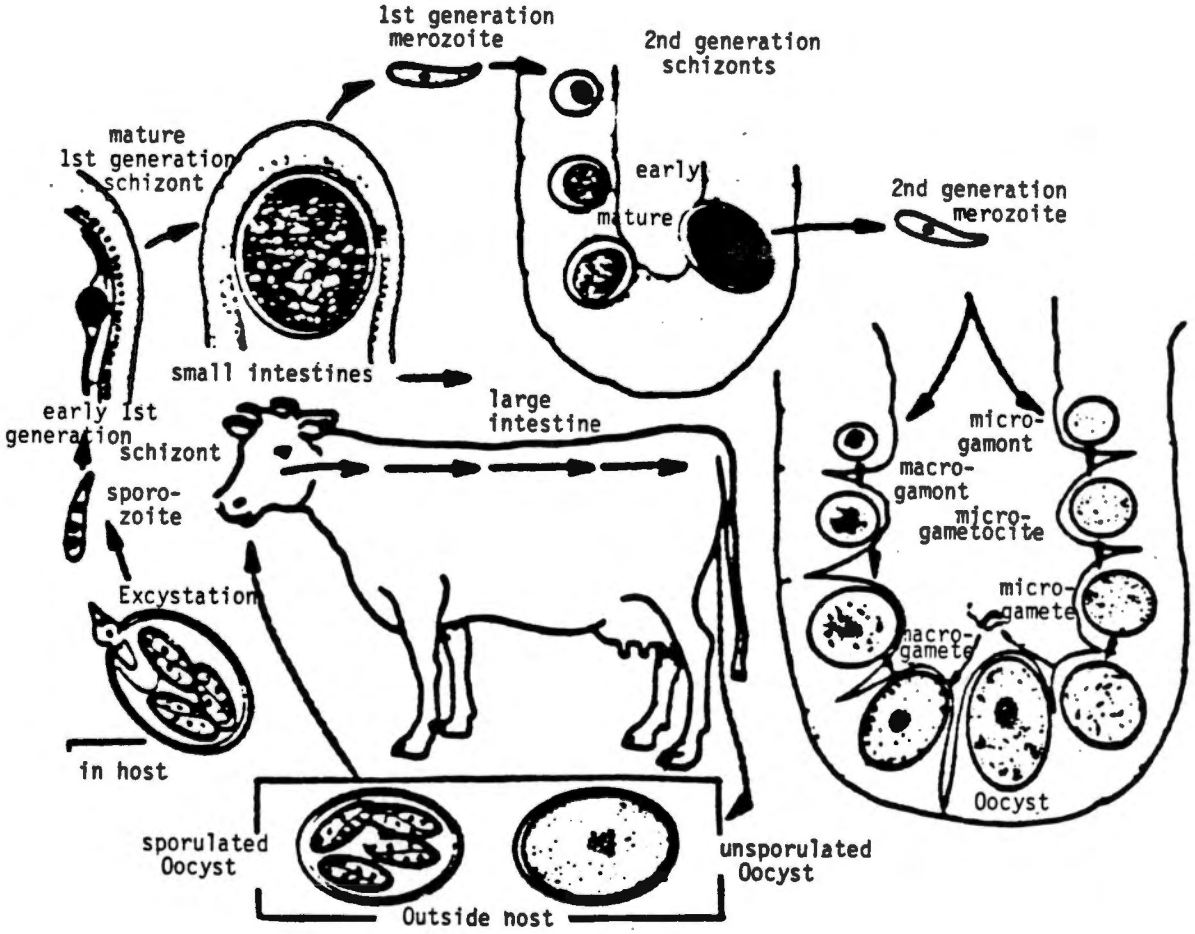

Figure I. Life cycle of Eimeria bovis. (15)

2 or 3 generations (21). The second and third generations of schizogony cause the clinical signs associated with coccidiosis (15). Each coccidial species has a different number of generations.

During the last schizogonous generation, a merozoite invades a new host cell and becomes either a macrogamont (female sex cell) or a microgamont (male sex cell) (21). The nucleus of the microgamete elongates, cytoplasm forms around it, and two flagella develop on each organism. The mature microgamont contains numerous flagellated microgametes. Maturation of the macrogamont does not include nuclear division (15). After macrogamonts mature, a macrogamete is formed. Mature microgamonts rupture and release microgametes. Microgametes find and invade host cells containing macrogametes. Fertilization occurs when a microgamete comes in contact with a macrogamete, forming a zygote (21). An oocyst is formed from small bodies that had developed in the cytoplasm of the macrogamete that produce a wall around the zygote (15). Host cells containing oocysts rupture, and oocysts pass from the host in feces, and are released into the environment (21).

At the proper temperature, Eimeria oocysts sporulate; i.e. the cytoplasm divides to form four sporocysts, each containing two sporozoites (15). Host species can only be infected by ingesting sporulated oocysts.

Coccidia usually infect epithelial cells of the gut mucosa during their developmental stage (15), but there are exceptions to this. For example, large, first-generation schizonts of E. bovis and E. zuernii can be found in endothelial cells of the central lacteals in the small intestine (27), and in connective tissue cells of the lamina propria (50), respectively.

Life cycles of E. bovis and E. zuernii are somewhat similar (37). The life cycle of E. bovis has been studied more than any other bovine coccidia (15). First-generation schizonts are in the central lacteals of the villi in the ileum of the small intestine (45). First-generation schizonts are about 300 μm in diameter, contain about 120,000 merozoites and mature in approximately 15 days (15). Second-generation schizonts occur in epithelial cells of the crypts in the cecum and upper colon (45). Second-generation schizonts are about 10 μm, contain about 30 merozoites and mature in approximately 2 days (15). Second-generation schizonts of E. bovis are difficult to find in the infected gut. Macrogamonts and microgamonts are found in epithelial cells of the crypts of the cecum and upper colon (25). The prepatent period for E. bovis is 17-21 days after ingestion of a sporulated oocyst (21).

First-generation schizonts of E. zuernii are about 250 μm in diameter, contain numerous merozoites and mature in

approximately 16 days (15). These schizonts are located in connective tissue cells of the lamina propria in the lower part of the small intestine (50). Second-generation schizonts are located in epithelial cells of the cecum and colon (45). Second generation schizonts are approximately 15 by 19 μm, and contain about 30 merozoites (15). Second-generation schizonts are usually numerous and are easily found in the infected gut. Macrogamont and microgamonts occur in the epithelial cells of the cecum and colon (50). Oocysts of E. zuernii are first seen in the feces of infected calves on the sixteenth or seventeenth day after ingestion of sporulated oocysts (21).

Transmission

Coccidiosis is transmitted by ingestion of sporulated oocysts. Infection is acquired from contaminated feed, water, and soiled pastures, or by licking a contaminated hair coat (21).

Clinical Signs

Coccidiosis is a contagious disease that causes anorexia, loss of weight, and hemorrhagic and mucoid diarrhea (21). In severe cases, feces are liquid, bloody and may contain strands of intestinal muscosa (15). Animals may become emaciated, dehydrated, weak, and listless. Rectal prolapse may result from straining without defecation (15).

The clinical course of coccidiosis ranges from 4 to 14 days, and the mortality rate may be as high as 24% in severe outbreaks (17). Death is primarily a result of diarrhea, which causes a loss of electrolytes and dehydration; however, hemorrhaging or secondary complications such as opportunistic infections may contribute to mortality. Animals that recover from severe infections may suffer permanent production losses (15).

Clinical and Subclinical Coccidiosis

Clinical and subclinical coccidiosis cause economic and health problems for cattle producers. Both forms result in a decline of herd condition and, if left untreated, mortality can occur. Clinical coccidiosis pertains to responses elicited by the course of the disease, i.e. the symptoms. It has been estimated that 5% of infected animals show clinical signs of coccidiosis (41). Subclinical coccidiosis, in constrast, refers to a period before appearance of typical symptoms of the disease, or infected animals that do not show signs of a clinical infection. The remaining 95% suffer from subclinical coccidiosis, which can result in increased economic loss (41). Since subclinical coccidial infections do not exhibit signs of the disease, animals could be infected without cattle producers knowledge. Losses due to clinical and subclinical

coccidiosis result from a decrease in absorption of nutrients due to damage to the intestinal lining (41).

Dehydration

Mortality from coccidiosis is usually associated with severe diarrhea, which causes loss of electrolytes and dehydration. In one study, calves with diarrhea lost 8 and 18 times more sodium and potassium respectively, than normal calves (6).

Coccidia destroy intestinal cells, which results in loss of blood and other fluids into the small intestine (45). During the coccidian life cycle, second generation schizonts mature within the intestinal cells and disrupt the cytoplasm (45). This disruption causes abnormal absorptive function of the cecal epithelia. Eimeria bovis causes nonabsorption of fluid (16) in the central lacteals, and Eimeria zuernii causes malabsorption of sodium ions (35) in the connective tissue. Blood and other fluid then pass in the feces, which are usually watery. When schizonts are mature, intestinal cells are sloughed from membranes and either leave scattered epithelial cells to cover the lamina propria or expose lamina propria with engorged capillaries (45). If these exposed capillaries are severely damaged, blood and plasma may be lost.

Electrolyte balance is disturbed by severe coccidiosis (16). Toxic products from the coccidia, cellular breakdown

and secondary bacterial or viral infections contribute to denaturation of proteins (16). The process of denaturing proteins results in increased osmotic pressure, drawing excess water from extracellular resources (16). Once these resources are depleted, electrolytes such as sodium and potassium are maintained through the sacrifice of intracellular resources (16). Dehydration leads to depletion of extracellular sodium if the animal has severe coccidiosis. Potassium is released into extracellular fluid when the permeability of cellular membranes is increased. High serum potassium values found by Roy et al., (48) were the result of release of cellular potassium combined with a decline of extracellular fluid. Extracellular potassium eventually rises to lethal levels, and mortality will occur as a result of a cardiac arrest (48).

Diagnosis

Diagnosis of coccidiosis should be based on the occurrence of signs, the clinical history of individual animals and herds, and the presence of oocysts, E. bovis and/or E. zuernii, in the feces (15). Most bovine coccidia can be identified to species by the morphologic characteristics of unsporulated oocysts (15). Characteristics such as size and shape of oocyst, color and texture of oocyst wall, and presence or absence of a micropyle (Figure II) are most often used to identify an

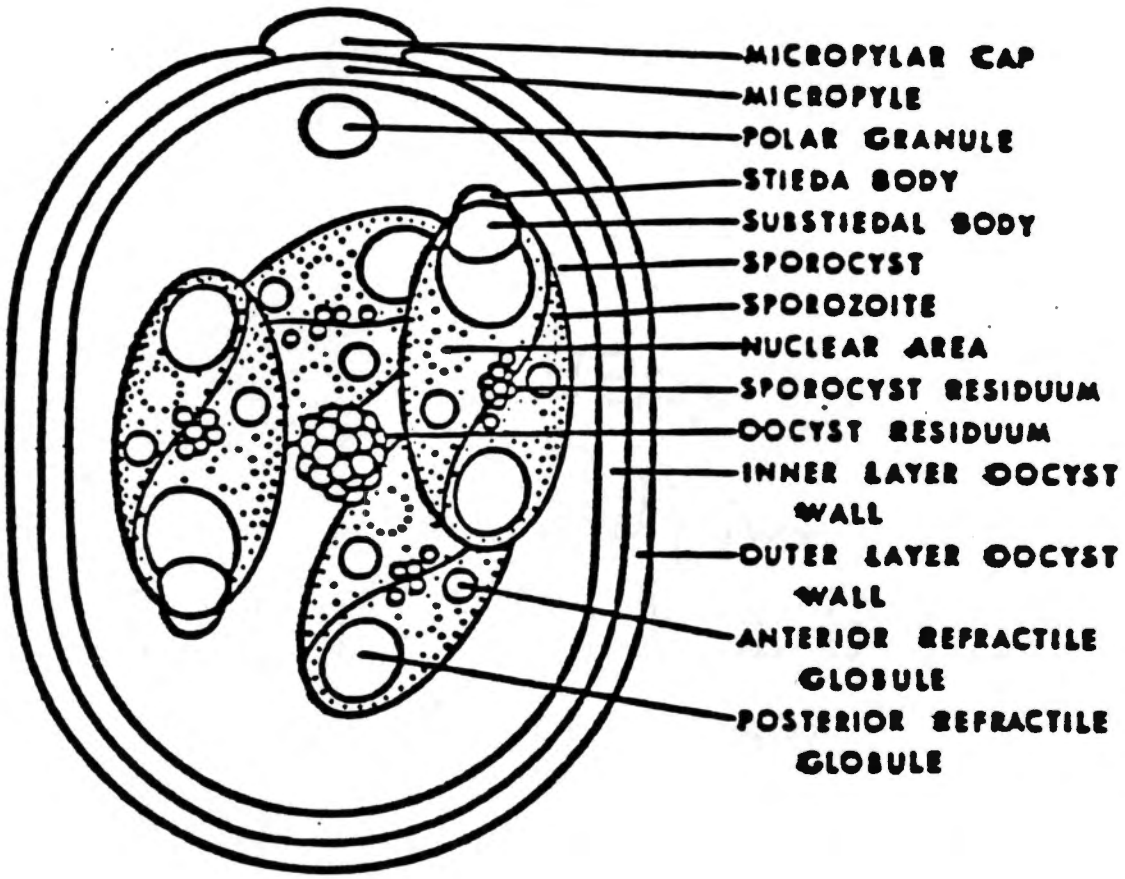

Figure II. Major components of a sporulated Eimeria bovis oocyst. (15)

an unsporulated oocyst. Large numbers of oocysts in the feces should not be the only basis for diagnosis. Diarrhea may begin a few days prior to oocyst discharge, therefore, several consecutive daily fecal examinations may be required to detect infected animals (41).

Cattle producers and veterinarians have problems diagnosing coccidiosis because clinical signs are associated with the late portion of the early sexual phase (41). Passage of oocysts follows signs of coccidiosis, therefore, if there are large numbers of oocysts in the feces, coccidia probably have already completed their life cycle (15). If treatment is given at this time, and secondary bacterial infections are controlled, animals will probably recover (21).

Treatment

Although coccidiosis is considered a disease of young animals, older animals are frequently infected with Eimeria. The severity of clinical coccidiosis depends on the number of sporulated oocysts ingested and the general health of the infected host (15). A objective of control could thus be reducing the number of oocysts available for ingestion. However, no minimum infective dose for coccidia has been established (41). Proper sanitation and good animal husbandry practices are important in preventing coccidiosis.

Water and feed troughs should be constructed and located to reduce fecal contamination. Newborn dairy calves should be provided with clean, dry quarters when removed from the dam (15).

Treatments in the past included sulfonamides in the drinking water and amprolium in the feed. Recently, polyether antibiotics, such as lasalocid and monensin, originally developed as coccidiostats for poultry, have been effective in preventing coccidiosis in cattle.

A major difficulty in treating clinical coccidiosis is that signs of the disease do not appear until the life cycle is almost complete. By this time, the gut may be severely damaged. Most anticoccidial drugs are only effective during early stages of a coccidian life cycle. Thus, the difficulty in treating coccidiosis is that by the time signs appear, parasites have already passed through the stage in which anticoccidial drugs are most effective. Infected animals often recover without treatment due to acquired resistance to the disease (45). However, treatment with anticoccidial drugs should be administered at the earliest clinical signs because it may reduce the severity of the disease and decrease mortality. Antibiotics should also be administered to reduce secondary infections. Electrolyte solutions and fluids should be administered to control

dehydration. During treatment, animals should be isolated in a clean environment to prevent further contamination.

Treatment with sulfamethazine at a dosage of 0.15 g/lb body weight every other day for a period of 10 to 18 days after infection or a single treatment of the drug at 1.5 g/lb weight effectively controls coccidiosis (23). Sulfamethazine given at a dosage of 21.5 mg/kg on days 12 and 14 after infection reduced occurrences of dysentery and length of duration of diarrhea (24).

During recent years, numerous drugs that were developed as anticoccidials for use in the poultry industry have been effective as anticoccidials in cattle. Amprolium, a thiamine antagonist, was the first new drug to be used in cattle as a coccidiostat. Amprolium is effective against bovine coccidiosis due to Eimeria bovis at rates of 143, 36, or 22 mg/kg body weight if given daily for 21 days (26). Amprolium is recommended orally at a dosage of 10 mg/kg of body weight for treatment of active infections with Eimeria bovis and Eimeria zuernii (21). A dosage of 5 mg/kg of body weight daily for 21 days is used as an aid in preventing coccidiosis caused by Eimeria bovis and Eimeria zuernii during periods of exposure to sporulated oocysts (21).

Decoquinate aids in controlling coccidiosis caused by Eimeria bovis and Eimeria zuernii in calves and older cattle. Decoquinate fed at 0.5 mg/kg body weight for at

least 28 days during periods of exposure to sporulated oocysts aids in contolling the disease (21). For decoquinate to be effective for cattle, it must be fed to provide 22.7 mg/100 lb. of body weight/head/day (41).

Recently, ionophorous antibiotics such as monensin and lasalocid have proven to be effective against bovine coccidia. Despite their anticoccidial properties, monensin and lasalocid are labeled as feed additives to increase feed efficiency and rate of gain in cattle.

Monensin fed at 1 mg/kg body weight/day throughout the incubatation period of an experimental infection of Eimeria bovis prevented development of clinical signs of coccidiosis (18). However, monensin did not have total coccidiostatic effects. Monensin fed at 16.5 or 33 g/metric ton of feed for 31 days prevented development of clinical signs of coccidiosis (38).

Efficacy of Lasalocid Against Coccidiosis

Efficacy of lasalocid against coccidiosis has not received significant attention in cattle. Incidence of coccidiosis in cattle is highest in calves up to 2 years old (15). Therefore, young calves were and are being used to test effects of lasalocid against coccidiosis. Consequences of coccidiosis in cattle in the United States are not known,

however, incidence of the disease is very high, and direct and indirect costs are substantial (17,53).

Oocyst Discharge

Studies have evaluated lasalocid as a coccidiostat using different concentrations in the feed (12,19,20,51). However, the recommended level of lasalocid to control coccidiosis is 1 mg/kg of body weight (41).

Lasalocid at 1 to 3 mg/kg of body weight was highly effective against experimentally infected calves (19). The hypothesis that a larger inoculum of oocysts would require a higher concentration of lasalocid was investagated by (20,51). Lasalocid at .75 to 1.0 mg/kg body weight was equally effective against coccidiosis despite the level of infection (20,51). It has been hypothesized that calves that were premunized with coccidia would be resistant to coccidiosis without treatment. Premunization alone did not prevent coccidiosis after animals were exposed to large numbers of oocysts (12).

Due to stress of weaning for dairy calves and wet conditions in feedlots, signs of naturally occurring coccidiosis are very common. Lasalocid at 1 mg/kg of body weight has been shown to reduce fecal oocyst shedding in calves with natural exposure to coccidiosis (30). Lasalocid at 33 to 44 mg/kg of the diet was highly effective against

naturally occurring coccidiosis in feedlot cattle (31,32,36).

Efficacy of Lasalocid on Growth

Gain responses are generally related to changes in voluntary feed intake, therefore, large decreases in feed consumption are not often associated with increased gains. Ionophores such as lasalocid and monensin have been shown to change fermentation patterns in the rumen (3,47). It was shown by Chen and Wolin (11) that lasalocid and monensin inhibited acetate and butyrate producing rumen bacteria. As a result of these changes in the rumen (11) discovered that methane production was reduced, due to a decline in the growth of methane producers. Propionic acid fermentation is more energy efficient and as a result decreases the amount of lost methane related to the output of acetic and butyric acids (33,54).

Inclusion of ionophores in diets of cattle increase rumen propionate production (43). Ionophores fed to feedlot cattle have been shown to reduce the rumen acetate to propionate ratio and enhance feed efficiency by a decline in feed intake (3,8,28,46). Reduced feed consumption causes either no change or an increase in growth rates when feedlot cattle are fed lasalocid (9,10,13). Ruminal fermentation may be limited by inefficiency of acetic and butyric acid

fermentations (49). An increase in feed efficiency is usually related to changes in rumen fermentation. Consequently, inclusion of ionophores in the diet may result in lower intakes and similar gains, or similar intakes and higher gains.

Body Weight Changes

Average daily gains (ADG) of dairy calves infected with coccidia were increased by lasalocid when fed at .75, 1, and 3 mg/kg body weight compared to infected, unmedicated calves and those fed lasalocid at .05 mg/kg (19,51).

An increase in ADG ranging from 4 to 12% has been documented with steers fed lasalocid on finishing diets (5,10,36). Lasalocid improved gains of steers fed a 75% corn silage ration by 19% (31). Roughage diets supporting low to moderate rates of gain had a higher response. Feeding lasalocid at 135 mg/day increased gains of steers fed alfalfa cubes by 38% (52). Calves weighing approximately 187 kg fed corn silage with different supplements had increased gain responses due to lasalocid (32).

Lasalocid usually increases ADG more than monensin (5,9,20) although similar gains have been reported (10,13). In addition, feed to gain ratio was lower in lasalocid than monensin fed cattle (9,13). Ionophore compounds are usually not palatable, therefore, levels of lasalocid should be

selected that do not impair feed consumption (2). Consequences of reduced feed consumption on animal performance are generally undesirable.

Feed Consumption

Reduced feed consumption in unmedicated dairy calves oocurs 14 to 18 days after inoculation of coccidia and is associated with high oocyst discharge (19,51). If a low dosage of lasalocid is given as a treatment, intake will be slightly lower than if a higher dosage was given. Data on reduced feed consumption when monensin or lasalocid were fed is contradictory (10,46,52). However, all ionophores are usually not palatable, therefore they need to be given at a concentration which affects intake the least (2).

CHAPTER III

MATERIALS AND METHODS

Animal Assignments and Management

Sixteen Holstein bull calves were assigned randomly at birth to a 2 x 2 factorial arrangement of calf starter containing 0 (L^-) or 40 ppm (L^+) lasalocid, and oral infection with 0 (C^-) or 30,000 (C^+) oocysts (Eimeria bovis). Calves were left with dam to 3 d of age when they were moved to individual pens in an open sided barn bedded with sawdust. They were fed 1.8 kg of milk replacer twice daily (0800 and 1600 h) from nipple-bottles until weaning at 28 d. Beginning at 3 d of age (SE = .46) and continuing for 12 wk calf starter containing 0 or 40 ppm lasalocid was offered for ad libitum consumption once daily (0830 h). Refused calf starter was weighed back daily and reported weekly. Fresh water was available at all times.

Single oral inoculum of 0 or 30,000 oocysts of Eimeria bovis was administered 28 d after initiation of the study. A dosage of 300,000 oocysts was originally planned as in (51); however, because coccidia obtained were particularly virulent (Dr. C. A. Speer; personal communication), the number of oocysts was reduced to 30,000.

Experimental calf starters were commercially prepared, (Table I) containing 17% CP (DM basis) and 75% TDN (DM

Table I

Formulation of commercial experimental starter[1].

Item, %	Calf Starter
Corn, ground	20.0
Soybean meal, 48%	7.5
Soybean hulls	20.0
Wheat midds	25.0
Cottonseed hulls	7.5
Cottonseed meal	5.8
Alfalfa meal	5.0
Molasses	3.0
Bentonite/limestone	3.1
Vitamins/minerals/additives	3.1
CP[2]	17.0
TDN	74.0
ADF	7.5

[1]TCR I commercial calf starter. Ingredients expressed on an as fed basis.

[2]Nutrients expressed as a percent of dry matter.

basis). Although concentrations were slightly lower than those recommended for calf starter by NRC (CP, 18% of DM; TDN, 80% of DM) (42), starters contained high fiber by-products (ADF = 7.5% of DM) to stimulate ruminal development. Formulation of L^+ to contain 40 ppm lasalocid was at the expense of corn. Remaining ingredients were similar to control calf starter.

Sampling and Analysis

Starters were sampled once weekly and stored (-20°C) prior to monthly compositing and analysis for DM, CP and ether extract (1), NDF (22), Ca^{2+}, K^+, Mg^{2+}, Na^+ (atomic absorption spectrophotometry), and P (29).

Body weights were measured at birth, on d 0 (first d of the study), and every 7 d thereafter.

Fecal scores were determined daily at a.m. feeding to indicate the occurrence of scours. Feces were scored by visual observation, using a scale of:

1 = normal feces; firm hard appearance

2 = slightly loose feces; soft, somewhat liquid

3 = moderate scours; feces do not hold firm; are moderately thin and watery

4 = severe scours; thin, watery; may contain blood

A sample of feces was collected from each calf once weekly and fecal oocysts were counted as in (51).

Respiration rate (breaths/min) and rectal temperature were determined daily, at a.m. feeding, from d 28 to the end of the study. Approximately 10 ml of blood was collected once weekly by jugular venipuncture into evacuated tubes containing no anticoagulant. Blood was collected approximately 5.5 h after a.m. feeding. Serum was removed after coagulation and stored (-20°C) prior to analysis of Na^+ and K^+ concentrations by atomic absorption spectrophotometry. A second sample of jugular blood was collected into evacuated tubes containing 45 USP units of lithium heparin as anticoagulant for measurement of packed cell volume.

Statistical

Growth, intake, health, blood, and coccidial data were analyzed as a split-plot design using a generalized linear mixed models algorithm (7). The model used was:

$Y_{ijk} = \mu + T_i + A_{(i)j} + P_k + (TP)_{ik} + e_{(ijk)}$, where:

Y_{ijk}= dependent variable,

μ = overall mean,

T_i = effect of ith treatment,

$A_{(i)j}$ = effect of jth animal nested within ith treatment,

P_k = effect of kth period,

$(TP)_{ik}$ = effect of treatment x period interaction,

$e_{(ijk)}$ = residual.

Term $A_{(i)j}$ was used as error term to test differences due to treatment. Treatments and periods were defined as fixed effects, and animal was defined as a random effect. Fecal oocyst counts were transformed to logarithms prior to analysis.

Initial BW was analyzed as a completely randomized design by ANOVA. Final BW was analyzed by analysis of covariance as a completely randomized design, using initial BW as covariable.

Included in analyses were single degree of freedom contrasts evaluating lasalocid (L^- vs. L^+), coccidial infection (C^- vs. C^+) and interaction. Probability of .05 was used unless otherwise noted.

CHAPTER IV

RESULTS AND DISCUSSION

Chemical composition of experimental starters is in Table II. Calf starter averaged 19.4% CP, which was higher than formulated. Calf starter averaged 35.6% NDF, due to inclusion of cottonseed and soybean hulls at 7.5 and 20% of the diet, respectively.

Calves on treatment C^-L^- and C^+L^+ weighed less initially than other calves (Table III; $P<.01$), even though animals were randomized to treatments. To test if initial BW affected measures of performance significantly, data were reanalyzed by analysis of covariance using the model described above, but with initial BW included as a covariable. No measure of performance except serum K^+ was significantly affected by initial BW, $P>.05$; therefore, unadjusted means are presented.

Least squares means of final BW (Table III) were significantly affected by treatment. These values, covariately adjusted for initial BW, indicate that calves uninfected with coccidia were heavier than those infected. Calves on treatment C^+L^- tended to weigh less than other calves, although the interaction between lasalocid and coccidia was not significant ($P=.11$).

Average daily gains (Table III) were increased when

Table II

Chemical composition of feeds[1].

Item	Control	Lasalocid
DM, %	89.4	90.3
	-------(% of DM)-------	
CP	19.2	19.5
NDF	35.7	35.4
Ether Extract	.38	.48
Ca	.89	.92
P	.64	.64
Mg	.32	.32
K	1.21	1.21
Na	.41	.34

[1]Mean of 5 observations.

Table III

Least squares means of body weight, gain, and intake in calves on experimental treatment.

Item	Treatments[1]				SE	Contrasts[2]		
	C^+L^+	C^+L^-	C^-L^+	C^-L^-		1	2	3
Body wt, kg								
Initial	38.2	41.3	44.2	35.9	1.7	NS	NS	**
Final[3]	103.8	80.8	104.7	105.7	4.9	NS	*	NS
Daily gain	.75	.50	.81	.75	.11	*	*	NS
Gain to Feed[4]	.38	.27	.34	.44	.05	NS	*	**
Intake, kg/d								
DM[4]	1.8	1.4	2.1	1.7	.27	**	NS	NS
Milk	.13	.13	.13	.13	.06	NS	NS	NS
Grain	1.7	1.3	1.9	1.5	.31	**	NS	NS
CP[4]	.47	.38	.51	.43	.04	**	NS	NS
NDF[4]	.78	.64	.85	.72	.08	**	NS	NS
TDN[4,5]	1.5	1.2	1.7	1.4	.20	**	NS	NS

[1]Treatment = oral infection with 0 (C^-) or 30,000 (C^+) coccidialoocysts; and feeding 0 (L^-) or 40 (L^+) ppm lasalocid incalf starter.

[2]Contrasts: 1= lasalocid vs no lasalocid; 2= coccidia vs nococcidia; 3= interaction; NS=$P>.05$; *=$P<.05$; **=$P<.01$.

[3]Covariately adjusted for initial BW.

[4]Significant week*treatment interaction ($P<.05$).

[5]Calculated value of grain (TDN=80% of DM) + Milk(TDN=129% of DM).

calves were fed L^+, and decreased when calves were infected with C^+. Addition of L^+ to the feed improved gains by 8% in uninfected calves and by 50% in infected calves. Rates of gain were somewhat more than (34,40,44) due to the influence of L^+. Calves on C^+L^- treatment resumed a normal rate of BW gain (Figure III) and DMI (Figure IV) by wk 9, suggesting that these calves developed resistance to the effects of coccidiosis. Naturally occurring resistance to coccidia has been documented (17,41). Body weight gain was reduced only when fecal oocysts exceeded approximately 1,000/g feces, during wk 7 and 8. At no time did fecal oocysts reach this level in calves on C^+L^+ treatment; therefore BW gain was unaffected by coccidial challenge in these calves. Calves that were not infected with coccidia never discharged oocysts, indicating no cross-contamination between treatment groups. It should be noted that calves were housed in a barn previously uninhabited by other cattle, therefore, oocyst counts of zero were not unexpected.

Lasalocid fed at .5, 1, and 3 mg/kg of BW has ameliorated growth depression caused by coccidia in calves (19,30,51). Lasalocid decreased clinical signs of coccidiosis in feedlot studies (31,32,36). Thus, it appears that young calves may benefit from both improvement in rates of gain due to changes in ruminal fermentation (20) as well as improvements due to control of coccidia.

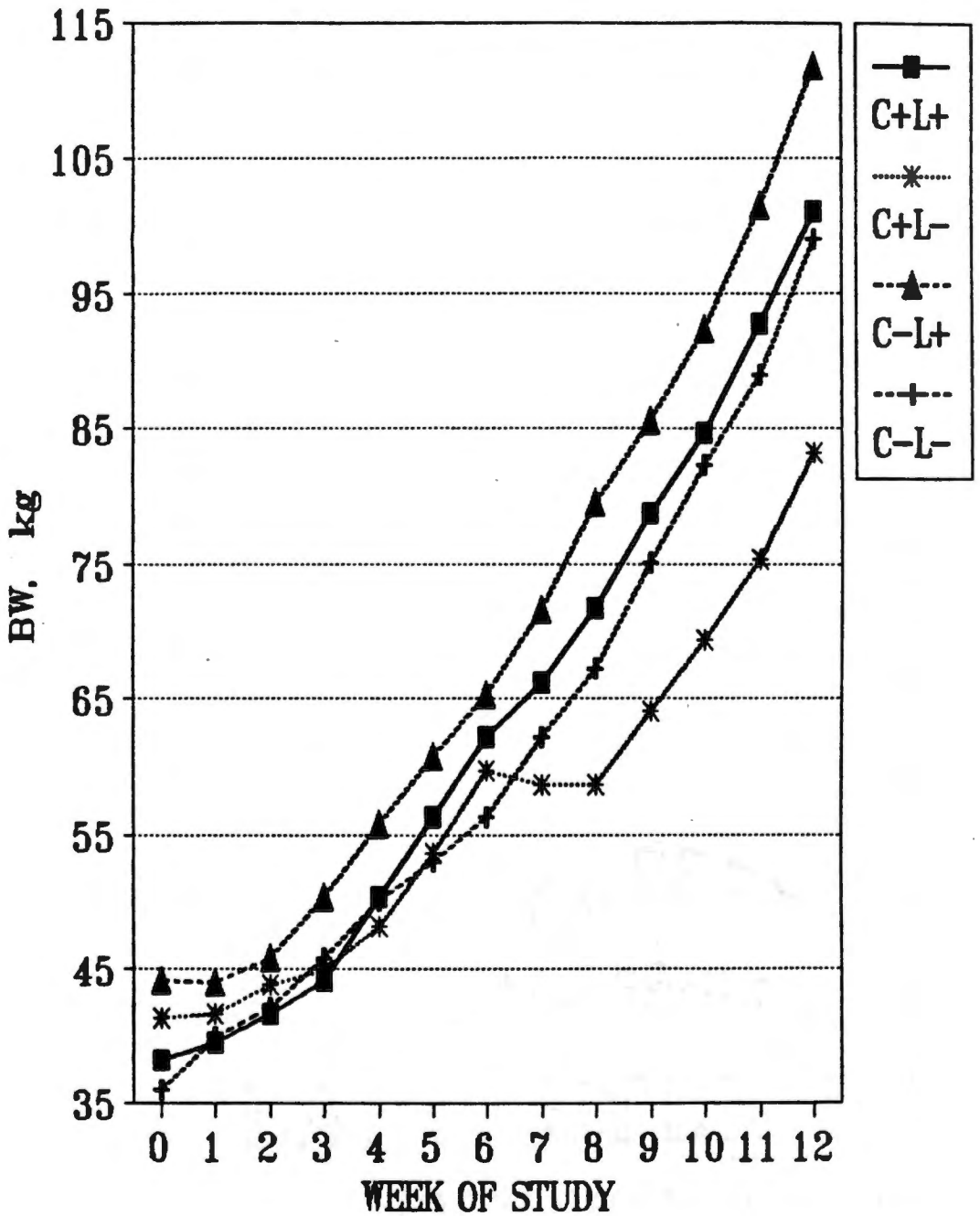

Figure III. Least squares means of BW in calves fed starter containing 0 (L^-) or 40 (L^+) ppm lasalocid and infected with 0 (C^-) or 30,000 (C^+) oocysts (Eimeria bovis) at wk 4.

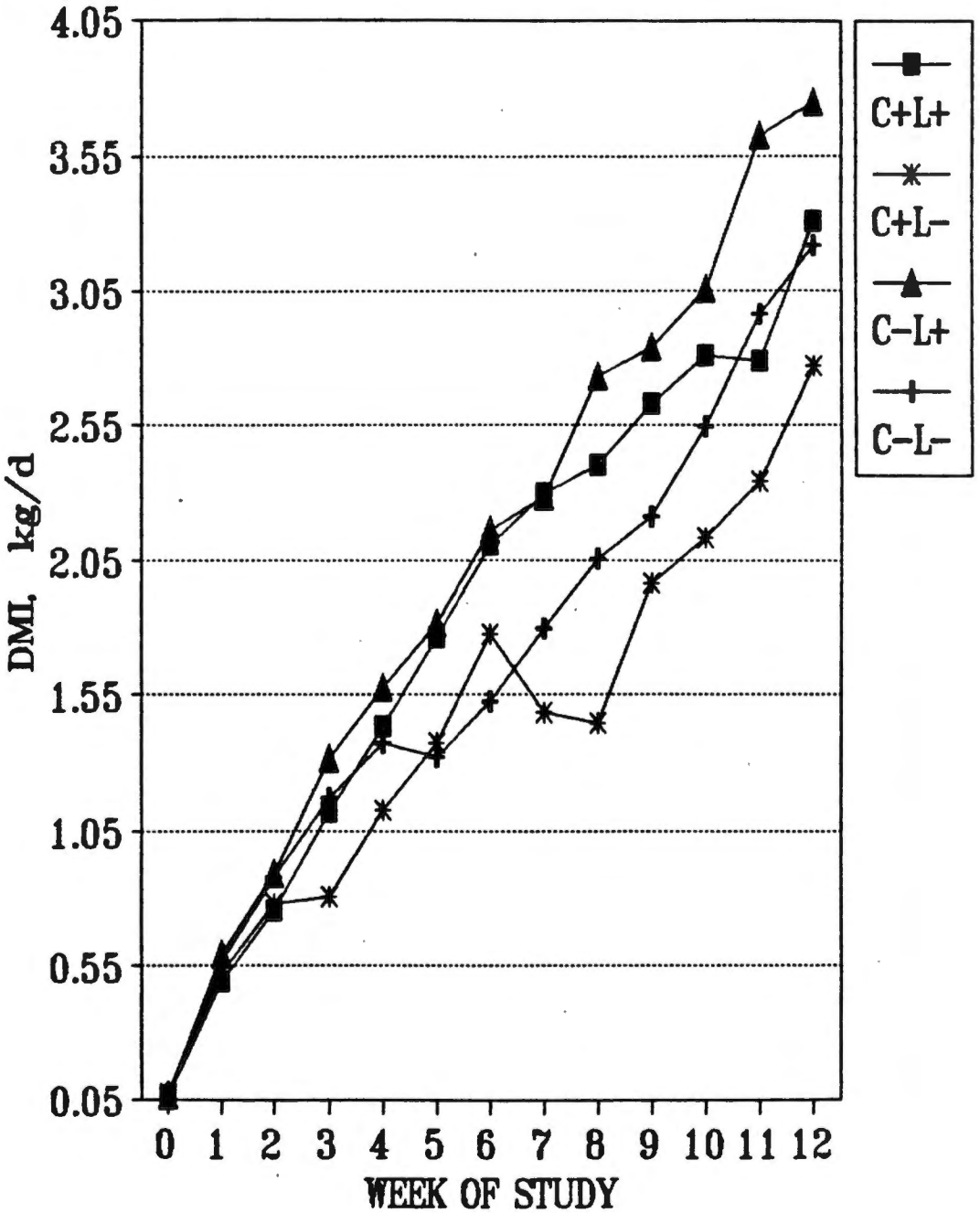

Figure IV. Least squares means of DMI in calves fed starter containing 0 (L^-) or 40 (L^+) ppm lasalocid and infected with 0 (C^-) or 30,000 (C^+) oocysts (Eimeria bovis) at wk 4.

Gain to feed ratio (Table III) was reduced when calves were infected with C^+, but a highly significant interaction ($P<.01$) indicated that calves on C^+L^- treatment had markedly reduced feed efficiency. The reduction in feed efficiency of nearly 30% was probably due to reduced DMI and loss of nutrient absorption in the small intestine. Coccidia can destroy the lining of the small intestine, causing nutrients to be incompletely absorbed (41). That calves fed L^+ did not exhibit improved feed efficiency suggests that these calves had a high rate of passage. Calves fed L^+ had higher intakes (Table III), therefore digestibility may have been lower.

Foreyt et al. (20) reported that increased feed efficiency in cattle given ionophores may be due to changes in VFA composition in the rumen (i.e., acetic acid concentrations decreases and propionic acid concentration increases, whereas total concentration of VFA remains unchanged). These changes result in less energy lost as methane gas due to decreased methane production (3,14,47).

Intake of DM and grain were improved ($P<.01$) by feeding L^+ and tended to be reduced by coccidial infection (Table III). Calves on treatment C^+L^- tended to consume less DM and grain, although interactions were not significant ($P>.05$). Intake of milk was unaffected by treatment.

A significant week-by-treatment interaction ($P<.05$) indicated that intake response was affected by week of study. During wk 6-8, intake of DM decreased (Figure IV) in calves on treatment C^+L^-, presumably due to loss of appetite caused by coccidiosis. Anorectic effects of coccidiosis have been reported (41,53).

Least squares means of logarithms of coccidial oocyst numbers (Table IV) showed a marked increase in calves infected with C^+. However, in calves fed L^+, log of fecal oocysts was reduced ($P<.01$) from 1.71 to 1.14. Oocysts began appearing in feces of C^+L^- and C^+L^+ calves approximately 21 d after infection (Figure V). Calves receiving L^+ shed fewer oocysts than untreated calves ($P<.01$). These results support the findings that lasalocid decreased oocyst numbers at the asexual stage. Approximately 4 wk after infection (wk 8), numbers of oocysts shed by calves infected with C^+ declined (Figure V) through the end of the study, suggesting that calves developed resistance to the coccidia, or that no reinfection of coccidial oocysts occurred. Lasalocid effectively reduced the number of oocysts shed by calves on treatment C^+L^+, although coccidiostatic effect was not total.

Lasalocid minimizes development of coccidia during the early asexual stage of infection by killing the first generation schizonts. The second or third generations of

Table IV

Least squares means of fecal score, body temperature, respiration, logarithms of coccidia, hematocrit, and serum minerals, sodium and potassium in calves weaned on experimental treatment.

Item	Treatments[1] C^+L^+	C^+L^-	C^-L^+	C^-L^-	SE	Contrasts[2] 1	2	3
Fecal score[3,4]	1.9	2.0	1.6	1.4	.14	NS	**	*
Body temp, °C	38.5	38.5	38.4	38.4	.13	NS	NS	NS
Respiration[5]	35.3	31.4	34.7	33.6	.62	NS	NS	NS
Coccidia[3,6]	1.1	1.7	0	0	.28	**	**	**
Hematocrit, %PCV	31.4	31.7	32.4	32.8	.72	NS	NS	NS
Serum Na, mg/dl	373.6	372.7	370.8	378.8	2.6	NS	NS	NS
Serum K, mg/dl	18.3	18.3	18.1	18.9	.36	NS	NS	NS

[1]Treatment = oral infection with 0 (C^-) or 30,000 (C^+) coccidial oocysts; and feeding 0 (L^-) or 40 (L^+) ppm lasalocid in calf starter.

[2]Contrasts: 1= effects of lasalocid vs no lasalocid; 2= effects of coccidia vs no coccidia; 3= interaction; NS=$P>.05$; *=$P<.05$; **$P<.01$.

[3]Significant week*treatment interaction ($P<.05$).

[4]Fecal score: 1= normal feces; firm hard appearance; 2= slightly loose feces; soft somewhat liquid; 3= moderate scours; feces do not hold firm are thin and watery; 4= s severe scours; thin, watery; may contain blood.

[5]Respiration: breaths/minute.

[6]Log of oocysts/g feces.

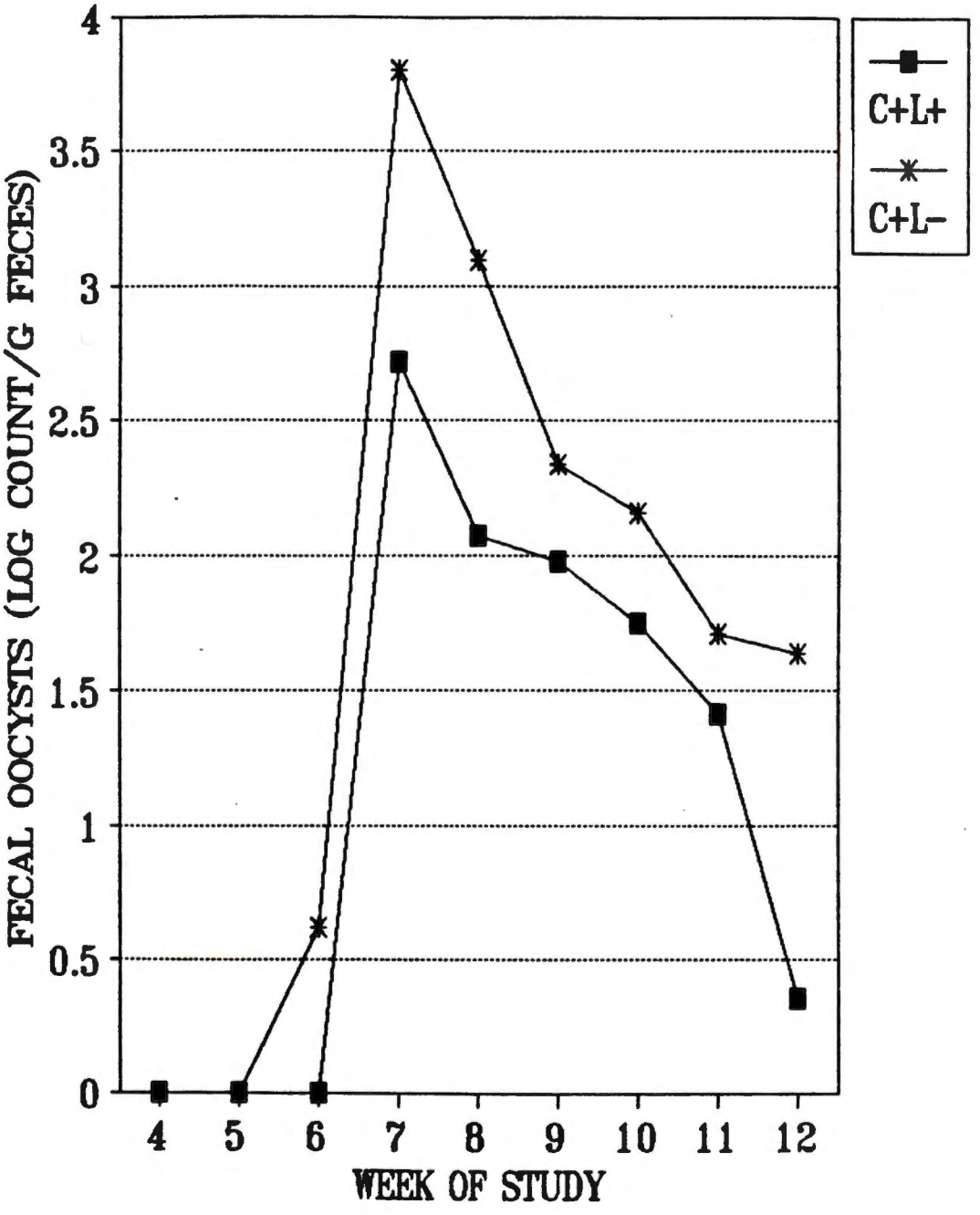

Figure V. Least squares means of logarithms of coccidia oocyst/g in calves fed starter containing 0 (L^-) or 40 (L^+) ppm lasalocid and infected with 0 (C^-) or 30,000 (C^+) oocysts (Eimeria bovis) at wk 4.

schizogony cause most of the damage to the intestines. Because lasalocid's effects are manifested in the early asexual stage of infection, it is more appropriately used as a prophylactic, rather than as a therapeutic agent.

Diarrhea was observed in calves 3 wk after coccidial infection. Calves on C^+L^- treatment developed moderate to severe diarrhea (fecal score $\geq$ 3) during the shedding of oocysts, and one of these developed bloody diarrhea. Calves on C^+L^+ treatment developed moderate diarrhea (fecal score $\geq$ 2) during the shedding of oocysts (Table IV). One calf on C^-L^+ treatment had diarrhea during the study, but its feces did not contain oocysts or blood.

As a result of occurrences of diarrhea, fecal scores were increased significantly in calves treated with C^+ (Table IV). Average fecal scores were 1.9 and 1.5 in calves treated with C^+ and C^-, respectively. Calves on treatment C^-L^+ showed a slight increase in fecal score, whereas calves on treatment C^+L^+ showed a decrease.

Respiration and rectal temperature were unaffected by treatment. Respiration rates ranged from 31.4 to 35.3 breaths/min (Table IV). This range compares favorably with the normal respiration rate for calves, which is 30 to 32 breaths/min (4). Rectal temperature averaged 38.5°C (Table IV), similar to normal rectal temperature of 38.0 to 39.3°C (4).

Packed cell volume, and serum Na^+ and K^+ were unaffected by treatment (Table IV), and averaged 32%, and 374 and 18 mg/dl, respectively. Fitzgerald (16) reported normal potassium levels in serum from uninfected calves ranged form 19.5 to 20.3 mg/dl. Our potassium levels in serum from uninfected calves were 18.1 to 18.9 mg/dl. Sodium ranged from 307.5 to 322 mg/dl (16). Our sodium ranged form 370.8 to 378.8 mg/dl. Changes in sodium or potassium levels in serum are generally minor, unless signs of coccidiosis are severe (16).

Researchers (12,51) have reported effects of coccidiostats in dairy calves from 6 to 10 wk of age. However, greater attention should be given to the effects of coccidiosis in younger calves (4 wk of age). Assuming a 3 wk incubation period, and manifestation of clinical signs in calves 3-6 wk of age (30), it is clear that infection by coccidia occurs early in the life of the young calf. Also, because early weaned calves may not consume large amounts of starter prior to weaning, combination of weaning stress and coccidiosis may increase morbidity and mortality significantly. Results of this study are similar to those of (19) confirming the efficacy of lasalocid in controlling coccidiosis.

The recommended dosage of lasalocid to control coccidiosis is 1.0 mg/kg of BW. Intake of lasalocid from wk

0 to 4 averaged .47 mg/kg BW (Figure VI), which is below recommendation. However, intake of starter (and lasalocid) increased with weaning; at wk 5 intake of lasalocid was above recommendation (1.3 mg/kg BW/d) and remained constant to the end of the study. At time of coccidial infection, intake of lasalocid was lower than recommended due to low feed intake prior to weaning. Our results indicate that intake of lasalocid should precede weaning to reduce coccidial infection in 3 to 4 wk old dairy calves. The easiest way to accomplish this would be to add lasalocid to milk replacer. This would increase lasalocid intake before calves began eating medicated dry feed.

Results from our study suggest that lasalocid fed at 40 ppm in starter is effective in reducing coccidial challenge and increases growth in early weaned dairy calves when coccidia are introduced at 4 wk of age. Lasalocid fed at 40 ppm in calf starter may benefit calves exposed to coccidia immediately after weaning at 4 wk both from increased rate of gain as well as reduced clinical signs of coccidiosis. Results corroborate those of (30), in which calves were exposed naturally to coccidia.

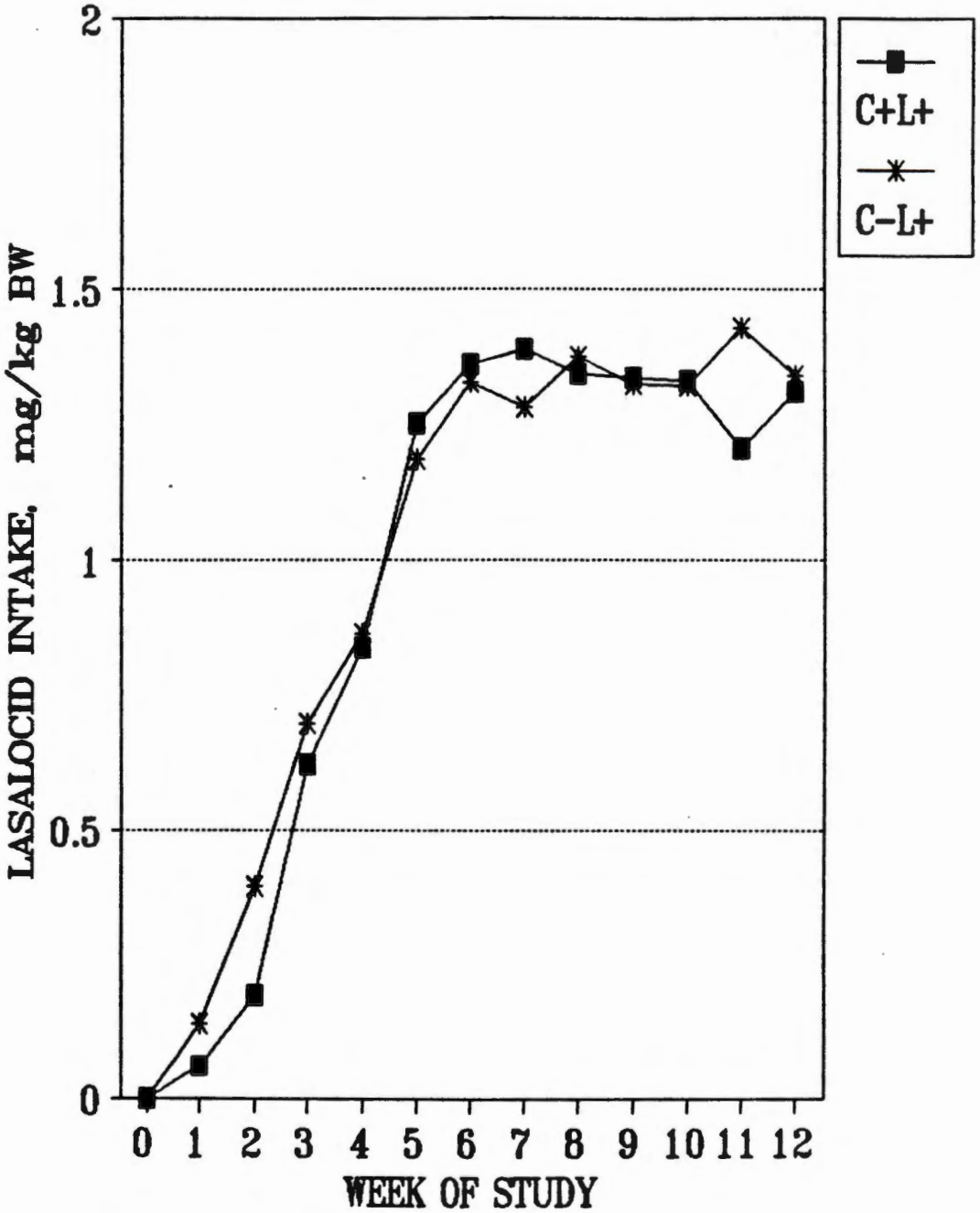

Figure VI. Mean lasalocid intake, mg/kg BW in calves fed starter containing 40 (L^+) ppm lasalocid and infected with 0 (C^-) or 30,000 (C^+) oocysts (Eimeria bovis) at wk 4.

CHAPTER V

CONCLUSIONS

Average daily gains and intake were increased by lasalocid and decreased by coccidial infection. Fecal score was greater in calves infected with coccidia. A significant interaction suggested a slight increase in fecal score with C^-L^+, whereas C^+L^+ showed a decrease. Logarithms of coccidial oocyst numbers showed that fecal oocyst counts were increased by infection of calves by coccidia but reduced by feeding L^+. Respiration, rectal temperature, hematocrit, Na^+ and K^+ concentrations were unaffected by treatment. Lasalocid increased growth in this study by reducing rumen acetate to propionate ratio, causing higher propionate production. This is more energy efficient because the amount of lost methane related to the output of acetic and butyric acids is reduced. Lasalocid minimized effects of coccidial infection in this study by interrupting the asexual stage of development of the coccidia.

LITERATURE CITED

LITERATURE CITED

1. Association of Official Analytical Chemists. 1980. Official methods of analysis. 13th ed. Assoc. Offic. Analy. Chem., Washington D.C.
2. Baile, C. A., C. L. McLaughlin, E. L. Potter and W. Chalupa. 1979. Feeding behavior changes of cattle during introduction of monensin with roughage or concentrate diets. J. Anim. Sci. (Suppl. 1) 47:414 (Abstr).
3. Bartley, E. E., E. L. Herrod, R. M. Bechtle, D. A. Sapienza, and B. E. Brent. 1979. Effect of monensin or lasalocid with and without niacin or amiclorol, on rumen fermentation and feed efficiency. J. Anim. Sci. 49:1066.
4. Battaglia, R. A., and V. B. Mayrose. 1981. Handbook of livestock management technique. Burgess Company, Minneapolis., MN.
5. Berger, L. L., and S. C. Ricke. 1980. Comparison of lasalocid and monensin for feedlot cattle. J. Amim. Sci. (Suppl. 1) 51:345 (Abstr).
6. Blaxter, K. L., and W. A. Wood. 1958. Some observations on the biochemical events associated with diarrhea in calves. Vet. Rec. 65:889.
7. Blouin, D. C. and A. M. Saxton. 1990. General linear mixed models (Ver. 1.0) User's Manual. Louisiana Ag.

Exp. Stat. LA State Univ., Baton Rouge, LA.

8. Boling, J. A., N. W. Bradley and L. D. Campbell. 1977. Monensin levels for growing and finishing steers. J. Anim. Sci. 44:867.

9. Brethour, J. R. 1979. Lasalocid for finishing steers. J. Anim. Sci. (Suppl. 1) 48:357 (Abstr).

10. Brown, R. E. and A. Davidovich. 1979. The performance response of growing-finishing cattle fed graded levels of lasalocid. J. Amim. Sci. (Suppl. 1). 49:358 (Abstr).

11. Chen, M. and M. J. Wolin. 1979. Effect of monensin and lasalocid-sodium on the growth of methanogenic and rumen saccharolytic bacteria. Appl. Environ. Microbiol. 38:72.

12. Conlogue G., W. J. Foreyt, R. B. Wescott. 1984 Bovine coccidiosis protective effects of low-level infection and coccidiostat treatments in calves. Am. J. Vet. Res. 45:863.

13. Davis, G. V. 1978. Effects of lasalocid sodium on the performance of finishing steers. J. Anim. Sci. (Suppl. 1) 47:414 (Abstr).

14. Dennis, S. M., T. G. Nagaraja, and E. E. Bartley. 1981. Effects of lasalocid or monensin on lactate-producing or ASMG rumen bacteria. J. Anim. Sci. 52:418.

15. Ernst, J. V., and G. W. Benz. 1986. Intestinal coccidiosis in cattle. The Veterinary Clinics of North America/Parasites: Epidemiology and Control. W.B. Saunders, Philadelphia, PA.

16. Fitzgerald, P. R. 1967. Effect of bovine coccidiosis on blood serum sodium and potassium levels of calves. Am. J. Vet. Res. 28:667.

17. Fitzgerald, P. R., 1975. The significance of bovine coccidiosis as a disease in the United States. Bovine Pract. 10:28.

18. Fitzgerald, P. R., and M. E. Mansfield. 1973. Efficacy of monensin against bovine coccidiosis in young Holstein-Friesian calves. J. Protozool. 20:121.

19. Fitzgerald, P. R., and M. E. Mansfield. 1979. Efficiency of lasalocid against coccidia in cattle. J. Parasitol. 65:824.

20. Foreyt, W. J., D. H. Rice, and R. B. Wescott. 1986. Evaluation of lasalocid as a coccidiostat in calves: Titration, efficacy, and comparison with monensin and decoquinate. Am. J. Vet. Res. 47:2031.

21. Georgi, J. R. 1985. Parasitology for veterinarians. Fourth Edition. W.B. Saunders Company, Philadelphia, PA.

22. Goering, H. K., and P. J. Van Soest. 1970. Forage fiber analyses. Agric. Handbook No. 379. Agr. Res. Serv.,U.S. Dept. Agr. Washington D.C.

23. Hammond, D. L., G. W. Clark, M. L. Miner, W. A. Trost, and A. E. Johnson. 1959. Treatment of experimental bovine coccidiosis with multiple small doses and single large doses of sulfamethazine and sulfabromomethazine. Am. J. Vet. Res. 20:708.

24. Hammond, D. M., D. L. Ferguson, and M. L. Miner. 1960. Results of experiments with nitrofurazone and sulfamethazine for controlling coccidiosis in calves. Cornell Vet. 50:351.

25. Hammond, D. M., F. L. Anderson, and M. L. Miner. 1963. The occurrence of a second asexual generation in the life cycle of Eimeria bovis in calves. J. Parasit. 49:428.

26. Hammond, D. M., R. Fayer, and M. L. Miner. 1966. Amprolium for control of experimental coccidiosis in cattle. Am. J. Vet. Res. 27:199.

27. Hammond, M., G. W. Bowman, L. R. Davies, and B. T. Simms. 1946. The endogenous phase of the life cycle of Eimeria bovis. J. Parasit. 32:409.

28. Hanson, T. L. and T. Klopfenstein. 1979. Monensin, protein source and protein levels for growing steers. J. Anim. Sci. 48:474.

29. Hawk, P. B., B. L. Oper, and W. H. Summerson. 1948. Practical Physiological Chemistry. Blakiston Co., Philadelphia, PA.

30. Hoblet, K. H., T. P. Charles, and R. R. Howard. 1989. Evaluation of lasalocid and decoquinate against coccidiosis resulting from natural exposure in weaned dairy calves. Am. J. Vet. Res. 50:1060.

31. Horton, G. J., and W. E. Brandt. 1981a. Lasalocid or monensin for finishing steers fed a high-silage diet. J. Anim. Sci. (Suppl. 1) 53:406 (Abstr).

32. Horton, G. J., and W. E. Brandt. 1981b. Response of backgrounding calves fed a high silage diet and different protein sources to zeranol implants and lasalocid. J. Anim. Sci. (Suppl. 1) 53:406 (Abstr).

33. Hungate, R. E. 1966. The Rumen and Its Microbes. Academic Press Inc., New York, New York.

34. Jenny, B. F., S. E. Mills, W. E. Johston, and G. D. O'Dell. 1978. Effect of fluid intake and dry matter concentration on scours and water intake in calves fed once daily. J. Dairy Sci. 61:765.

35. Kemp, R. L., and D. D. Susie. 1978. Clinical pathology of E. zuernii coccidiosis in cattle. Abs. 53rd Annual Meet. Am. Soc. Parasit. 78.

36. Kuhl, G. L., M. Esser, M. E. Bergeland and L. B. Embry. 1980. Efficacy of lasalocid with solar-dried, acid-

treated, and ensiled shelled corn finishing rations. J. Anim. Sci. (Suppl. 1) 51:376 (Abstr).

37. Levine, N. D., and V. Ivens. 1970. The coccidian parasites (Protozoa, Sporozoa) of ruminants. Illinosis Biological Monographs. Urbana, University of Illinosis Press. 47.

38. McDougald, L. R. 1978. Monensin for the prevention of coccidiosis in calves. Am. J. Vet. Res. 39:1748.

39. Messersmith, R. E. 1985. Bovatec Symposium III. Hoffman La Roche Inc., Nutley, N.J.

40. Migioia, S. N. 1982. Comparison of a conventional feeding program with a two-stage complete ration program utilizing cottonseed hulls as a fiber source for replacement heifers. M.S. Thesis, Univ. of Tenn, Knoxville.

41. Muirhead, S. 1989. Coccidiosis infections often go undetected in beef, dairy cattle. Feedstuffs. 15:87.

42. National Research Council. 1989. Nutrient requirements of dairy cattle, 6th rev. ed. Washington, D.C.: National Academy Press.

43. Prange, R. W., C. L. Davis, and J. H. Clark. 1978. Propionate production in the rumen of Holstein steers fed either a control or monensin supplemented diet. J. Anim. Sci. 46:1120.

44. Quigley, J. D.,III, L. A. Caldwell, G. D. Sinks, and R. N. Heitmann. 1990. Blood glucose, nonesterified fatty acids and ketones response to weaning and feed intake in young calves. J. Dairy Sci (Accepted).

45. Radostits, O. M., and P. H. Stockdale. 1980. A brief review of bovine coccidiosis in western canada. Can. Vet. J. 21:227.

46. Raun, A. P., C. O. Cooley, E. L. Potter, R. P. Rathmacher and L. F. Richardson. 1976. Effect of monensin on feed efficiency of feedlot cattle. J. Anim. Sci. 43:670.

47. Richardson, L. F., A. P. Raun, E. L. Potter C. O. Cooley and R. P. Rathmacher. 1976. Effect of monensin on rumen fermentation in vitro and in vivo. J. Anim. Sci. 43:657.

48. Roy, J. B., K. W. Hawkins, H. Gillian, J. M. Lang, and P. L. Ingraham. 1959. The effect of white scours on the sodium and potassium concentration in the serum of newborn calves. Brit. J. Nutr. 13:219.

49. Shaw, J. C., W. C. Ensor, J. F. Tellechea and S. D. Lee. 1960. Relation of diet to rumen volatile fatty acids, digestibilty, efficiency of gain and degree of unsaturation of body fat in steers. J. Nutr. 71:203.

50. Stockdale, P. G. 1976. Schizogony and gametogony of Eimeria zuernii (Rivolta, 1878) Martin, 1909. Vet. Parasit. 1:367.

51. Stromberg, B. E., J. C. Schlotthauer, B. D. Armstrong, W. E. Brandt, and C. Liss. 1982. Efficacy of lasalocid sodium against coccidiosis (Eimeria zuernii and Eimeria bovis) in calves. Am. J. Vet. Res. 43:583.

52. Thonney, M. L., E. K. Heide, D. J. Duhaime, R. J. Hand and D. J. Perosio. 1981. Growth, feed efficiency and metabolic concentrations of cattle fed high forage diets with lasalocid or monensin supplements. J. Anim. Sci. 52:427.

53. Williams, L. 1984. Dealing with the frustration of the war on coccidiosis. DVM 15:38.

54. Wolin, M. J. 1960. A theoretical rumen fermentation balance. J. Dairy Sci. 43:1452.

VITA

The author was born July 9, 1966, in Clarksville, Tennessee. He has lived with his parents, Mr. and Mrs. Howard Sinks, and brother, Michael, on a beef cattle, swine, and tobacco farm in Montgomery County, Tennessee. The author attended Montgomery Central High School. Upon graduation from high school in 1984, he enrolled in the agriculture program at Austin Peay State University.

At Austin Peay State University, the author was accepted into Delta Tau Alpha Honor Society of Agriculture and received the Academic Achievement Award in Agriculture. He was a member of the Pre-Vet Club. In January of 1988, the author worked at the Animal Clinic in Clarksville. In June of 1988, the author received his B.S. degree in Agriculture.

The author entered Graduate School at The University of Tennessee, Knoxville, in August 1988 and began work toward a Master's degree in Animal Science. The author worked as a Graduate Research Assistant and gained valuable experience in laboratory analyses. The author also worked as the Animal Caretaker for the Animal Science Department.