EFFECTS OF FEEDING LASALOCID AND MONENSIN UPON MINERAL STATUS OF STEERS, AND PARTIAL ABSORPTION AND RENAL HANDLING OF MINERALS IN SHEEP

by

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(ABSTRACT)

Studies were conducted to determine the effects of feeding lasalocid and monensin upon mineral status of ruminants, changes in digestive and renal physiology which bring about altered mineral status, and the effects of dietary K upon the actions of ionophores. Two 84-d grazing trials were conducted with steers fed no ionophore, 200 mg lasalocid, or 150 mg monensin. Monensin tended to increase gain of steers, but lasalocid had no effect. Feeding supplemental lasalocid and monensin altered Ca, P, Mg, Na, K, Cu, Fe and Zn status of grazing steers, but effects were not consistent.

Twenty-four crossbred steers were individually fed in a 147-d finishing trial to study a possible K x monensin interaction. Steers were fed diets *ad libitum* containing .4% or 2.3% K, with or without 23 ppm monensin, dry basis. Feeding monensin with low K increased serum Na, Mg, inorganic P, Cu and Zn, but monensin fed with high K had no effect or decreased these serum minerals.

Fifteen wethers, fitted with abomasal and ileal cannulae, were fed a basal diet alone or supplemented with 23 ppm lasalocid or monensin; Apparent absorption of Mg, K and Fe increased when ionophores were fed.

Magnesium flow through the small and large intestines was decreased by lasalocid and monensin. Effects of lasalocid and monensin differed for metabolism, digestive tract flow and(or) partial absorption of Ca, P, Cu, Fe and Zn.

Twelve ewe lambs were used in a renal clearance experiment. Lambs were fed a basal diet with no ionophore, 23 ppm lasalocid, or 23 ppm monensin, dry basis. Ionophores were fed starting on d 1 of the experimental period. Serial collections of blood and urine were made during d 1 and d 5 of the experimental period. Monensin reduced urine flow rate at d 1 and d 5. Feeding monensin lowered serum clearance and urinary excretion rates of Na and K at d 5. Serum clearance and urinary excretion rates of Ca were lower in sheep fed ionophores at d 1. Feeding monensin decreased serum clearance and urinary excretion rates of Cu at d 1. At d 5, urinary excretion rates of Zn were greater when lasalocid was fed, and lower when monensin was fed, compared to feeding no ionophore.

These studies indicate that dietary lasalocid and monensin can alter mineral status of ruminants, although their effects may differ. The effects of ionophores may be dependent upon dietary K levels.

DEDICATION

This dissertation is dedicated to the memory of my mother,

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INTRODUCTION

In an effort to increase the economic return of animal production, livestock producers have used non-nutritive feed additives such as ionophores to improve efficiency. Lasalocid and monensin are ionophores currently approved for feeding to ruminants. Both are used to increase feed efficiency and rate of weight gain in finishing cattle, and to increase rate of gain in pasture cattle. Lasalocid is used to prevent coccidiosis in sheep.

These compounds improve ruminant performance primarily by promoting an energetically favorable shift in ruminal carbohydrate fermentation, decreasing acetate, while increasing propionate production. They also promote other changes in nutrient metabolism which may be related to improved animal growth.

As ionophores, lasalocid and monensin act basically to alter the flow of cations across cell membranes. Lasalocid is a divalent ionophore, binding both divalent cations(such as Ca^{2+} and Mg^{2+}) and monovalent cations (including Na^{+} and K^{+}). Monensin is a monovalent ionophore, binding only monovalent cations, primarily Na^{+} and K^{+}. Recent research has indicated that these compounds, fed at levels to increase ruminant performance, alter mineral metabolism. Therefore, feeding ionophores may be beneficial in the prevention of nutritional diseases such as grass tetany (hypomagnesemia), and milk fever (hypocalcemia). Dietary ionophores may also alleviate or attenuate mineral toxicities in ruminants.

A two-year grazing study was conducted to determine the effects of supplemental lasalocid and monensin upon mineral status of grazing steers. Studies using multicannulated sheep to determine digestive tract flow and partial absorption of minerals, and renal clearance to determine renal handling of minerals in sheep, were conducted to determine the physiological effects of feeding ionophores which bring about altered mineral metabolism. The effectiveness of lasalocid and monensin to improve animal production may be altered by changes in dietary cation levels, as well. An experiment was conducted to examine the existence of a K x monensin interaction for performance, ruminal fermentation, protein status, and mineral status of finishing steers.

REVIEW OF LITERATURE

Effects of Ionophores on Cation Transport.

The effects of ionophores upon ruminant performance and metabolism are ultimately due to the effect of these compounds on the flow of cations across cell membranes. Ionophores are organic compounds of moderate molecular weight, that form lipid soluble complexes with cations (Pressman, 1976). The kinetics of complexing and decomplexing with cations and the diffusion rate of ionophore-cation pairs are highly favorable, allowing transport turnover rates across cell membranes of thousands per second. Carboxylic ionophores, such as lasalocid and monensin, have linear backbones with oxygen-containing hetrocyclic rings inserted. These ring groups include ether, carboxyl, hydroxyl, or carbonyl groups. The carboxylic ionophores form electrically neutral zwitterionic complexes with cations, enveloping the cations, and displacing a solvation shell with oxygen atoms. The lipid-soluble complexes allow the transport of cations across cell membranes, down their concentration gradients.

Lasalocid is a 20 atom molecule which carries monovalent cations in a 1:1 ratio, but is capable of forming dimers to carry divalent cations (Pressman and Fahim, 1982). Liganding is accomplished by cation-binding to both the carboxyl head and the hydroxyl tail of the linear structure (Pressman, 1976). Therefore, the cation sits on, rather than within the ionophore ring. This binding characteristic, along with the ability of lasalocid to form dimers, allows the ionophore to carry a relatively broad array of cations, with a ranking of Cs > Rb = K > Na > Li > Sr > Ca > Mg. Potassium is preferentially carried at 10 times the rate for Na.

Lasalocid has been shown to elicit a Ca^{2+} for $2K^+$ exchange (Haynes et al., 1980). A secondary increase in Na^+/Ca^{2+} exchange across cell membranes has also been demonstrated with lasalocid (Mallaise et al., 1980).

Monensin is a 24 atom ionophore which forms complexes only in the ionized form, obtained at physiological pH and above (Pressman and Fahim, 1982). Liganding of cations with this ionophore involves only the carboxylic head group (Pressman, 1976). Thus, the molecule is highly selective, transporting Na > K > Rb > Li > Cs. Sodium ions are preferred 10 times over K^+. Sandeaux et al. (1982) demonstrated that monensin induces a Na^+-H^+-exchange or antiport. Monensin was also shown to carry Na^+ in a 1:1 ratio. The increase in intracellular Na^+ and increased extracellular K^+ brought about by monensin tend to stimulate the action of Na^+-K^+-ATPase. This was first demonstrated in mouse fibroblast cells (Smith and Rozengurt, 1978), and has since been seen in human skin fibroblasts, hamster kidney cells and murine cells, hence, has become recognized as a general monensin-induced phenomenon (Mendoza et al., 1980). The increased activity of the Na^+-K^+ pump has been deemed responsible for a hyperpolarization of cell membranes exposed to monensin (Lichstein and Samuelov, 1982). Monensin has been demonstrated to evoke a secondary increase in Na^+/Ca^{2+} exchange across cell membranes (Jacques et al., 1981).

Antimicrobial Effects of Ionophores.

The effects of lasalocid and monensin upon altering concentration gradients of ions across cell membranes is believed to be responsible for their antimicrobial effects (Bergen and Bates, 1984). These authors suggested that monensin dissipates the proton gradient, depleting cells of ATP, in response to a monensin-induced Na^+-H^+ exchange. Monensin-susceptable bacteria would be those which utilize substrate level phosphorylation for generating ATP, using this ATP to expel H^+ from the cell interior. Those bacteria capable of oxidative phosphorylation, synthesizing ATP while extruding intracellular protons, would prosper in an environment containing monensin. The authors also suggested that individual microbes may produce a shift in their fermentation pathways in order to increase ATP production or decrease intracellular H^+ production, in response to monensin.

Russell (1987) has demonstrated that the anti-microbial effect of monensin upon Streptococcus bovis is actually due to its dissipation of the K^+ gradient, rather than Na^+. He reported that 5 mg/liter monensin decreased growth rate of S. bovis immediately, and no growth was seen after 3 h. Monensin decreased intracellular K, and pH, while increasing intracellular Na. Although monensin prefers Na to K, Russell proposed that the 70-fold K gradient (higher within the cell), compared to the 2.7-fold Na gradient (higher inside), resulted in greater transport of K^+ by monensin, through a K^+-H^+ exchange. As a result of this change in ion levels within and without the cell, monensin decreased the proton motive force, by increasing the proton chemical potential, without altering the electrical potential of the cell.

These theories suggest that the antimicrobial effects of ionophores may be altered by manipulating cation levels within the rumen environment. Dawson and Boling (1984) developed monensin-resistant strains of Bacteriodes ruminicola in media containing 1.3, 4,6, or 12.3 mM K. Low-K levels decreased the minimum inhibitory concentration of monensin needed to halt growth, while high K increased the minimum inhibitory concentration. These workers later reported differences between ruminal bacterial species in their response to monensin and lasalocid in media of varied K concentrations (Dawson and Boling, 1987).

Growth of Eubacterium ruminantium was inhibited by low concentrations of ionophores at all K levels, while Streptococcus bovis resisted high concentrations of ionophores at all K levels. However, the minimum inhibitory concentration of lasalocid and monensin increased with increased K for Bacteriodes succinogenes, Butyrivibrio fibrosolvens, Ruminococcus albus, Ruminococcus flavefaciens, and one strain of Bacteriodes ruminicola. Increasing K concentration from 7.9 to 23.3 mM decreased the lag time and increased cell yield of one strain of B. ruminicola and two strains of Selenomonas ruminantium grown in ionophore-containing media, although growth was still greatest at 1.3 mM K. The authors suggested that, overall, increasing K concentrations in the rumen will decrease the magnitude of the K gradient, preventing the antimicrobial effects of ionophores.

Mode of Action of Ionophores.

Fermentation. The improved performance resulting from lasalocid and monensin supplementation is primarily due to an energetically favorable shift in ruminal fermentation. Monensin decreased the acetate:propionate ratio by increasing propionate production and decreasing acetate production, *in vitro* (Richardson et al., 1976; Wallace et al., 1980), and *in vivo* in cattle (Dinius et al., 1976; Perry et al., 1976; Richardson et al., 1976), and sheep (Calhoun et al., 1979). Improved performance of finishing cattle fed lasalocid has been associated with decreased acetate:propionate ratio in the ruminal fluid (Brown et al., 1979; Guitterez et al., 1982; Ricke et al., 1984). Horton and Stockdale (1981) also reported decreased acetate:propionate ratio and increased feed efficiency in finishing lambs, which was confirmed by Patterson et al. (1983). In vitro ruminal fermentation studies have consistently shown an increase in propionate and decreased acetate production with the addition of lasalocid (Herod et al., 1979; Fuller and Johnson, 1981).

The fermentation of carbohydrates to propionate conserves more energy than fermentation to acetate or butyrate (Hungate, 1966). The increase in propionate (mol/100 mol) when monensin is fed is coupled with a decrease in methane production (Van Nevel and Demeyer, 1977; Wallace et al., 1980). A decrease in methanogenesis has also been demonstrated with in vitro cultures following the addition of lasalocid (Herod et al., 1979; Fuller and Johnson, 1981). Research has shown that a negative correlation exists between propionate and methane production (Van Nevel et al., 1969), and it was suggested that monensin increases propionate production due to a shift from formate and methane to succinate or propionate production (Van Nevel and DeMeyer, 1977). Monensin has been demonstrated to increase propionate production even when added to in vitro systems in which methanogenesis has been inhibited (Slyter, 1979). Chen and Wolin (1979) suggest that lasalocid and monensin directly affect the rumen microbial population in vitro, selecting for succinate-forming and propionate-producing bacteria, while inhibiting those that produce H_2 and formate. Similar microbial effects have been reported by Dennis et al. (1981) and Henderson et al. (1981).

Cation x Ionophore Interactions in the Rumen. *In vitro* fermentation experiments have been conducted to determine the effects of varied cation levels upon the alteration of ruminal fermentation patterns produced by ionophores. Chirase et al. (1988a) examined the effects of ,07% and .14% Mg, and .6% and 2.5% K, with and without 20 ppm lasalocid, upon microbial fermentation in a continuous culture system. They reported a significant Mg x lasalocid interaction for butyrate production, with increased Mg levels decreasing butyrate in the presence of lasalocid. A K x lasalocid interaction was demonstrated for propionate and butyrate, with high K increasing propionate and decreasing butyrate in the presence of lasalocid. That report also examined Mg and K interactions with monensin. The authors demonstrated a significant Mg x monensin interaction for acetate:propionate ratio. Monensin produced a greater decrease in acetate levels with high Mg, and a greater increase in propionate concentration with low Mg. Acetate:propionate ratio decreased to a greater extent with monensin in a high-Mg medium vs. a low-Mg medium. No K x monensin interaction was demonstrated.

Schwingel et al. (1987) examined the effects of K level upon VFA production by mixed ruminal microbes exposed to ionophores. Potassium was found to have a linear effect upon acetate:propionate ratio in the presence of 2.5 g/liter lasalocid, with the ratio increasing with increased K level. However, monensin decreased acetate:propionate at 150 mM K, compared to 50 mM K. Rumpler et al. (1986) examined the effects of varied Na and K levels fed to steers receiving ionophores upon ruminal methanogenesis in cattle. They reported that feeding 2.5% supplemental Na decreased methane production 19% with monensin, and tended to decrease methane levels with lasalocid, compared with no added Na. Addition of 2.5% K to the diet tended to increase methane production with lasalocid, and decrease methane with monensin.

Although this shift in the ruminal fermentation pattern may be the primary effect of ionophores in ruminants, Raun et al. (1976) calculated that the increased propionate produced when monensin was fed increased the metabolizable energy of the diet only 3 to 6%, which could not account for the improvements observed in feed efficiency. This indicates that an effect other than an increased efficiency of carbohydrate fermentation is responsible for a portion of the improvement attributed to ionophore supplementation.

Some of the additional effects may be due directly to the increased propionate produced when ionophores are supplemented. Propionate infusions have been shown to increase irreversable glucose loss and rate of carbon fixation from blood carbonate (Judson and Leng, 1973), both indications of increased gluconeogenesis. Bergman et al. (1966) showed that up to 50% of propionate is used for glucose synthesis, and stated that propionate may contribute to the glucose pool. Monensin increased propionate production and irreversable glucose loss in cattle (Van Maanen et al., 1978). However, only 20% of the increased propionate produced could be accounted for by the increase in gluconeogenesis, suggesting that propionate may be sparing other substances normally used for energy.

Nitrogen Metabolism. Some of these substances may be amino acids, since both lasalocid (Patterson et al., 1983; Ricke et al, 1984) and monensin (Beede et al., 1980; Adams et al., 1981) have been shown to increase N retention. It has also been suggested that gluconeogenic amino acids are spared by the increased propionate produced during monensin supplementation (Byers, 1980). Eskeland et al. (1974) showed that propionate infused into the blood increased N retention and decreased blood amino acids, compared to acetate and butyrate infusions, indicating that propionate may provide increased energy for protein synthesis.

The effect of ionophores upon N metabolism may be due to an increase in N escaping the rumen when these compounds are fed. *In vitro* studies have demonstrated decreased N digestion in a continuous culture system with 32.5, 65, or 130 ppm lasalocid added (Fuller and Johnson, 1981). Herod et al. (1979) found lasalocid decreased microbial protein synthesis with rumen fluid unadapted to ionophores, but no effect was seen with adapted rumen fluid. Monensin decreased microbial protein synthesis and increased protein escape from the rumen (Poos et al., 1979; Isichei and Bergen, 1980; Muntifering et al., 1980). Muntifering et al. (1980) reported an increased percentage of organic matter and N digestion occurred in the intestine of steers rather than in the rumen when monensin was added to corn diets.

Rumen Fill and Rate of Passage. Ionophore-induced changes in digestive physiology have also been recorded. Byers and Schelling (1984) determined digestive tract fill, using D_2O dilution, in cattle grazing high-quality and poor-quality pasture, and supplemented with or without lasalocid or monensn. Lasalocid consistently decreased gastrointestinal fill among cattle grazing high-quality forage, but results were highly variable in animals fed poor-quality pasture. These findings may help explain why Jacques et al. (1987) reported no effect on performance or forage utilization from cows grazing poor-quality winter range and receiving supplements containing monensin. Reduced digestive tract fill was reported in cattle grazing high-quality pasture when monensin was supplemented, but this ionophore did not affect fill when cattle were on mature pasture. Monensin has also been shown to decrease rate of passage (Pond and Ellis, 1979; Pond et al., 1980), increase rumen turnover time (Tolbert et al., 1979; Adams et al., 1981), and decrease rumen turnover rate (Lemenager et al., 1978a; Owens et al., 1979) in pasture-fed cattle.

Systemic Effects of Ionophores.

Ionophores may also act systemically within cell membranes of the ruminant. Donoho et al. (1978) reported that steers metabolized 50 to 60% of ^{14}C-monensin fed, and the metabolites were excreted primarily through the bile. Davison (1983), using two, 3-mo-old, bile-cannulated calves, found as much as 39% of ^{14}C from ^{14}C-monensin could be absorbed. Bile served as the primary route of ^{14}C excretion. Although monensin was not identified in the bile, a demethylated metabolite of monensin was found. Additional investigations on ^{14}C-monensin metabolism have shown ^{14}C present in the muscle, kidney, fat, lung, heart, liver, and spleen of steers fed 300 mg/d of unlabeled monensin for 2 wk, followed by ^{14}C-monensin fed 2 d prior to slaughter (Herberg et al., 1978). During a second trial, ^{14}C was detected in the muscle, fat , kidney, liver and heart of cattle 12 h after an oral dose of 150 mg/d ^{14}C-monensin.

Using a radiochemical assay for the determination of pharmacologically active monensin, Fahim and Pressman (1981) reported that active monensin, or its metabolites, were present in the plasma, heart, lung, brain, muscle and fat of rabbits given 200 ug/kg body weight monensin iv, or 10 mg/kg body weight orally. Pressman et al. (1981), using this assay, learned that chickens fed 110 ppm monensin daily, followed by monensin withdrawl for 5 d, still had substantial levels of pharmacologically active monensin present in the tissues, especially the liver (.7 ppm monensin). 1981). Donoho (1984), reported that inconsistent results were obtained using the ^{22}Na partitioning assay to determine monensin levels in tissues, and disputed the findings of Fahim and Pressman (1981) and Pressman et al. (1981). No published accounts of lasalocid metabolism are available, but it is also assumed to be rapidly absorbed by tissues, due to its high lipid solubility.

The effects of lasalocid and monensin upon altering cation flux across cell membranes may explain, in part, the improvement in animal energetics seen when these compounds are fed. _In vitro_ studies have demonstrated that ion pumping, primarily of Na^+ and K^+, comprises a major part of maintenance energy in ruminants (Milligan et al., 1985). Experiments using ovine skeletal muscle, duodenal epithelium and liver have shown 20% of maintenance energy is used to attain Na^+ and K^+ transport across plasma membranes. Active Ca^{2+} transport uses additional energy, although less than 10% of energy is needed for maintenance. Energy use is also increased by lactation, growth, increased feed intake and exposure to cold. The ability of ionophores to alter cation flux may therefore alter energy needs to achieve cation transport.

Monensin has been demonstrated to increase Na^+ levels in guinea pig atrial cells (Glitsch et al., 1970) and rat pheochromocytoma cells (Boonstra et al., 1981), and increase K^+ efflux from rat renal papilla minces (Knapp et al., 1977). Calcium levels have increased in guinea pig and sheep brain synaptosomes (Gill et al., 1981; Coutinho et al., 1983) following exposure to monensin. Monensin has also been shown to decrease phosphate uptake by rabbit brain synaptosomes (Salamin et al., 1981). Lasalocid has been demonstrated to increase Ca^{2+} influx into rabbit taena coli and increase the intracellular release of Ca^{2+} from rabbit aorta (Ohashi et al., 1983).

Effects of Ionophores on Mineral Metabolism.

The ability of lasalocid and monensin to alter the flow of ions into animal tissues suggests that these compounds may alter mineral metabolism in animals receiving ionophores in the diet. This possibility was first investigated in poultry, which are fed ionophores to prevent coccidiosis. Hurst et al. (1974) found that monensin in the diets of broiler chicks did not alter the NaCl requirement. Damron and Harms (1981), using broiler chicks fed with or without 121 ppm monensin for 56 d, indicated that monensin had no effect upon Na requirement. However, the growth depression of chicks, often occurring during monensin supplementation, was overcome by the addition of .3% K as K_2CO_3 in corn-soy and fishmeal diets (Cervantes et al., 1982). The authors suggested that this growth improvement may be due to an improved electrolyte balance of Na, K, and Cl, which was set awry by monensin's effect of increasing intracellular Na^+ and decreasing intracellular K^+ concentrations. Similar results have been demonstrated by the addition of .2% K using magnesium-potassium sulfate in the diet to prevent monensin-induced growth depression of broiler chicks (Charles and Duke, 1981).

Elsasser (1984) was the first to directly question whether ionophores alter ion availability in feedstuffs and supplements for ruminants. He reported that 10 and 30 ppm monensin decreased serum Ca levels and increased serum inorganic P concentrations in sheep fed high-concentrate diets, although lasalocid at these levels had no effect. Elsasser (1984) also examined liver concentrations of Cu, Fe and Zn in wethers fed a high-concentrate diet, with or without 30 mg lasalocid or monensin daily. Liver biopsies were taken after 2 mo feeding, and following 10 d of $CuSO_4$ drench of 100 mg Cu/d. Lasalocid and monensin increased liver Cu, both before and after $CuSO_4$ dosing. The author also examined direct effects of ionophores upon mineral absorption from the digestive tract, using broiler intestinal tissue. Chickens were fed no ionophore, 121 ppm lasalocid, or 121 ppm monensin, or were infused 1.5 x 10^{-4}M lasalocid and monensin through an intestinal loop. Metabolism of duodenally-infused ^{59}Fe, ^{64}Cu or ^{45}Ca was examined. Lasalocid increased mucosal counts following ^{59}Fe administration, while monensin decreased radioactive counts.

Starnes et al. (1984) reported on metabolism of minerals in ruminants fed ionophores. Growing steers were fed a high energy, basal diet with no ionophore, 33 ppm lasalocid, or 33 ppm monensin. Both lasalocid and monensin increased apparent absorption of Na, Mg, and P, and increased Mg and P retention. Metabolism of K and Ca was not altered by addition of ionophore to the diet. Lasalocid and monensin decreased soluble K and Ca in ruminal fluid. Ruminal osmolality decreased when ionophores were fed. Serum Cu and Zn concentrations increased when lasalocid and monensin were fed. No significant differences in mineral metabolism were found between lasalocid and monensin. Decreased ruminal fluid K levels found by these workers contradict an earlier report by Lemenager et al. (1978b), who found a mean increase in ruminal K concentration when monensin was added to extruded urea-grain and soybean meal supplements for cows.

Other reports have indicated altered mineral status of ruminants fed lasalocid and monensin. Edlin et al. (1984) fed 96 finishing steers a high-concentrate diet containing no ionophore, 30 g/ton lasalocid, 30 g/ton lasalocid with 9 g/ton tylosin, or 30 g/ton monensin with 9 g/ton tylosin. All ionophore treatments decreased serum K at 28 d, and increased serum inorganic P at 56 d. Costa et al. (1985) reported that 125 mg/d monensin, fed to steers receiving _ad libitum_ hay and straw, increased Zn retention 52.4%, and tended to increase Se retention, as measured using a Compton Whole Body Counter, following oral doses of $Na_2{}^{75}SeO_3$ or $^{65}ZnCl_2$. A similar effect of monensin upon Se status had been reported by Anderson et al. (1983). Pregnant ewes were fed a control diet of silage and barley grain alone, with 10 mg monensin•hd^{-1}•d^{-1}, with 5 mg Se injected at 0 and 4 wk, or with 10 mg monensin fed and 5 mg Se injection. At parturition, both monensin and Se increased glutathione peroxidase levels three-fold over control-fed animals, while monensin with Se injections had an additive effect. Similar results were reported in lambs from ewes at 0, 4, 8 and 12 wk of age. Serum Se values in lambs mimiced the enzyme results. Galitzer et al. (1986) examined blood chemistry and tissue changes in cattle fed toxic levels of lasalocid and monensin. Serum inorganic P increased, while serum Ca, Cl and K decreased 24 h after animals received 50 and 100 mg/kg body weight lasalocid, or 25 mg/kg body weight monensin.

Kirk et al. (1985a,b) reported on the effects of monensin upon mineral metabolism in sheep. Eighteen wether lambs were used in two metabolism trials, and fed a 70% concentrate diet, with or without 20 ppm monensin. Monensin decreased Na retention 86.2%, while apparent absorption of K increased 16.7%, and K retention increased 52.6% when monensin was fed. Monensin addition to the diet also increased Mg retention 42%. Apparent absorption of P increased 40% and P retention increased 26.8%. Apparent absorption and retention of Zn increased 50% and 45%, respectively, when monensin was fed. Calcium metabolism was not altered, although urinary Ca excretion decreased 60% when monensin was added to the diet. Ruminal fluid Zn decreased 33% when monensin was fed.

These workers also examined changes in tissue mineral levels in response to feeding monensin (Kirk et al., 1985a,b). Ileal Na concentration decreased 13.8%, and liver and bone Ca decreased 45.4% and 2.9%, respectively. Previously, Vijchulata et al. (1981) reported increased liver P levels in steers fed dried citrus pulp and cage layer manure with monensin, compared to the diet without this ionophore. Masek et al. (1985) fed steers green-chopped fodder or haylage and silage, with or without monensin, and examined tissue mineral levels. Monensin decreased liver Cd, longissimus dorsi Cd and Cu, diaphragm Cd and Zn, and increased l. dorsi Mn and diaphragm Fe.

The effects of ionophores upon mineral status have also been investigated in grazing ruminants. Spears and Harvey (1984) fed 72 growing steers, in a 126-d grazing study, with .91 kg corn containing no ionophore, 200 mg lasalocid$\bullet$hd$^{-1}\bullet$d^{-1}, or 300 mg lasalocid$\bullet$hd$^{-1}\bullet$d^{-1}. Steers receiving lasalocid showed decreased serum Mg at 28, 56 and 112 d, and decreased serum K at 112 d, while serum Ca and Na were not affected. Chirase et al. (1988b) fed 32 mature, pregnant beef cows grazing oat pasture .25 kg ground corn with or without 200 mg lasalocid•hd^{-1}•d^{-1}. Lasalocid supplementation reduced the rate of decline in serum Mg prior to parturition from .0051 mg•dl^{-1}•d^{-1} to .0025 mg•dl^{-1}•d^{-1}. Serum Ca increased and serum Na decreased when lasalocid was fed during mid-lactation. Spears et al. (1987) conducted a metabolism study with steers fed green-chopped, tall fescue with no ionophore, 200 mg monensin, 100 mg lysocellin (an experimental ionophore), or 200 mg lysocellin daily. Monensin significantly increased the apparent absorption of Ca, Mg, P and K, and increased Ca and K retention. Ruminal fluid soluble Ca decreased, while soluble ruminal fluid P increased when lysocellin was fed.

Research to determine the physiological mechanisms involved in ionophore-induced alteration of mineral metabolism has recently been conducted. Armstrong et al. (1988) conducted two experiments to determine changes in circulating minerals at 15 min intervals, in response to an intravenous (i.v.) dose of ionophores. In the first experiment, heifers displayed decreased serum K and Mg throughout the time following i.v. dose of 18 mg monensin. In a second experiment, steers dosed with 40 mg monensin showed decreased serum Mg and P from 30 to 120 min, but values returned to levels of animals receiving ethanol, control dosage at 240 min. Plasma K levels at 60 min decreased with monensin, compared to ethanol- and 40 mg lasalocid-dosed steers.

Greene et al. (1988) examined the site and level of mineral digestion in duodenal and ileal cannulated steers fed a 70% concentrate diet with or without 25 ppm monensin. Feeding monensin increased the apparent absorption of Mg (18.4 vs. 32.5%), with a 52% increase in absorption from the preintestinal region. Secretion of Mg into the small intestine increased with monensin. No change was seen in Ca absorption. Apparent absorption of Zn increased (26.1 vs. 32.9%) with monensin, with absorption occurring primarily in the preintestine (Greene et al., 1986a). Therefore, monensin did not alter the primary site of absorption of these minerals, although the overall extent of Mg and Zn absorption was increased.

The effects of lasalocid and monensin upon in vitro digestive tract tissue mineral absorption has also been reported (O'Connor and Beede, 1986). Bovine duodenal tissue was placed in a Ussing chamber, and 0, 2.5, 5.0, or 10.0 ppm lasalocid or monensin were added to the mucosal bath. Lasalocid induced a linear decrease in mucosal Ca levels, decreasing 10.9% for 10.0 ppm vs. no lasalocid. Monensin resulted in a 7.6% decrease in mucosal Na, and 12.6% decrease in mucosal Mg for 10.0 ppm, compared to 0 ppm. These findings indicate increased absorption of Ca with lasalocid and increased absorption of Na and Mg with monensin in duodenal tissue.

Johnson et al. (1987) examined the net absorption of minerals from the gastrointestinal tract of steers fitted with catheters in the portal vein, mesenteric vein and femoral artery, and fed with or without 300 $mg \bullet hd^{-1} \bullet d^{-1}$ monensin. Monensin increased arterial Ca levels from 10.5 mg/dl to 11.3 mg/dl. Portal inorganic P levels decreased from 6.70 to 7.72 mg/dl, while portal Na and K increased with monensin. Portal-arterial differences were recorded for Ca and Mg, with an increased net absorption of Mg when monensin was fed. Monensin did not alter blood flow.

Effects of Ionophores Upon Ruminant Performance.

Monensin has been shown to increase feed efficiency in feedlot cattle by maintaining gains equal to unsupplemented animals, while decreasing feed intake (Brown et al., 1974; Perry et al., 1976; Raun et al, 1976). Improvements in feed efficiency have also been reported in sheep fed high-concentrate diets supplemented with monensin (Calhoun et al., 1979; Joyner et al., 1979). Monensin is also effective in increasing live weight gain, as well as improving feed efficiency of cattle on high-forage regimens (Potter et al., 1976a).

Lasalocid has been demonstrated to increase rate of gain and feed efficiency of cattle fed high-concentrate diets, compared to animals fed these diets with no ionophore, or with equal levels of monensin (Brethour, 1979; Brown et al., 1979; Berger et al., 1981). Lasalocid has also been shown to increase rate of weight gain in pasture-fed cattle (Spears and Harvey, 1984), and increase rate of gain and feed efficiency of cattle receiving high forage diets (Brown et al., 1979; Guitterez et al., 1982). This ionophore also increases feed efficiency and rate of gain of fattening lambs (Horton and Stockdale, 1981).

Lasalocid and monensin appear to alter the energetic efficiency of ruminants. Owens and Gill (1981) fed steers whole shelled corn with 0, 20, or 30 g/ton lasalocid or monensin. Lasalocid and monensin increased the metabolizable energy content of the diet 5.1% and 2.4%, respectively. Fecal starch was decreased for lasalocid-fed, compared to control- and monensin-fed steers. Delfino et al. (1988) found lasalocid lowered the NEm requirement of cattle fed a 90% concentrate diet, with no change in NEg. They related improved animal gain and feed efficiency to an increase in the metabolizable energy density of the diet. Although heat production was increased 7% with lasalocid feeding, methane loss was decreased. Byers (1980) found monensin decreased energy maintenance requirements, and(or) allowed more efficient utilization of the net energy of the diet for maintenance. He further reported that the rate of protein deposition increased, while the rate of fat deposition decreased in steers fed monensin.

Lasalocid and monensin have been effective in prevention and(or) treatment of nutritional diseases. Both ionophores have been shown to prevent lactic acidosis in grain-engorged cattle, with greater reduction in lactate production with lasalocid (Nagaraja et al., 1981). Dennis et al. (1981) found lasalocid and monensin decreased growth of lactate-producing bacteria, but did not alter growth of lactate-users in the rumen. Lasalocid decreased the incidence of bloat 92% and monensin decreased it 64% in cattle fed high levels of grain (Bartley et al., 1983). Monensin was more effective in preventing legume bloat, decreasing incidence 66%, while lasalocid decreased legume bloat 26%. Katz et al. (1986) reported similar results, and credited the effect of monensin to decrease protozoal numbers and an associated decrease in gas production and slime. Lasalocid was relatively ineffective in reducing protozoal numbers.

Cation x Ionophore Interactions on Ruminant Performance and Metabolism.

Current research examine the possible alteration of ionophore effects upon ruminant performance and nutrient metabolism by changing dietary cation levels. Work has been primarily reported with diets of varied K and Na levels fed with ionophores. Funk et al. (1986) examined the effects of lasalocid fed with two levels of dietary K upon performance and digestion in lambs. Sixty-four lambs were fed a 65% concentrate diet containing 1.1% and 0.9% K, with 21.3 mg lasalocid/kg, and 2.1% or 2.5% K, with 20 mg lasalocid/kg. There was a significant K x lasalocid interaction for feed intake, with increased consumption for lasalocid-fed animals receiving high K, compared with lower K in the diet. No interaction was found in daily gains. In a second trial, abomasal samples were taken at slaughter from lambs receiving these diets to determine digestibility, using indigestible ADF as a marker. A K x lasalocid interaction was found for NDF digestibility, with increased digestibility in lambs fed lasalocid at low dietary K. A K x lasalocid interaction was also recorded for ruminal acetate and propionate molar percentages. Lasalocid decreased acetate and increased propionate levels in samples from lambs fed lasalocid with 1.1% K, vs 1.1% dietary K alone. However, lambs receiving high K diets had no difference in VFA molar percentages if fed with or without lasalocid. Blood urea nitrogen (BUN) levels also displayed a K x lasalocid interaction, with lasalocid decreasing BUN at low dietary K, but increasing it with high K in the diet.

Gay et al. (1987) reported no K x lasalocid interactions in performance of yearling steers fed a 70% concentrate diet with .71%, .90%, or 1.1% K, with or without 300 mg lasalocid$\cdot hd^{-1} \cdot d^{-1}$. Doran et al. (1986) also found no K x monensin interaction in ruminal buffering capacity, N, K and total VFA of ruminally-cannulated steers fed a 63% milo diet containing .64% or .86% K, with or without 27 ppm monensin. No K x monensin interaction was found for rate of solid or liquid phase passage, _in situ_ DM disappearance, or ruminal NH_3, although valerate molar proportion showed a K x monensin interaction. Oscar et al. (1986) have reported increased feed intake and decreased feed efficiency in steers receiving 33 ppm lasalocid with 2.0% K, compared with lasalocid and .95% K.

Alteration of dietary monovalent cation levels may alter ionophore induced changes in mineral metabolism in ruminants. Spears and Harvey (1987) fed Angus steers a high-concentrate diet with .25% Na and .5% K, .05% Na and .5% K with 22 ppm lasalocid, .25% Na and .5% K with 33 ppm lasalocid, .05% Na and 1.4% K with 33 ppm lasalocid, or .25% Na and 1.4% K with 33 ppm lasalocid. Although no changes in performance were reported, significant changes in mineral status were noted. Lasalocid increased erythrocyte K, decreased soluble Mg and Cu in ruminal fluid, and decreased serum Zn and inorganic P at 90 d, compared with steers receiving the same dietary Na and K levels without lasalocid. Increasing Na in the diet of lasalocid-fed steers, from .05% to .25%, increased the molar percentage of ruminal fluid acetate at 28 d and 90 d, and decreased ruminal fluid propionate at 90 d. Increasing K from .5% to 1.4% decreased soluble Na concentrations in the ruminal fluid, and increased ruminal fluid soluble K, when fed with lasalocid. Steers fed lasalocid had decreased levels of Na and increased K in the ruminal fluid when 1.4% K was fed, rather than .5% K. Lasalocid decreased liver K, Fe, and Zn, heart K and Mg, and muscle P, compared to steers fed the same Na and K levels with no lasalocid.

Kelley and Preston (1986), used regression analysis for steers fed .24%, .40%, .60% or .73% K, with or without 200 mg monensin$\bullet$hd$^{-1}\bullet$d^{-1}. They determined that monensin increased metabolic fecal K, decreased endogenous urinary K, and greatly increased K retention/K intake, although not changing the K maintenance requirement. Greene et al. (1986b) also examined a K x monensin interaction in ruminally-cannulated sheep fed an 80% concentrate diet with 0 or 20 mg/kg monensin. Sheep were infused, twice daily, with KCl solution to administer 0, 7.6 or 31.6 g additional K/d. Monensin decreased fecal Mg excretion, increasing Mg apparent absorption. A K x monensin interaction was reported for ruminal fluid acetate, propionate and acetate:propionate ratio, with increased effectiveness of monensin at higher infused K levels. Monensin increased apparent absorption of K and Ca, and increased Ca retention. Serum Na levels increased 15% when monensin was fed. Monensin did not alter Na or P absorption or retention.

Chirase et al. (1986) supplemented mature, pregnant beef cows grazing oat pasture with .25 kg ground corn containing 0 or 200 mg lasalocid•hd^{-1}•d^{-1}. Animals also had access to a 6% or 18% Ca mineral mix, free-choice. Cows receiving lasalocid consumed more mineral supplement (16.5 g/d vs 12.6 g/d) than cows not receiving lasalocid, with a resultant increase of 17.8% in serum P, and a decrease in serum Ca/P ratio. Wethers receiving oat hay and a corn-soybean meal supplement with lasalocid at 0 or 20 mg/kg and Ca at .067% and 1.3% were used in a switchback design trial to further examine a Ca x lasalocid interaction (Chirase et al., 1987). With low additional Ca in the diet, lasalocid did not alter serum Ca, while feeding high Ca with lasalocid decreased serum Ca 19.1%, vs compared to high supplemental Ca alone.

Grings and Males (1988) found that Mg supplement source may alter the effects of a Mg x monensin interaction. Cows were fed ammoniated straw and bromegrass haylage after calving, with a protein supplement, including 7.5 g/kg MgO or 11 g/kg $MgSO_4$, with or without 1 g/kg monensin. Prior to calving, MgO with monensin decreased serum Mg levels compared to MgO alone, while no change was noted for the $MgSO_4$ with monensin, post-calving.

Sappington et al. (1987) fed wethers and ewe lambs a basal diet containing 4.4 mg Cu/kg with or without 10 mg/kg Cu added in a 2 x 4 factorial arrangement, with no ionophore, 27.5 mg/kg monensin, 27.5 mg/kg lasalocid, or 27.5 mg/kg lysocellin. Although no Cu x ionophore interaction was discovered, all ionophores increased liver Cu concentrations, particularly monensin, which also increased serum aspartate aminotransferase levels. Lemarie et al. (1987) found no Cu x lasalocid interaction for performance or tissue Cu levels in crossbred lambs fed an 88% concentrate diet containing 0 or 33 ppm lasalocid, with 10, 20 or 30 ppm Cu.

Oscar et al. (1987) examined a possible Ni x monensin interaction in steers. Animals were fed a corn-cottonseed hull based diet containing .30 mg/kg Ni with a 2 x 2 factorial array of treatments, including Ni supplementation at 0 or 5 mg/kg and monensin at 0 or 33 mg/kg. A Ni x monensin interaction was found in ruminal epithelial urease activity. Monensin increased urease activity when no Ni was added, but had no effect with Ni supplementation. However, Starnes et al. (1984) had previously reported decreased ruminal urease activity in monensin-fed steers. Monensin has been demonstrated to inhibit Ni uptake by Methanobacterium bryantii (Jarnell and Sprott, 1982). Urease is believed to be a metalloenzyme containing Ni. Therefore, monensin would be expected to decrease urease activity.

JOURNAL ARTICLE I.

EFFECTS OF LASALOCID AND MONENSIN UPON PERFORMANCE AND MINERAL STATUS OF GRAZING STEERS

ABSTRACT

Two 84-d trials were conducted to determine the effects of feeding supplemental lasalocid and monensin upon performance and mineral status of steers grazing grass-legume pastures. In trial 1, 30 crossbred steers were allotted to six pastures, each containing five steers. In trial 2, 48 crossbred steers were allotted to nine pastures, with six steers grazing each of six pastures, and four steers on each of three pastures. In each trial the groups of cattle were group-fed in pastures, 863 g ground corn and 45 g trace mineralized salt with 1) no ionophore, 2) 200 mg lasalocid, or 3) 150 mg monensin per head daily. Initial animal weights, jugular blood samples, urine samples, and liver biopsies were obtained at d 0, prior to administration of supplement. Live weights, blood, and urine samples were obtained at d 14, 28, 42, 56, 70 and 84. Final liver biopsies were obtained at d 84. Animal groups were rotated among pastures within each replication every 28 d. Pasture forage samples were collected at 7 d intervals. Monensin tended to increase daily gains of steers, but lasalocid had no effect upon performance in both trials. Feeding lasalocid and monensin increased ($P<.05$) serum Ca concentration in trial 1, and tended to increase it in trial 2. Urinary excretion of Ca increased ($P.05$) when ionophores were fed in trial 1. Serum inorganic P concentration was increased ($P<.10$) when ionophores were fed, during trial 2. Serum Mg concentration was reduced ($P<.01$) in steers receiving

monensin, compared to those fed lasalocid, in trial 1. Feeding ionophores decreased ($P<.05$) serum Mg during trial 2. Urinary excretion of Mg increased ($P<.10$) when ionophores were fed during trial 2. Urinary excretion of Na increased ($P<.05$) when ionophores were fed in trial 1, and tended to increase in trial 2. Serum Cu concentration was reduced ($P<.05$) when feeding monensin, compared to lasalocid in trial 2. Steers fed monensin had decreased serum Zn ($P<.05$), compared to steers fed lasalocid, in trial 2. Liver Zn concentration decreased when lasalocid was fed, and increased when monensin was fed during trial 1 ($P<.10$). Feeding lasalocid and monensin in supplements for grazing steers appears to alter mineral status of these animals. However, inconsistent changes in mineral status may result from nutrient or environmental interactions with ionophores.
(KEY WORDS: Lasalocid, Monensin, Mineral Status, Performance, Steers, Pasture)

INTRODUCTION

Lasalocid and monensin are carboxylic ionophores, often added to supplements for grazing cattle. Lasalocid has been shown to increase rate of weight gain in pasture-fed cattle (Spears and Harvey, 1984), and increase rate of gain and feed efficiency of cattle fed high-forage diets (Brown et al., 1979; Guitterez et al., 1982). Monensin is also effective in increasing live weight gain, as well as improving feed efficiency of cattle receiving high-forage regimens (Potter et al., 1976).

The improvement in cattle performance is associated with an energetically favorable shift in the rumen fermentation pattern, resulting in decreased acetate to propionate ratio (Brown et al., 1979; Richardson et al., 1976; Wallace et al., 1980; Guitterez et al.,1982). The basic effect of these ionophores, however, is to alter the flow of cations across cell membranes (Pressman, 1976). Lasalocid has been demonstrated to transport Na^{+}, K^{+}, Ca^{2+} and Mg^{2+}, while monensin carries Na^{+} and K^{+}. Feeding ionophores has been demonstrated to alter mineral metabolism in cattle (Starnes et al., 1984) and sheep (Kirk et al., 1985a,b), presumably due to the ability to carry cations.

The effects of ionophores upon mineral status of grazing rumiants has been investigated. In a 126-d study, Spears and Harvey (1984) fed 72 growing steers .91 kg corn containing no ionophore, 200 mg lasalocid$\bullet$hd$^{-1}\bullet$d^{-1} or 300 mg lasalocid$\bullet$hd$^{-1}\bullet$d^{-1}. Steers receiving lasalocid had decreased serum Mg at 28, 56 and 112 d, and decreased serum K at 112 d, while serum Ca and Na were not affected. Chirase et al. (1988) fed 32 mature, pregnant beef cows, grazing oat pasture, .25 kg ground corn with or without 200 mg lasalocid$\bullet$hd$^{-1}\bullet$d^{-1} Lasalocid supplementation reduced the rate of decrease in serum Mg, prior to parturition. Serum Ca increased and serum Na decreased when lasalocid was fed during mid-lactation. Spears et al. (1987) conducted a metabolism study using steers fed green-chopped, tall fescue with no added ionophores, 200 mg monensin, 100 mg lysocellin (an experimental ionophore), or 200 mg lysocellin/d. Monensin significantly increased the apparent absorption of Ca, Mg, P and K, and increased Ca and K retention. Ruminal fluid soluble Ca decreased, while ruminal fluid soluble P increased when lysocellin was fed.

Previous studies to determine the effects of ionophores upon mineral status of grazing ruminants have not examined differences between lasalocid and monensin, nor have they examined effects of ionophores upon trace element status in ruminants. This study was conducted to determine the effects of lasalocid and monensin upon performance, and Ca, P, Mg, Na, K, Cu, Fe and Zn status of grazing steers.

EXPERIMENTAL PROCEDURE

Two 84-d trials were conducted to determine the effects of lasalocid and monensin upon performance and mineral status of steers grazing grass-legume pastures. Ample forage was available at all times during both trials. In trial 1, 30 Angus x Hereford and exotic crossbred steers were blocked according to weight and breeding and allotted to two replications of three pastures, each containing five steers. In trial 2, 48 Angus x Hereford and exotic crossbred steers were blocked by weight and breeding and allotted to three replications of three pastures. Six steers grazed each of six pastures, and four steers were placed in each of three pastures. In both trials, animals were group-fed, in pastures, 863 g ground corn and 45 g trace mineralized salt$\cdot$hd$^{-1}\cdot$d^{-1} and the groups within each replicate were allotted to 1) no ionophore 2) 200 mg lasalocid, or 3) 150 mg monensin. Mineral content of supplements used in trials 1 and 2 are presented in Table 1. Initial animal weights, jugular blood samples, urine samples, and liver biopsies were obtained at d 0, prior to administration of supplement. Live weights, blood and urine samples were obtained at d 14, 28, 42, 56, 70 and 84. Liver biopsies were taken at d 84. Animal groups were rotated among pastures within each

TABLE 1. AVERAGE MINERAL CONCENTRATIONS OF SUPPLEMENTS AND PASTURES[a].

Item	Trial 1	Trial 2
Supplement		
Calcium, %	.018	.010
Phosphorus, %	.275	.281
Magnesium, %	.116	.085
Potassium, %	.370	.352
Sodium, %	2.66	2.34
Copper, ppm	14.6	21.6
Iron, ppm	132	120
Zinc, ppm	156	173
Pasture		
Calcium, %	.476	.427
Phosphorus, %	.300	.272
Magnesium, %	.272	.239
Potassium, %	2.33	1.54
Sodium, ppm	181.5	91.0
Copper, ppm	5.94	3.68
Iron, ppm	271	149
Zinc, ppm	28.2	21.8

[a]Dry basis.

replication every 28 d. Pasture forage samples were collected at 7 d intervals. Mineral content of collected forage from trial 1 and 2 are presented in Table 1.

Blood samples were obtained by jugular puncture. Blood was centrifuged, serum was removed, and frozen for subsequent analysis. Urine samples were obtained using mechanical stimulation. Urine was filtered through ashless filter paper and frozen for subsequent analysis. Liver biopsies were rinsed with deionized water and frozen prior to freeze-drying. Forage samples were dried in a 70 C forced-air oven and ground through a 1 mm screen in a stainless steel Wiley mill. Liver and forage samples were wet-ashed in nitric and perchloric acids for mineral determination (Hern, 1979)

Serum, urine, liver and forage samples were analyzed for Ca, Mg, Cu, Fe and Zn concentration by atomic absorption spectrophotometry, and Na and K by flame emission spectrophotometry. Samples were diluted using $LaCl_3$ solution for determination of Ca and Mg. Phosphorus was determined by the colorimetric procedure of Fiske and Subbarow (1925). Creatinine concentration was determined in urine (Oser, 1965).

Data were analyzed using the General Linear Model procedure of the Statistical Analysis System (SAS, 1986). The model used for overall variables in trial 1 was: Y = u + a(replication) + b(block within replication) + c (dietary treatment) + d(period) + e(replication x period) + f(dietary treatments x period) + experimental error. The model used for overall variables in trial 2 was: Y = u + a(replication) + b(dietary treatment) + c(replication x dietary treatment) + d(period) + e (dietary treatment x period) + experimental error. Daily gains of steers during the preceding wintering period were used as covariates in trial 2. Replication and dietary treatment effects were tested by replication x dietary treatment. Examination of overall variables excluded data obtained at d 0. Variables at each sampling day were analyzed by the above models, omitting effects due to period and interactions including period. Orthogonal contrasts were, control- vs. ionophore-fed steers and lasalocid- vs. monensin-fed steers. Dunnett's test for treatment mean comparison to controls was conducted when ionophores had opposite effects, compared to controls on the variables examined (Zar, 1984).

RESULTS AND DISCUSSION

There were no statistically significant effects of feeding supplemental lasalocid or monensin upon performance of grazing steers, in trials 1 and 2 (Table 2). Feeding monensin tended to increase daily gain of steers, but feeding lasalocid had no effect upon weight gain in either trial. Daily gains of grazing steers were similar in both trials. These reports are at variance with previously published data. Lasalocid has been demonstrated to increase rate of weight gain in pasture-fed cattle (Spears and Harvey, 1984), and increase rate of gain and feed efficiency of cattle fed high-forage diets (Brown et al., 1979; Guitterez et al., 1982). Monensin has also been shown to increase live weight gain and feed efficiency of cattle receiving high-forage diets (Potter et al., 1976).

In trial 1, feeding lasalocid and monensin increased ($P<.05$) overall serum Ca concentration by 2% in grazing steers, compared to feeding no ionophore (Table 3). At d 56, serum Ca concentration was

TABLE 2. EFFECTS OF LASALOCID AND MONENSIN UPON PERFORMANCE OF GRAZING STEERS.

Item	Ionophore			SE
	None	Lasalocid	Monensin	
	kg			
Trial 1				
Initial wt.	310	305	306	4.9
Final wt.	372	368	374	6.5
Daily gain	.74	.74	.81	.05
Trial 2				
Initial wt.	272	273	274	5.0
Final wt.	332	334	342	6.1
Daily gain	.72	.73	.82	.04

TABLE 3. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM MINERAL CONCENTRATION IN GRAZING STEERS.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Trial 1	mg/100 ml			
Calcium[a]	9.36	9.58	9.54	.07
Inorganic P	8.52	8.26	8.46	.16
Magnesium[b]	1.81	1.84	1.72	.03
Sodium	247.7	256.5	258.9	14.8
Potassium	16.97	16.88	17.41	1.48
	ug/100 ml			
Copper	71.0	73.8	70.6	.23
Iron	197.4	199.2	207.6	.79
Zinc	91.2	88.0	92.7	.24
Trial 2	mg/100 ml			
Calcium	11.23	11.33	11.37	.23
Inorganic P[c]	7.19	7.42	7.40	.10
Magnesium[a]	2.20	2.12	2.16	.02
Sodium	334.1	336.6	338.6	.63
Potassium	21.37	21.06	20.86	.39
	ug/100 ml			
Copper[b]	71.0	76.1	70.5	1.22
Iron[d]	193.4	202.7	192.3	4.26
Zinc[b]	86.4	87.3	83.2	1.25

[a]Control differs from ionophores ($P<.05$).
[b]Lasalocid differs from monensin ($P<.01$).
[c]Control differs from ionophores ($P<.10$).
[d]Lasalocid differs from monensin ($P<.10$).

greater (P<.10) for steers receiving monensin, than steers receiving lasalocid (Appendix Table 1). Feeding lasalocid or monensin also tended to increase serum Ca concentration in grazing steers in trial 2. Increased serum Ca concentration in steers may indicate increased absorption of Ca. Monensin has been demonstrated to increase apparent absorption and retention of Ca in cattle (Spears et al., 1987) and sheep (Greene et al., 1986). O'Conner and Beede (1986) reported a linear decrease in bovine duodenal mucosal bath Ca with increased lasalocid concentration applied to the mucosal bath of a Ussing chamber, indicating increased absorption of Ca. Chirase et al. (1988) found lasalocid supplemented to grazing beef cows increased serum Ca during mid-lactation. However, Elsasser (1984) reported that 10 to 30 ppm monensin decreased serum Ca in sheep receiving high-concentrate diets.

Overall urinary excretion of Ca per 100 mg creatinine increased (P<.05) when ionophores were added to the supplement in trial 1 (Table 4). Lasalocid increased urinary Ca excretion 61%, and monensin increased it 31%. Feeding lasalocid increased urinary Ca excretion, compared to feeding monensin at d 28 (P<.10) and d 56 (P<.05) (Appendix Table 2). At d 70, feeding lasalocid increased (P<.05) urinary excretion of Ca, compared to feeding no ionophore, or monensin. In trial 2, there were no significant differences for overall urinary excretion of Ca. Urinary Ca excretion was greater (P<.05) in steers fed monensin, compared to steers fed lasalocid, at d 28. However, urinary excretion of Ca decreased at d 70 (P<.05) and at d 84 (P<.10) in steers fed monensin, compared to steers fed lasalocid. Kirk et al. (1985b) found no change in apparent

TABLE 4. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY MINERAL EXCRETION IN GRAZING STEERS.

Item	Ionophore None	Lasalocid	Monensin	SE
Trial 1	------mg/100 mg creatinine-------			
Calcium[a]	1.79	2.88	2.34	.25
Magnesium[b]	7.36	10.80	13.28	1.80
Sodium[a]	35.10	111.31	94.09	24.5
Potassium	2052	2253	2247	207.8
	------ug/100 mg creatinine-------			
Copper	15.4	22.2	34.1	6.9
Iron	90	171	342	102.0
Zinc[a]	37.0	59.9	53.7	6.8
Trial 2	------mg/100 mg creatinine-------			
Calcium	3.54	3.07	2.63	1.52
Magnesium[c]	32.11	24.57	45.08	3.97
Sodium	280.5	335.6	346.6	43.7
Potassium	5544	4531	4716	457
	------ug/100 mg creatinine-------			
Copper[a]	151.6	64.3	68.4	34.5
Iron[a]	516.0	303.7	272.5	81.7
Zinc[b]	159.7	94.2	93.0	28.7

[a]Control differs from ionophores ($P<.05$).
[b]Control differs from ionophores ($P<.10$).
[c]Lasalocid differs from monensin ($P<.001$).

absorption or retention of Ca in wethers receiving 20 ppm monensin, but urinary Ca excretion decreased 60% when monensin was fed.

In trial 1, steers fed lasalocid had a slight decrease in liver Ca, while steers fed monensin had a large increase (P<.10) in liver Ca, compared to control cattle (Table 5). Liver Ca concentration was reduced during trial 2 for all treatment groups, with no difference between treatments. Kirk et al. (1985b) found 20 ppm monensin decreased liver Ca 45% in wethers.

There were no overall differences in serum inorganic P concentration in grazing steers fed supplements with or without ionophores in trial 1 (Table 3). At d 42, serum inorganic P decreased (P<.05) in steers fed ionophores (Appendix Table 3). At that time, steers receiving supplemental monensin had greater (P<.001) serum inorganic P concentration than steers receiving lasalocid. In the second trial, serum inorganic P concentration increased 3% (P<.10) when ionophores were supplemented. Adding ionophores to the supplement increased (P<.05) serum inorganic P, at d 42 and 70. Little or no P was detected in urine of steers in trial 1 or 2. Elsasser (1984) reported increased serum inorganic P concentration in sheep fed 10 or 30 ppm monensin. Edlin et al. (1984) found lasalocid and monensin increased serum inorganic P in finishing cattle. Starnes et al. (1984) reported that lasalocid and monensin increased apparent absorption and retention of P in steers. Kirk et al. (1985b) found feeding 20 ppm monensin increased apparent absorption and retention of P in sheep.

Liver P concentration was not altered by feeding ionophores in trial 1 or 2 (Table 5). All treatment groups had a net increase in liver P,

TABLE 5. EFFECTS OF LASALOCID AND MONENSIN UPON LIVER CALCIUM, PHOSPHORUS, AND MAGNESIUM CONCENTRATION IN GRAZING STEERS[a].

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Calcium, ppm				
Trial 1				
Initial Ca	181.0	287.9	201.2	30.8
Final Ca	318.8	285.3	358.7	46.5
Change in Ca[b]	137.9	-2.6	157.5	57.3
Trial 2				
Initial Ca	139.5	164.7	181.7	17.3
Final Ca	120.6	122.8	138.2	15.9
Change in Ca	-17.6	-32.6	-49.7	24.4
Phosphorus, ppm				
Trial 1				
Initial P	495	523	500	23
Final P	565	559	547	27
Change in P	70	37	48	40
Trial 2				
Initial P	1061	1043	1032	45
Final P	1127	1172	1110	35
Change in P	65	126	78	56
Magnesium, ppm				
Trial 1				
Initial Mg	542.4	557.3	552.7	27.6
Final Mg	627.3	608.7	585.6	21.6
Change in Mg	84.9	51.4	32.9	40.4
Trial 2				
Initial Mg	766.2	680.3	684.7	33.8
Final Mg	559.4	606.0	616.2	26.5
Change in Mg	-199.5	-81.6	-68.7	44.6

[a]Dry basis.
[b]Lasalocid differs from monensin ($P<.10$).

during both trials. Vijchulata et al. (1981) reported increased liver P concentration in steers fed dried citrus pulp and cage layer manure with monensin, compared to steers fed this diet without monensin.

Overall, serum Mg concentration was 6.5% lower ($P<.01$) in steers receiving monensin than in those receiving lasalocid, in trial 1 (Table 3). There was a significant ($P<.10$) day x treatment interaction for serum Mg (Fig. 1). Serum Mg was similar between treatment groups for the first 28 d, then values were usually lower for steers fed monensin. At d 56, feeding monensin decreased serum Mg concentration, compared to controls ($P<.05$) and lasalocid ($P<.01$). At d 70, serum Mg concentration was lower ($P<.05$) in steers fed monensin compared to lasalocid, and tended to be lower compared to controls. In trial 2, serum Mg concentration decreased ($P<.05$), when ionophores were fed, 3.6% with lasalocid and 1.8% with monensin. At d 14, serum Mg concentration was greater ($P<.10$) in steers fed monensin, than in steers fed lasalocid (Appendix Table 4). Serum Mg was reduced ($P<.05$) when ionophores were fed at d 42. Spears and Harvey (1984) found lasalocid decreased serum Mg concentration in grazing steers. Grings and Males (1988) reported that feeding monensin with MgO supplements to pregnant cows, fed wheat straw diets, depressed serum Mg concentration, but monensin with $MgSO_4$ did not alter serum Mg. However, Chirase et al. (1988) found 200 mg lasalocid$\cdot$hd$^{-1}\cdot$d^{-1}, fed to beef cows grazing oat pasture, reduced the rate of decline of serum Mg .0051 mg$\cdot$dl$^{-1}\cdot$d^{-1} to .0025 mg$\cdot$dl$^{-1}\cdot$d^{-1}, prior to parturition. Most research has indicated an increase in apparent absorption and(or) retention of Mg by ruminants receiving ionophores in high-concentrate (Starnes et al., 1984; Kirk et al., 1985b; Greene et al., 1986), and high-forage (Spears

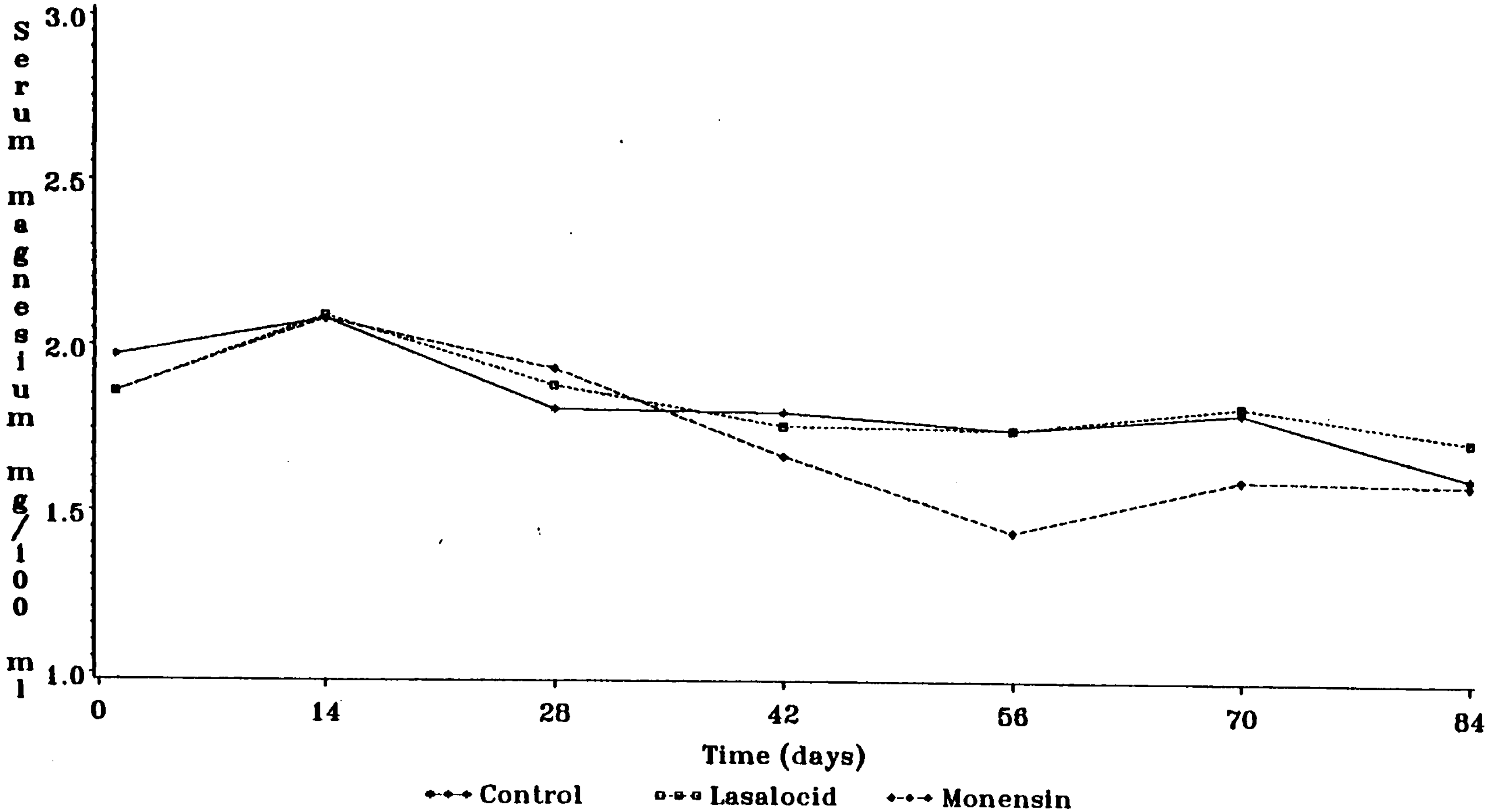

Figure 1. Serum magnesium concentration in grazing steers (Trial 1). Day x treatment interaction (P<.10). Lasalocid differs from monensin (P<.05). SE=.210.

et al., 1987) diets. Johnson et al. (1987) reported a net increase in net Mg absorption, calculated by hepatic artery-portal vein difference, in steers fed monensin. O'Conner and Beede (1986) also reported decreased mucosal bath Mg concentration following monensin addition to bovine duodenal mucosal preparations in a Ussing chamber, indicating increased Mg absorption.

Overall urinary excretion of Mg per 100 mg creatinine increased ($P<.10$) when ionophores were fed in trial 1, 47% with lasalocid, and 80% with monensin (Table 4).In trial 2, overall urinary Mg excretion was 83.5% greater ($P<.001$) by steers receiving monensin, than those receiving lasalocid. Steers fed monensin tended to have greater urinary excretion of Mg than steers fed no ionophore. Monensin increased urinary Mg excretion, compared to lasalocid, at d 14 ($P<.10$), d 42 ($P<.01$) and d 56 ($P<.10$). Magnesium absorbed in excess of needs is excreted through the urine (Ammerman et al., 1972), and may be used as a better indicator of Mg status than serum Mg in ruminants (Halse, 1970). The findings of increased urinary Mg excretion with lasalocid and monensin in trial 1, and tendency toward increased Mg excretion with monensin in trial 2, indicate improved Mg absorption. However, these results do not agree with depressed serum Mg in steers receiving ionophores during these two trials.

All treatment groups displayed a net increase in liver Mg concentration during trial 1 (Table 5), but a net decrease during trial 2. There were no significant effects of treatment upon liver Mg in either trial.

In trial 1, overall serum Na concentration was not significantly different between treatment groups (Table 3). Addition of ionophores to the supplements increased (P<.10) serum Na in grazing steers, at d 70 (Appendix Table 6). At d 84, however, feeding ionophores decreased (P<.10) serum Na. In trial 2, serum Na concentration increased (P<.10) when ionophores were supplemented at d 28. Starnes et al. (1984) found both lasalocid and monensin addition to the diet increased apparent absorption of Na in steers. O'Conner and Beede (1986) found a linear decrease in bovine duodenal mucosal bath Na concentration when increased monensin levels were added to the mucosal bath of tissue preparations in a Ussing chamber, indicating increased Na absorption.

Overall urinary excretion of Na per 100 mg of creatinine by steers increased (P<.05) when ionophores were fed, during trial 1, 3.2-fold with lasalocid and 2.7-fold with monensin (Table 4). Large increases in urinary Na excretion were seen at d 14 (P<.10), d 28 (P<.10), d 42 (P<.05), and d 56 (P<.10), when ionophores were added to supplements (Appendix Table 7). Negative least square means are the result of large standard errors. During trial 2, urinary excretion of Na tended to increase when ionophores were fed. Urinary Na excretion increased when ionophores were fed at d 42 (P<.10) and d 70 (P<.05). Kirk et al. (1985b) reported increased urinary Na excretion and decreased Na retention in sheep fed monensin.

Steers in all treatment groups displayed a net decrease in liver Na concentration, during trial 1, with no significant differences between treatment groups (Table 6). In trial 2, there were no significant differences between supplement treatments for liver Na concentration in grazing steers, although control-fed steers had a mean increase in liver Na, while ionophore-fed steers had a mean decrease in liver Na.

TABLE 6. EFFECTS OF LASALOCID AND MONENSIN UPON LIVER SODIUM AND POTASSIUM CONCENTRATION IN GRAZING STEERS[a].

Item	Ionophore None	Lasalocid	Monensin	SE
Sodium, ppm				
Trial 1				
Initial Na	3829	4096	3669	264
Final Na	3264	3550	3518	134
Change in Na	-565	-546	-151	292
Trial 2				
Initial Na	3689	4240	3295	358
Final Na	3805	3488	3348	365
Change in Na	95	-413	-314	582
Potassium, ppm				
Trial 1				
Initial K	8529	7342	9519	773
Final K[b]	10150	8786	7996	489
Change in K[c]	1621	1444	-152	954
Trial 2				
Initial K	6840	8036	9325	595
Final K	8165	7721	7866	809
Change in K[d]	1708	365	-1087	879

[a]Dry basis.
[b]Control differs from ionophores (P<.05).
[c]Lasalocid differs from monensin (P<.10).
[d]Control differs from ionophores (P<.10).

In trial 1, overall serum K concentration in grazing steers was not altered by feeding supplemental ionophores (Table 3). Monensin increased ($P<.05$) serum K concentration compared to lasalocid, at d 28, and d 56 (Appendix Table 8). However, at d 84, steers receiving monensin had lower ($P<.05$) serum K than those fed lasalocid. In trial 2, serum K concentration decreased ($P<.05$) when monensin was fed, compared to feeding lasalocid, at d 42. At d 84, however, monensin increased ($P<.10$) serum K, compared to lasalocid. Spears and Harvey (1984) found lasalocid decreased serum K at 112 d in grazing cattle. Edlin et al. (1984) reported decreased serum K in finishing steers fed lasalocid or monensin. Kirk et al. (1985b) found monensin increased apparent absorption and retention of K in wethers. Greene et al. (1986) reported increased apparent absorption of K in sheep fed monensin. Kelley and Preston (1986) found monensin increased metabolic fecal K, decreased endogenous urinary K, and greatly increased K retention per unit of K intake, without altering K maintenance requirement in cattle.

Urinary excretion of K per 100 mg creatinine by steers was not significantly altered by the addition of ionophores to the supplement, in trial 1 (Table 4). In trial 2, urinary K excretion was lower ($P<.05$) for steers fed ionophores at d 14 (Appendix Table 9).

In trial 1, final liver K concentration was lower ($P<.05$) when ionophores were fed, 13.4% with lasalocid and 21.2% with monensin (Table 6). Change in liver K differed ($P<.10$) between ionophores, with lasalocid resulting in 1444 ppm increase in liver K, and monensin resulting in a 1523 ppm decrease in liver K. In trial 2, change in liver K was less

(P<.10) in steers fed ionophores, with lasalocid being 78.6% less than controls, and monensin resulting in a net decrease in liver K.

There were no significant differences in serum Cu concentration of grazing steers between treatment groups, in trial 1 (Table 3). Serum Cu concentration, during trial 2, was reduced (P<.01) 7.4% overall, when monensin was fed, compared to lasalocid. At d 14, monensin decreased (P<.10) serum Cu, compared to lasalocid (Appendix Table 10). Starnes et al. (1984) reported increased serum Cu in steers fed lasalocid or monensin.

There were no overall effects of ionophores upon urinary excretion of Cu, in trial 1 (Table 4). At d 42 urinary Cu excretion by steers fed monensin was lower (P<.05) than by steers fed lasalocid (Appendix Table 11). In trial 2, overall urinary excretion of Cu decreased (P<.05) when ionophores were fed, 57% with lasalocid, and 72% with monensin. This was primarily due to a large increase in urinary Cu excretion by control-fed steers at d 14. Monensin decreased (P<.05) urinary Cu excretion 28.3% compared to lasalocid at d 56.

In trial 1, final liver Cu concentration was 58.4% lower (P<.10) in steers receiving monensin than those receiving lasalocid (Table 7). The change in liver Cu was not significantly altered, although steers fed the control and monensin supplements displayed decreased liver Cu, while those fed the lasalocid supplement had increased liver Cu. In the second trial, all treatments resulted in a net increase in liver Cu over the course of the study, with no differences between treatment groups. Elsasser (1984) reported increased liver Cu in sheep receiving 30 mg/d dietary lasalocid or monensin daily. Sappington et al. (1987) also found

TABLE 7. EFFECTS OF LASALOCID AND MONENSIN UPON LIVER COPPER, IRON, AND ZINC CONCENTRATION IN GRAZING STEERS[a].

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Copper, ppm				
Trial 1				
Initial Cu	292.4	432.5	329.0	40.5
Final Cu[b]	166.1	614.6	255.6	141.6
Change in Cu	-126.2	182.0	-73.4	154.7
Trial 2				
Initial Cu	65.9	70.8	59.7	7.9
Final Cu	94.0	84.9	80.5	12.6
Change in Cu	26.5	10.5	20.8	6.8
Iron, ppm				
Trial 1				
Initial Fe	827.8	749.8	448.4	199.6
Final Fe[c]	246.9	348.2	398.1	46.3
Change in Fe	-580.9	-401.6	-50.3	215.5
Trial 2				
Initial Fe	501.0	557.2	505.2	64.8
Final Fe	525.1	480.8	484.5	48.4
Change in Fe	31.6	-76.6	-21.1	75.1
Zinc, ppm				
Trial 1				
Initial Zn	151.8	222.0	148.7	35.5
Final Zn	161.8	166.9	227.8	39.7
Change in Zn[b]	10.1	-55.1	79.2	49.7
Trial 2				
Initial Zn	98.8	115.0	95.9	9.6
Final Zn	75.2	87.3	83.4	5.3
Change in Zn	-22.6	-29.0	-12.4	9.6

[a]Dry basis.
[b]Lasalocid differs from monensin ($P<.10$).
[c]Control differs from ionophores ($P<.05$).

increased liver Cu concentration in sheep receiving 27.5 ppm lasalocid or monensin.

The use of liver Cu concentration as an indicator of Cu status has been challenged (Combs, 1987). Several researchers have reported a poor correlation for liver Cu with Cu deficiency (Mylrea, 1958; Smith and Coup, 1973; Kellaway, 1978). Suttle and McMurray (1983) proposed the use of a combined assay including erythrocyte superoxide dismutase, hair Cu concentration, and plasma Cu concentration as a more accurate indicator of Cu status.

There were no overall treatment effects upon serum Fe concentration in grazing steers, during trial 1 (Table 3). Serum Fe concentration increased ($P<.05$) in steers receiving monensin, compared to those receiving lasalocid, at d 28 (Appendix Table 12). At d 84 steers fed supplements containing ionophores had greater ($P<.10$) serum Fe concentration, than those fed the supplement without ionophores. In trial 2, serum Fe concentration was 5.1% lower ($P<.10$) in steers receiving monensin in the supplement, compared to those receiving lasalocid. Elsasser (1984) reported increased ^{59}Fe uptake by chicken duodenal tissue with lasalocid addition and decreased ^{59}Fe uptake when monensin was added, indicating increased absorption of Fe with lasalocid and decreased absorption with monensin.

Urinary excretion of Fe per 100 mg creatinine was not altered by feeding supplemental ionophores, in trial 1 (Table 4). At d 42 urinary Fe excretion was lower ($P<.05$) when monensin was fed, compared to feeding lasalocid (Appendix Table 13). In trial 2, feeding ionophores reduced ($P<.05$) overall urinary excretion of Fe, 41% with lasalocid, and 47% with monensin. This was primarily due to a large increase in urinary Fe excretion by control-fed steers at d 14. Feeding monensin increased ($P<.10$) urinary Fe excretion, compared to lasalocid, at d 70.

In trial 1, final liver Fe concentration was higher ($P<.10$) in steers fed ionophores, compared to those fed no ionophores, 41% with lasalocid and 61% with monensin (Table 7). All treatment groups displayed a net decrease in liver Fe, with no differences due to treatment. In trial 2, liver Fe was not significantly different between supplements, although control steers had a mean increase in liver Fe, and steers fed ionophores had a mean decrease in liver Fe. Elsasser (1984) found no effects of lasalocid or monensin upon liver Fe concentration in sheep.

There were no significant differences between treatment groups for serum Zn concentration in grazing steers,in trial 1 (Table 3). In trial 2, overall Zn concentration was 5% lower ($P<.01$) for steers fed monensin, compared to lasalocid. At d 70, steers fed monensin had lower ($P<.05$) serum Zn than steers fed lasalocid (Appendix Table 14). Kirk et al. (1985b) reported large increases in apparent absorption and retention of Zn in sheep receiving 20 ppm dietary monensin. Greene et al. (1986) reported a 26.0% increase in apparent absorption of Zn in steers fed 25 ppm monensin. Costa et al. (1985) fed 125 mg monensin/d to steers receiving hay and straw in the diet, resulting in a 52.4% increase in Zn retention, as measured by a Compton Whole Body Counter following $^{65}ZnCl_2$ oral dose.

In trial 1, overall urinary excretion of Zn per 100 mg creatinine increased ($P<.05$) when ionophores were fed (Table 4). Lasalocid increased urinary Zn excretion 62%, and monensin increased it 46%. Monensin increased ($P<.05$) urinary Zn excretion at d 14, but decreased ($P<.05$) it at d 70, compared to lasalocid (Appendix Table 15). At d 70, urinary excretion of Zn increased ($P<.10$) with lasalocid and monensin, compared to controls. In trial 2, overall Zn excretion in the urine decreased ($P<.10$) when ionophores were fed, 41% with lasalocid and 42% with monensin. Feeding ionophores reduced ($P<.05$) urinary excretion of Zn, compared to controls, at d 28 and 42.

In the first trial, change in liver Zn concentration differed ($P<.10$) between ionophores supplemented (Table 7). Liver Zn increased 79.2 ppm when monensin was fed, but decreased 55.1 ppm when lasalocid was fed to grazing steers. In trial 2, liver Zn concentration was not significantly different between treatment groups, with all groups displaying a net decrease in liver Zn. Elsasser (1984) found no effects of lasalocid or monensin upon liver Zn concentration in sheep.

Conclusions. Results from these trials indicate that lasalocid and monensin can alter mineral status of grazing steers. The results also demonstrate that the effects of the ionophores upon mineral status may differ. Inconsistent effects of feeding supplemental ionophores upon mineral status of grazing steers were seen when comparing trials 1 and 2, suggesting that nutrient or environmental effects may interact with ionophores to alter the animal response.

Pasture mineral levels were lower for trial 2 vs trial 1, particularly for Na, K and the trace elements. The effects of ionophores have been demonstrated to be dependent upon dietary levels of K (Funk et al., 1986; Greene et al., 1986; Chirase et al., 1987; Dawson and Boling, 1987; Schwingel et al., 1987), Mg (Chirase et al., 1988), Ca (Chirase et al., 1987), and Ni (Oscar et al., 1987). Therefore, differences in mineral concentration of forage grazed by steers, due to yearly differences, may have altered the physiological response to supplemental ionophores.

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JOURNAL ARTICLE II.

PERFORMANCE, CARCASS CHARACTERISTICS, RUMINAL VOLATILE FATTY ACIDS AND MINERAL STATUS OF FINISHING STEERS FED LOW AND HIGH POTASSIUM WITH OR WITHOUT MONENSIN

ABSTRACT

A 147-d finishing study was conducted with 24 crossbred steers individually fed a high-concentrate diet. Steers were blocked according to weight and breeding, and allotted to low-and high-K diets, with and without monensin, in a 2 x 2 factorial arrangement. Cattle had access to diets from 0800 to 1100 h, and 1600 to 0700 h. Steers were adjusted to diets for 7 d. Blood and ruminal fluid samples were collected at 1100 h on d 0, 28, 58, and 84. Daily gain of steers decreased ($P<.05$) 13.6% when high K was fed. Feed:gain ratio increased ($P<.05$) 9.4% when high K was fed, and decreased ($P<.01$) 13.2% when monensin was fed. Feeding high K increased ($P<.01$) dressing percentage 4.0%. Total ruminal VFA concentration was decreased ($P<.001$) 17.8% with high dietary K. Molar proportion of ruminal propionate increased ($P<.001$) 21.1% and acetate:propionate ratio decreased ($P<.001$) 29.5% when monensin was fed. Ruminal butyrate decreased ($P<.01$) 24.5% and 23.5% when high K and monensin were fed, respectively. Ruminal valerate decreased 47.4% with high dietary K ($P<.001$), and 33.2% with monensin ($P<.01$). Ruminal fluid Na decreased ($P<.001$) 44.7% with high dietary K. Feeding high K increased ($P<.01$) serum K concentration 7.2%, and increased ruminal K ($P<.001$) 3.6-fold. There was a K x monensin interaction ($P<.01$) for serum Mg.

Monensin fed with low K increased serum Mg 2.3%, but monensin with high dietary K decreased serum Mg 8.0%. Serum Mg was decreased ($P<.001$) 12.8% with high dietary K. Ruminal fluid Mg increased ($P<.05$) 15.1% when high K was fed. Serum Ca increased ($P<.001$) 5.0% with high dietary K, and increased ($P<.05$) 2.4% with monensin. Ruminal fluid Ca decreased ($P<.05$) 23.9% when monensin was fed. A K x monensin interaction ($P<.01$) was found for serum inorganic P. Monensin increased serum inorganic P 3.6% with low dietary K, but decreased it 8.3% with high dietary K. Ruminal fluid inorganic P increased ($P<.001$) 12.9% with high dietary K. A K x monensin interaction ($P<.01$) occurred for serum Cu. Monensin increased serum Cu 19.1% when fed with low K, but had no effect when fed with high K. Monensin increased ($P<.01$) serum Cu 9.5%. Serum Fe decreased ($P<.05$) 8.1% when monensin was fed. Ruminal fluid Fe decreased ($P<.05$) 19.9% when high K was fed. A K x monensin interaction ($P<.05$) was found for serum Zn. Monensin increased serum Zn 9.1% with low dietary K, but decreased it 4.3% with high dietary K. Monensin decreased ($P<.05$) ruminal fluid Zn 17.8%. Although there were no K x monensin interactions for steer performance, carcass characteristics, or ruminal VFA content, the effects of monensin upon mineral status of finishing steers appears to be dependent upon dietary K concentration.
(KEY WORDS: Beef Cattle, Monensin, Potassium, Mineral Status, Ruminal Fermentation)

INTRODUCTION

Monensin, a carboxylic ionophore, is added to feedlot cattle diets to improve feed efficiency. This improvement in performance is due primarily to a decrease in ruminal acetate:propionate ratio of cattle fed monensin (Raun et al., 1976). The basic action of monensin is to alter cation transport across cell membranes. (Pressman, 1976). Monensin carries both Na^+ and K^+ across membranes, preferring Na^+ to K^+, 10 to 1. Possibly because of this action, monensin has been demonstrated to alter mineral metabolism in cattle (Starnes et al., 1984) and sheep (Kirk et al., 1985a,b). Dawson and Boling (1984) have demonstrated that greater concentrations of monensin were needed to inhibit Bacteroides ruminicola growth in high-K medium than were required for growth inhibition in low-K media. Recently, Russell (1987) has shown that monensin acts to selectively inhibit Streptococcus bovis by producing a K^+-H^+ antiport, allowing K^+ to leave the cell and H^+ to enter, resulting in depletion of ATP used to expel intracellular H^+. He suggested that monensin transported K^+ vs Na^+ due to the greater K^+-gradient, with higher K^+ concentration within the cells. These findings suggest that the effects of monensin upon rumen bacteria may be decreased by increasing ruminal K^+ content.

Changing K levels have been demonstrated to alter the response of bacterial fermentation to monensin. Greene et al. (1986) found monensin was more effective in decreasing ruminal fluid acetate and acetate:propionate ratio, and increasing ruminal propionate in sheep receiving increased intraruminal infusions of K. Chirase et al. (1987), however, using a continuous culture fermentation system, found no K x monensin interaction for VFA production. Rumpler et al. (1986) reported that increasing dietary K enhanced inhibition of methanogenesis by steers fed monensin. Schwingel et al. (1987) demonstrated lower acetate:propionate ratio with monensin added to in vitro fermentation systems with 150 mM K, than with monensin added with 50 mM K. These positive K x monensin interactions appear to conflict with the findings of Dawson and Boling (1984) and Russell (1987).

Doran et al. (1986b) reported a significant K x monensin interaction for ruminal valerate concentration in steers fed .64% or .86% K, with or without 27 ppm monensin. No K x monensin interaction was found for rumen buffering capacity, ruminal Na, K, total ruminal VFA, rate of solid or liquid phase passage, in situ DM disappearance, or ruminal NH_3 in ruminally-cannulated steers. Greene et al. (1986) found no K x monensin interaction for mineral metabolism in sheep receiving intraruminal infusions of 0, 7.6 or 31.6 g K daily, with or without monensin in the diet.

The existence of a K x lasalocid interaction has been examined for ruminant performance and nutrient metabolism. Funk et al. (1986) reported a significant K x lasalocid interaction for feed intake by fattening lambs, with increased consumption by lambs fed lasalocid with high dietary K, compared to feeding lasalocid with low K. A K x lasalocid interaction was found for NDF digestibility, with increased digestibility in lambs fed lasalocid with high dietary K, compared to those fed lasalocid with low dietary K. Lasalocid decreased blood urea N (BUN) with low dietary K, but increased it with high K in the diet. Oscar et al. (1986) reported increased feed intake and decreased feed efficiency in growing steers receiving 33 ppm lasalocid with 2.0% K, compared to lasalocid with .95% dietary K. However, Gay et al. (1987) reported no K x lasalocid interactions in performance of yearling steers fed a 70%

concentrate diet with .71, .90 or 1.1% K, with or without 300 mg lasalocid•hd^{-1}•d^{-1}. Spears and Harvey (1987) found 33 ppm lasalocid tended to improve gain and feed efficiency of finishing steers fed diets containing .05% Na and .5% K, .25% Na and .5% K, and .05% Na and 1.4% K, but not in steers fed diets with .25% Na and 1.4% K.

The effects of feeding monensin with varied dietary K upon cattle performance has not been investigated. An experiment was conducted to determine the possible existence of a K x monensin interaction for performance and carcass characteristics of finishing steers, as well as ruminal fluid VFA content, and protein and mineral status of these animals.

EXPERIMENTAL PROCEDURE

Twenty-four Angus x Hereford and exotic crossbred steers (avg wt, 334 kg) were used in a 147-d finishing study. The experiment was a randomized block design using a 2 X 2 factorial arrangement of treatments, with two levels of dietary K (.4% and 2.3%) and two levels of monensin (0 and 23 ppm), dry basis. Steers were blocked according to weight and breeding, and assigned to treatments. The diets containing low and high K were formulated to be equal in crude protein and NEg content (Table 8).

Animals were full-fed in individual stalls from 0800 h to 1100 h, and 1600 h to 0700 h, and were placed in open lots with access to water from 0700 h to 0800 h, and 1100 h to 1600 h. Steers were adjusted to the respective diets over 7 d, with the trial beginning after animals were receiving 100% of the diets.

Steers were weighed on two consecutive days at the beginning of the trial to obtain average initial weights, at 14 d intervals, and at the

TABLE 8. COMPOSITION OF LOW AND HIGH POTASSIUM DIETS.

Item	Diet	
	Low potassium	High potassium
Ingredient composition[a]		
Corn grain	70.5	72.6
Corn cob pellets	20.9	14.4
Soybean meal	7.0	7.0
Calcitic limestone	1.1	1.1
Trace mineralized salt	.5	.5
Potassium chloride	---	4.4
Dry matter[a]	87.8	88.3
Crude protein[b]	11.3	11.0
Mineral composition		
Potassium[b]	.40	2.32
Sodium[b]	.14	.17
Magnesium[b]	.15	.14
Calcium[b]	.63	.59
Phosphorus[b]	.26	.26
Copper[c]	6.4	6.0
Iron[c]	203.2	191.6
Zinc[c]	47.7	42.8

[a]Percent as-fed.
[b]Percent of DM.
[c]mg/kg DM.

end of the test. Blood and ruminal fluid were obtained at 1100 h on d 0, 28, 58, and 84. Blood was collected by jugular venipuncture, into tubes containing EDTA or no anticoagulant. Serum was obtained by immediate centrifugation, and frozen for subsequent analysis. Ruminal fluid was obtained by vacuum suction through a stomach tube. The fluid was strained through four layers of cheesecloth, and pH of fluid was determined electrometrically. Samples of the strained ruminal fluid were prepared for analysis of VFA, and ruminal NH_3-N. Ruminal fluid was filtered through ashless filter paper prior to mineral analysis.

Feed samples were obtained every 7 d and wet ashed in nitric and perchloric acids for mineral determination (Hern, 1979). Feed, serum and ruminal fluid samples were analyzed for Ca, Mg, Cu, Fe and Zn concentration using atomic absorption spectrophotometry. Samples were diluted using $LaCl_3$ solution for Ca and Mg determination. Analysis for Na and K was conducted using flame emission spectrophotometry. Phosphorus was determined by a colorimetric procedure (Fiske and Subbarow, 1925). Blood urea N was determined by the procedure of Colombe and Favreau (1963). Ruminal NH_3-N was determined by the procedure of Beecher and Whitten (1970). Serum aldosterone was determined by radioimmunoassay[1]. At the end of the study, steers were slaughtered and carcass data were obtained.

The effects of K, monensin and K x monensin interaction were determined for all variables using the General Linear Model Procedure of

[1]Coat-a-count aldosterone kit, Diagnostics Products Corporation, Los Angeles, CA 90015

the Statistical Analysis System (SAS, 1986). The model used in the GLM procedure to determine overall dietary effects was: Y = u + a(block) + b(day) + c(dietary K) + d(monensin) + e(dietary K x monensin). Significant dietary effects at each sampling were determined using this model, excluding the variable expressing effects due to sampling day.

RESULTS AND DISCUSSION

Daily gains of steers decreased (P<.05) 13.6% when high K was fed (Table 9). Feeding high K increased (P<.05) feed:gain ratio by 9.4%. Ferrell et al. (1983), in a study in which finishing steers were fed diets containing .43, .55, .70, .85, or 1.0% K, reported a quadratic effect of K for daily gain of steers after 150-d, with the poorest performance by animals fed .85% K. Doran et al. (1986a) found a trend toward increased feed consumption and daily gain, with decreased feed:gain ratio in finishing steers fed high K during the hot summer months. Oscar et al. (1986) found that feeding high K to growing steers increased DM intake and feed:gain ratio.

Feeding monensin did not significantly affect daily gain. A 13.5% decrease (P<.10) in DM intake was seen in steers receiving monensin. Feed:gain ratio was decreased (P<.01) 13.2% when the ionophore was fed. The improvements in feed efficiency in finishing steers is consistent with previous reports on cattle fed monensin, which have shown decreased feed intake with gains equal to unsupplemented animals, and a resultant improvement in feed efficiency (Raun et al., 1976; Goodrich et al., 1984).

There were no significant K x monensin interactions for performance of finishing steers. Spears and Harvey (1987) also reported

TABLE 9. PERFORMANCE AND CARCASS CHARACTERISTICS OF FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Item	Dietary treatment: .4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
Initial wt., kg	340.4	334.2	334.1	329.2	8.99
Final wt., kg[a]	548.5	538.1	502.0	517.6	18.19
Daily gain, kg[b]	1.42	1.38	1.14	1.28	.08
DM intake, kg/d[c]	10.63	9.54	9.99	9.10	.47
Feed:gain[bd]	7.58	6.95	8.76	7.22	.31
Carcass wt., kg	333.6	320.8	315.0	323.0	10.6
Dressing %[e]	60.8	59.6	62.7	62.5	.62
Marbling[f]	3.62	3.88	4.04	3.29	.31
Quality grade[g]	10.0	10.5	11.0	9.5	.57
Rib eye area, cm^2	83.03	77.10	79.22	79.22	2.19
Kidney-pelvic-heart fat, %	1.67	1.96	1.79	1.62	.17
Back fat, cm	1.47	1.45	1.32	1.32	.18
Yield grade	2.38	2.60	2.30	2.32	.26

[a]K effect (P<.10).
[b]K effect (P<.05).
[c]Monensin effect (P<.10).
[d]Monensin effect (P<.01).
[e]K effect (P<.01).
[f]Code: 3 = slight; 4 = small; 5 = modest.
[g]Code: 10 = high select; 11 = low choice; 12 = avg. choice.

no significant K x lasalocid interaction on performance of finishing steers. Funk et al. (1986), however, found a K x lasalocid interaction for feed intake by fattening lambs, with greater in DM intake by sheep fed lasalocid with high dietary K, than with low K in the diet.

Feeding high K increased ($P<.01$) dressing percentage 2.2 percentage units. Carcass quality grades ranged from high select to low choice, with yield grades of approximately 2.4. There were no effects due to monensin or K x monensin interaction for carcass characteristics.

High dietary K increased ($P<.10$) ruminal fluid pH 5.7% on d 84 (Table 10). Greene et al. (1983c) demonstrated increased ruminal pH in multicannulated steers receiving KCl in the diet. In the present study, monensin increased ($P<.05$) ruminal fluid pH 9.0% at d 1. Oltjen et al. (1977) reported increased ruminal pH in finishing steers fed monensin.

There was an overall decrease of 17.8% ($P<.001$) in total VFA in ruminal fluid of steers receiving high K, compared with those fed low K (Table 11). High dietary K decreased total VFA at d 28 ($P<.05$) and at d 84 ($P<.001$). Addition of monensin to the diets decreased ($P<.10$) total ruminal VFA at d 1. There was no evidence of a K x monensin interaction for total ruminal VFA. Monensin has been shown to decrease total ruminal VFA *in vitro* (Slyter, 1979; Wallace et al., 1980) and *in vivo* in cattle fed high-concentrate diets (Oltjen et al., 1977; Prange et al., 1978).

Dietary K level did not affect ruminal acetate molar proportion in finishing steers (Table 12). Increased dietary K has been shown to decrease ruminal acetate levels in sheep (Funk et al., 1986; Greene et al., 1986; Grings and Males, 1987). Feeding monensin resulted in an

TABLE 10. RUMINAL pH IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
Overall	5.81	5.92	5.82	5.86	.10
Days					
0[a]	5.91	6.50	5.87	6.35	.20
28	5.80	5.64	5.65	5.74	.18
58	5.69	5.85	5.61	5.57	.20
84[b]	5.68	5.56	6.02	5.79	.13

[a]Monensin effect ($P<.05$).
[b]K effect ($P<.10$).

TABLE 11. TOTAL RUMINAL VFA CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mmol/l				
Overall[a]	93.57	89.00	76.34	73.64	3.85
Days					
0[b]	99.04	80.68	95.54	82.95	7.84
28[c]	102.12	112.19	92.47	73.92	9.88
58	84.40	71.34	62.67	67.75	6.60
84[a]	96.57	93.12	60.40	69.95	4.33

[a]K effect ($P<.001$).
[b]Monensin effect ($P<.10$).
[c]K effect ($P<.05$).

TABLE 12. MOLAR PROPORTION OF RUMINAL ACETATE IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mol/100 mol				
Overall[a]	54.05	48.87	53.97	53.18	.13
Days					
0[b]	60.17	51.87	61.80	55.20	.17
28	52.75	48.80	45.34	48.50	.23
58	46.54	48.78	50.85	51.84	.19
84	52.66	45.26	56.98	57.16	.40

[a]Monensin effect (P<.10).
[b]Monensin effect (P<.01).

overall decrease (P<.10) in ruminal acetate of 5.5%, compared to steers receiving no ionophore. A decrease (P<.01) in ruminal acetate was seen at d 0 for animals fed monensin, after which no consistent effect was observed. Decreased ruminal acetate levels due to monensin have been reported in vitro (Van Nevel and DeMeyer, 1977; Richardson et al., 1976; Slyter, 1979; Wallace et al., 1980), and in vivo in cattle fed high grain diets (Richardson et al., 1976).

There was a trend toward a K x monensin interaction for ruminal acetate (P=.12). Monensin tended to decrease ruminal acetate with low dietary K, but had no effect with high K. Greene et al. (1986) have reported a significant K x monensin interaction for ruminal acetate in sheep receiving monensin with three levels of intraruminal K infusions. These authors reported a greater monensin-induced decrease in acetate levels in sheep receiving 31.6 g K daily than in sheep receiving 7.6 or 0 g K/d. However, Chirase et al. (1987) found no significant K x monensin interaction for acetate production using a continuous culture fermentation system. Funk et al. (1986) reported lasalocid decreased acetate levels with low dietary K, but not with high dietary K in lambs.

High dietary K increased (P<.05) ruminal propionate at d 28 (Table 13). Funk et al. (1986) reported increased ruminal propionate levels in sheep fed high K, but Grings and Males (1987) found no effect of dietary K upon ruminal propionate in sheep. In the present study, feeding monensin increased (P<.001) ruminal propionate at d 0, after which no consistent effect was noted. Monensin addition to the diets increased (P<.001) ruminal fluid propionate 21.1%, overall. Monensin has been shown to increase ruminal propionate levels in vitro (Van Nevel and DeMeyer,

TABLE 13. MOLAR PROPORTION OF RUMINAL PROPIONATE IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mol/100 mol				
Overall[a]	28.04	36.08	31.69	36.22	.16
Days					
0[a]	20.88	31.37	20.54	31.41	.22
28[b]	27.55	36.44	41.16	40.63	.31
58	39.77	42.74	33.14	38.16	.28
84	30.12	34.02	30.93	34.71	.44

[a]Monensin effect (P<.001).
[b]K effect (P<.05).

1977; Richardson et al., 1976; Slyter, 1979) and *in vivo* in steers fed high-concentrate diets (Richardson et al., 1976; Vijchulata et al., 1980a; Doran et al., 1986b).

No significant K x monensin interaction for molar proportion of ruminal propionate was noted. Greene et al. (1986) reported a significant K x monensin interaction for ruminal propionate in sheep receiving intraruminal infusions of 0, 7.6 or 31.6 g K/d, with or without monensin in the diet. Monensin produced a greater increase in ruminal propionate with infusion of 31.6 g K/d, than with 0 g K/d. Chirase et al. (1987) found no K x monensin interaction for propionate production using a continuous culture fermentation system. A K x lasalocid interaction has been reported for ruminal propionate in sheep fed low- and high-dietary K, with or without monensin (Funk et al., 1986). Lasalocid increased ruminal propionate in sheep fed low levels of K, but was ineffective with high dietary K. Chirase et al. (1987), however, found lasalocid was more effective in increasing propionate production with 2.5% K than with .6% K in a continuous culture fermentation system.

Increasing dietary K from .4% to 2.3% resulted in a decrease (P<.05) in ruminal acetate:propionate ratio at d 28 (Table 14). The actions of monensin in decreasing ruminal acetate and increasing ruminal propionate brought about an overall decrease (P<.001) in ruminal acetate:propionate ratio of 29.5% in finishing steers. Monensin decreased (P<.001) ruminal acetate:propionate ratio 41.1% at d 0. The decreased ruminal acetate:propionate ratio exhibited in ruminants fed monensin has been well-documented (Schelling, 1984).

No K x monensin interaction was found for ruminal

TABLE 14. RUMINAL ACETATE:PROPIONATE RATIO IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
Overall[a]	2.30	1.45	1.99	1.57	.15
Days					
0[a]	3.03	1.74	3.07	1.86	.24
28[b]	2.14	1.42	1.16	1.20	.25
58	1.25	1.18	1.78	1.40	.20
84	2.35	1.32	2.04	1.81	.48

[a]Monensin effect (P<.001).
[b]K effect (P<.05).

acetate:propionate ratio. Greene et al. (1986) reported monensin induced a greater decrease in acetate:propionate ratio in ruminal fluid from sheep receiving 31.6 g K/d by intraruminal infusion than monensin with 7.6 and 0 g K infused daily. However, Chirase et al. (1987) found no significant K x monensin interaction for ruminal fluid acetate:propionate ratio in a continuous culture fermentation system. Schwingel et al. (1987) reported increasing K levels lowered the acetate:propionate ratio in the presence of monensin with an in vitro system.

Feeding high K depressed (P<.01) ruminal butyrate levels 24.5%, overall, compared to feeding low K (Table 15). High dietary K decreased (P<.05) ruminal butyrate at d 28, and at d 84 of the study. Grings and Males (1987) reported a linear decrease in ruminal butyrate levels in sheep receiving increased dietary K. In the present study, monensin addition to the diets brought about an overall decrease (P<.01) of 23.5% in ruminal butyrate. Monensin decreased (P<.05) molar proportions of ruminal butyrate at d 0, 28 and 58. The effect of monensin in depressing ruminal butyrate has been reported both in vitro (Richardson et al., 1976; Slyter, 1979) and in vivo in cattle fed high-concentrate diets (Richardson et al., 1976; Vijchulata et al., 1980). No K x monensin interaction was demonstrated for molar proportions of ruminal butyrate in finishing steers. Previous work has also shown no K x monensin interaction for ruminal butyrate production (Greene et al., 1986; Chirase et al., 1987).

High dietary K decreased (P<.01) ruminal isobutyrate at d 28 of the study (Table 16). Doran et al. (1986b) reported decreased ruminal isobutyrate concentration with increased dietary K in steers fed a high-concentrate diet. In the present study, monensin decreased (P<.10)

TABLE 15. MOLAR PROPORTION OF RUMINAL BUTYRATE IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mol/100 mol				
Overall[ab]	12.62	10.36	10.24	7.11	.08
Days					
0[d]	15.06	12.22	14.07	9.65	.13
28[cd]	12.25	8.57	8.53	6.66	.11
58[d]	9.56	4.89	11.22	6.95	.12
84[c]	12.25	16.42	8.67	5.17	.22

[a]K effect (P<.01).
[b]Monensin effect (P<.01).
[c]K effect (P<.05).
[d]Monensin effect (P<.05).

TABLE 16. MOLAR PROPORTION OF RUMINAL ISOBUTYRATE IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mol/100 mol				
Overall	.48	.48	.49	.38	.004
Days					
0[a]	.71	.64	.78	.48	.008
28[ab]	.54	.37	.29	.26	.005
58	.19	.43	.49	.44	.012
84[c]	.32	.49	.45	.32	.007

[a]Monensin effect (P<.10).
[b]K effect (P<.01).
[c]K x monensin interaction (P<.10).

molar proportions of ruminal isobutyrate at d 0, and d 28 of the study. Monensin has been demonstrated to decrease isobutyrate levels *in vitro* (Slyter, 1979). A significant K x monensin interaction (P<.10) was found for molar proportion of isobutyrate at d 84, with monensin increasing isobutyrate when fed with low K, but decreasing isobutyrate with high K.

Feeding high K decreased (P<.001) ruminal valerate 47.4.%, overall, compared to feeding low K (Table 17). Ruminal valerate decreased (P<.001) at d 28 and d 84 for steers receiving high K, compared to those fed low K. Grings and Males (1987) reported a linear decrease in ruminal valerate in sheep fed increasing dietary K. In the present study, the addition of monensin to the diets decreased (P<.01) molar proportions of ruminal valerate in finishing steers 33.2% overall. Monensin decreased (P<.05) ruminal valerate at d 58 and d 84. Monensin has been shown to both increase (Slyter, 1979) and decrease (Richardson et al., 1976) valerate production *in vitro*, and increase (Richardson et al., 1976; Vijchulata et al., 1980) and decrease (Doran et al., 1986b) ruminal valerate *in vivo*, for cattle fed high-concentrate diets. There was no significant K x monensin interaction for molar proportions of ruminal valerate in finishing steers. Doran et al. (1986b) reported a K x monensin interaction for molar proportions of ruminal valerate in steers fed a high-concentrate diet. They found monensin brought about a greater decrease in ruminal valerate in steers fed higher levels of K.

Feeding high dietary K increased (P<.10) ruminal isovalerate 34.4%, overall, compared to feeding low K (Table 18). Feeding high K decreased (P<.05) ruminal isovalerate at 28 d. Monensin increased (P<.10) ruminal isovalerate 36.4%, overall. Reports on effects of monensin upon ruminal

TABLE 17. MOLAR PROPORTION OF RUMINAL VALERATE IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mol/100 mol				
Overall[ab]	3.97	2.61	2.05	1.41	.030
Days					
0	1.46	1.44	.90	1.00	.037
28[a]	6.25	5.23	3.07	2.28	.068
58[c]	3.80	1.34	2.90	1.28	.072
84[ac]	4.25	1.93	1.56	1.07	.043

[a]K effect ($P<.001$).
[b]Monensin effect ($P<.01$).
[c]Monensin effect ($P<.05$).

TABLE 18. MOLAR PROPORTION OF RUMINAL ISOVALERATE IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mol/100 mol				
Overall[ab]	.84	4.59	1.57	1.70	.021
Days					
0	1.71	5.45	1.90	2.27	.035
28[c]	.66	.59	1.61	1.65	.045
58	.15	1.81	1.40	1.33	.052
84	.40	1.88	1.40	1.56	.036

[a]K effect (P<.10).
[b]Monensin effect (P<.10).
[c]K effect (P<.05).

isovalerate production have been inconsistent. Slyter (1979) showed monensin increased isovalerate production *in vitro*, while Richardson et al. (1976) demonstrated monensin decreased isovalerate production *in vitro*. The latter authors also reported decreased ruminal isovalerate in steers fed high-concentrate diets (Richardson et al., 1976), but Doran et al. (1986b) reported monensin increased ruminal isovalerate in steers fed a high grain diet. There was no K x monensin interaction for molar proportions of ruminal isovalerate in finishing steers.

There were no effects of dietary K or monensin upon BUN in finishing steers (Table 19). Toha et al. (1987) reported that elevated dietary K increased plasma urea N (PUN) 6 h after feeding in sheep. Monensin has been demonstrated to increase PUN in cattle (Schlagheck et al., 1979; Thompson and Riley, 1979). In the present study, a significant K x monensin interaction ($P<.01$) was noted for BUN in steers at d 0. Monensin addition to the diet containing low K resulted in an increase in BUN, but adding monensin to the diet with high K decreased BUN. Funk et al. (1986) have reported a K x lasalocid interaction for PUN in fattening lambs. Sheep fed lasalocid with low dietary K had decreased PUN, while those fed lasalocid with high dietary K had increased PUN.

There were no effects of dietary K or monensin upon ruminal NH_3-N levels in finishing steers (Table 20). Grings and Males (1987) also found no effect of K upon ruminal NH_3-N levels in sheep. Monensin has been shown to reduce ruminal NH_3-N production *in vitro* (Van Nevel and DeMeyer, 1977; Chalupa, 1980), and *in vivo* in steers fed a high-concentrate diet (Doran et al., 1986b), suggesting the ionophore may increase protein escape from the rumen. Significant K x monensin interactions for ruminal

TABLE 19. BLOOD UREA NITROGEN CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mg/100 ml				
Overall	7.61	8.88	7.88	7.70	.45
Days					
0[a]	4.32	6.79	5.58	4.72	.54
28	5.13	6.21	5.37	4.60	.79
58	10.47	9.40	9.65	9.57	.95
84	10.53	13.11	11.06	11.93	1.32

[a]K x monensin interaction ($P<.01$).

TABLE 20. RUMINAL AMMONIA-NITROGEN CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				SE
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	
	mg/100 ml				
Overall	1.49	5.14	2.67	1.98	.44
Days					
0[a]	2.28	4.26	3.74	2.18	.96
28[b]	.33	.84	.83	.62	.15
58	1.72	2.46	2.44	2.52	.99
84	1.91	.17	3.87	2.38	1.21

[a]K x monensin interaction (P<.10).
[b]K x monensin interaction (P<.05).

NH_3-N concentration in finishing steers were found at d 0 (P<.10) and d 28 (P<.05) of this study. Addition of monensin to the diet containing low K increased ruminal NH_3-N levels, but monensin addition to the diet with high K decreased ruminal NH_3-N. Doran et al. (1986b) found no K x monensin interaction for ruminal NH_3-N levels in steers fed a high-concentrate diet.

High dietary K increased (P<.05) serum Na, compared to low K in the diet, at d 0 (Table 21). Feeding high dietary K has been shown to increase apparent absorption of Na in cattle and sheep (Greene et al., 1983b,c). Rahnema and Fontenot (1982) reported that i.v. infusion of KCl resulted in increased apparent absorption and decreased retention of Na in sheep. In the present study, a decrease (P<.05) in serum Na concentration due to monensin was found at d 0. Starnes et al. (1984) demonstrated increased apparent absorption of Na in steers receiving monensin or lasalocid. Kirk et al. (1985a) reported a decrease in Na retention in lambs fed 20 ppm monensin. O'Conner et al. (1986) reported that the addition of monensin to bovine duodenal epithelium preparations decreased mucosal bath Na, indicating an increase in Na absorption with addition of monensin.

A K x monensin interaction (P<.10) for serum Na was observed, overall. Feeding monensin with low dietary K increased serum Na concentration .7%, but feeding monensin with high K decreased serum Na 1.8%. A K x monensin interaction (P<.05) existed at d 0. Monensin fed with low K had no effect upon serum Na concentration, but monensin addition to the high K diet decreased serum Na. Greene et al. (1986) reported no K x monensin interaction for Na metabolism in sheep fed with

TABLE 21. SERUM SODIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	Dietary treatment				
	mg/100 ml				
Overall[a]	227.5	229.1	232.1	227.9	5.35
Days					
0[bcd]	234.2	234.6	248.2	233.2	9.09
28	230.7	229.7	230.7	229.8	7.27
58	214.4	222.2	220.7	220.3	14.96
84	230.7	229.9	229.0	228.3	9.01

[a]K x monensin interaction (P<.10).
[b]K x monensin interaction (P<.05).
[c]K effect (P<.05).
[d]Monensin effect (P<.05).

or without monensin, and receiving intraruminal infusions of 0, 7.6 or 31.6 g K/d. Spears and Harvey (1987) also found no evidence of a K x lasalocid interaction for serum Na in finishing steers.

An overall increase (P<.01) of 7.2% in serum K was found with high dietary K, compared to low K in the diet (Table 22). Feeding high K increased serum K concentration at d 28 (P<.001) and at d 58 (P<.10). Increasing dietary K has been demonstrated to increase serum K concentration in sheep (Greene et al., 1983a; Terashima et al., 1984; Reffert and Boling, 1985), and cattle (Greene et al., 1983c; Deetz et al., 1982). In the present study, there were no effects of monensin upon serum K concentration of finishing steers. Edlin et al. (1984) found dietary lasalocid and monensin depressed serum K concentration in finishing steers. Kelley and Preston (1986) found monensin increased K retention/K intake, increased metabolic fecal K and decreased endogenous urinary K in cattle. Kirk et al. (1985a) reported increased apparent absorption and retention of K in sheep fed 20 ppm monensin. Greene et al. (1986) reported monensin fed to ruminally-cannulated sheep increased apparent absorption of K. In the present study, there was no K x monensin interaction for serum K concentration in finishing steers

Sodium and K homeostasis is maintained primarily by the actions of aldosterone in ruminants (Blair-West et al., 1963). Serum aldosterone levels increase in ruminants in response to decreased serum Na (Blair-West et al., 1971; Bell et al., 1981) and increased serum K (Blair-West et al., 1963). There were no statistically significant effects of K, monensin, or a K x monensin interaction in serum aldosterone concentration in the present study (Table 23). Feeding high K tended to increase serum

TABLE 22. SERUM POTASSIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mg/100 ml				
Overall[a]	21.50	22.05	23.23	23.48	1.58
Days					
0	23.73	24.55	25.06	24.93	3.14
28[b]	23.15	22.54	25.86	26.71	2.20
58[c]	18.73	20.30	20.75	21.50	2.67
84	20.39	20.64	21.26	20.80	4.18

[a]K effect (P<.01).
[b]K effect (P<.001).
[c]K effect (P<.10).

TABLE 23. SERUM ALDOSTERONE CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				SE
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	
	ng/100 ml				
Overall	7.50	7.75	7.79	9.34	.76
Days					
0	7.37	8.57	6.91	9.00	2.08
28	7.08	7.28	6.30	7.85	1.26
58	6.50	7.26	8.78	9.79	1.54
84	9.07	7.91	9.43	10.72	1.12

aldosterone, overall. Riad et al. (1986) reported that an i.v. infusion of aldosterone in beef calves decreased salivary and urinary Na concentration, and increased salivary K concentration, with a resultant decrease in salivary Na:K ratio. No changes in serum Na or K were observed following aldosterone administration, which is consistent with reports on sheep given i.v. aldosterone infusions. (Safwate et al., 1982). Kumar and Singh (1981) also reported decreased salivary Na:K ratio in male buffalo calves given aldosterone infusions, i.v.. Urinary Na excretion also decreased following aldosterone administration.

Overall, feeding high K decreased (P>.01) ruminal Na 44.7%, compared with feeding low K (Table 24). Feeding high K decreased (P<.001) ruminal Na at d 0, 28, 58 and 84. McLean et al. (1985) found ruminal fluid Na decreased when sheep were administered KCl by intraruminal infusion. Reffert and Boling (1985) reported decreased ruminal Na in sheep fed 4% K, 6 h post-feeding. Grings and Males (1987) also found a linear decrease in ruminal Na in sheep fed increasing K. In the present study, no effect due to monensin was observed for ruminal Na concentration in finishing steers. A K x monensin interaction (P<.05) was observed at d 84, with monensin decreasing ruminal Na with low dietary K, and increasing ruminal Na when fed with high K.

Ruminal K levels increased (P<.001) 3.6-fold when steers were fed high K, rather than low K (Table 25). Feeding high K increased (P<.001) ruminal K at d 0, 28, 58 and 84. McLean et al. (1985) reported intraruminal K infusions increased ruminal K in ewes. Toha et al. (1987) and Grings and Males (1987) also reported elevated ruminal K levels with increased dietary K for sheep. There was no effect of monensin upon

TABLE 24. RUMINAL SODIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				SE
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	
	ppm				
Overall[a]	3180	2991	1652	1762	88.3
Days					
0[a]	3693	3384	2828	2726	155.5
28[a]	3331	3379	1489	1553	149.5
58[a]	2648	2830	1111	1509	174.6
84[ab]	2915	2495	1114	1263	90.8

[a]K effect (P<.001).
[b]K x monensin interaction (P<.05).

TABLE 25. RUMINAL POTASSIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				SE
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	
	ppm				
Overall[a]	1484	1347	5417	4872	189.5
Days					
0[a]	1472	1051	4008	3199	438.0
28[a]	1288	1214	5228	5070	200.2
58[a]	2409	1355	5706	5123	427.4
84[a]	1322	1422	6185	6096	176.9

[a]K effect (P<.001).

ruminal K concentration in finishing steers in the present experiment. Starnes et al. (1984) demonstrated decreased ruminal fluid K in steers fed monensin and lasalocid. However, Lemenager et al. (1978) reported monensin supplementation increased ruminal K in grazing cows. In the present study, there was no K x monensin interaction for ruminal K concentration.

Feeding high K decreased (P<.001) ruminal Na:K ratio 81.4%, compared to feeding low K, overall (Table 26). Feeding high K decreased (P<.001) ruminal Na:K ratio at d 0, 28, 58 and 84. Increased dietary K has been shown to decrease ruminal Na:K ratio in sheep (McLean et al., 1985) and cattle (McGregor and Armstrong, 1979). This effect may be secondary to an increase in serum aldosterone associated with increased dietary K. In the present study, there was a significant K x monensin interaction (P<.05) for ruminal Na:K ratio at d 84, with monensin decreasing ruminal Na:K ratio when fed with low K, and increasing Na:K ratio with high dietary K.

An overall decrease (P<.001) in serum Mg of 12.8% was observed when high K was fed, compared to feeding low K (Table 27). Feeding high dietary K decreased serum Mg at d 0 (P<.10), d 28 (P<.001), d 58 (P<.05), and at d 84 (P<.001). Feeding high K has been demonstrated to reduce Mg availability in sheep (Kunkel et al., 1953; House and Van Campen, 1971; Newton et al., 1972; Green et al., 1983a) and cattle (Field and Suttle, 1979; Greene et al, 1983c). High dietary K has also been shown to decrease serum Mg levels in ruminants (Kunkel et al., 1953; Field and Suttle, 1979; Greene et al., 1983a,b).

Monensin addition to the diets decreased (P<.10) serum Mg

TABLE 26. RUMINAL SODIUM:POTASSIUM RATIO IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
Overall[a]	2.48	2.48	.44	.49	.11
Days					
0[a]	2.98	3.22	1.10	1.14	.37
28[a]	2.60	2.54	.26	.31	.06
58[a]	1.89	2.36	.18	.30	.23
84[abc]	2.24	1.69	.18	.21	.10

[a]K effect ($P<.001$).
[b]K x monensin interaction ($P<.05$).
[c]Monensin effect ($P<.05$).

TABLE 27. SERUM MAGNESIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mg/100 ml				
Overall[ab]	2.17	2.22	1.99	1.83	.036
Days					
0[cde]	2.12	2.14	2.13	1.84	.073
28[b]	2.33	2.32	1.93	1.93	.073
58[f]	2.03	2.10	1.85	1.75	.095
84[ab]	2.20	2.30	2.02	1.81	.050

[a]K x monensin interaction ($P<.01$).
[b]K effect ($P<.001$).
[c]K x monensin interaction ($P<.05$).
[d]K effect ($P<.10$).
[e]Monensin effect ($P<.10$).
[f]K effect ($P<.05$).

concentration at d 0. Starnes et al. (1984) reported both monensin and lasalocid increased apparent absorption and retention of Mg in steers. Kirk et al. (1985b) found monensin increased Mg retention in sheep. Spears et al. (1987) reported that monensin increased the apparent absorption of Mg by steers fed green chopped, tall fescue. O'Conner et al. (1986) added monensin to the mucosal bath of bovine duodenal epithelial preparations and found decreased mucosal bath Mg, indicating an increase in Mg absorption. Monensin increased the net absorption of Mg in steers, as calculated by hepatic artery-portal vein difference (Johnson et al., 1987). Greene et al. (1988) found increased preintestinal absorption and small intestinal secretion of Mg in steers fed monensin, with an overall increase in total tract absorption of Mg when monensin was fed. However, Grings and Males (1988) found monensin decreased serum Mg concentration when added to MgO supplements for cows fed wheat straw-based diets, but serum Mg was unchanged when monensin was added to $MgSO_4$ supplement.

There was a K x monensin interaction ($P<.01$) for serum Mg concentration, overall. Monensin increased serum Mg 2.3% when fed with low K, and decreased serum Mg 8.0% when fed with high K. A K x monensin interaction was observed at d 0 ($P<.05$) and d 84 ($P<.01$) of the study. At d 0, monensin did not affect serum Mg when fed with low K, but decreased serum Mg when fed with high K. At d 84, monensin increased serum Mg with low dietary K, but decreased serum Mg with high K in the diet. Greene et al. (1986) found no K x monensin interaction for Mg metabolism in sheep fed with or without 20 ppm monensin, and receiving 0, 7.6, or 31.6 g K/d by intraruminal infusion. These results have implications concerning feeding monensin to lactating beef cows grazing high-K, tetany-prone forages (Fontenot, 1979).

Soluble ruminal fluid Mg increased (P<.05) 15.1%, overall, when high K was fed (Table 28). Feeding high K increased (P<.10) ruminal Mg at d 28. McLean et al. (1985) reported decreased ruminal Mg concentration in sheep administered intraruminal KCl. Monensin addition to the diets did not alter ruminal Mg concentration in finishing steers, in the present study. There was no significant K x monensin interaction for ruminal Mg concentration in finishing steers.

Feeding high K increased (P<.001) serum Ca 5.0% overall, compared to feeding low K (Table 29). Serum Ca concentration increased at d 0 (P<.001), and at d 28 (P<.05) for steers fed high K. Newton et al. (1972) have reported decreased fecal Ca excretion in sheep fed increased K. Greene et al. (1983a) found a linear decrease in fecal and total Ca excretion, and decreased serum Ca concentration in sheep fed increased K. Timet et al. (1983) reported that Na-stimulated Ca absorption from ruminal mucosal tissue was depressed by the addition of K. In the present experiment, feeding monensin increased (P<.05) serum Ca concentration 2.4%, overall. Starnes *et al*. (1984) have reported increased serum Ca in steers fed monensin or lasalocid. Greene et al. (1986) reported that monensin increased apparent absorption and retention of Ca in lambs. However, monensin was shown to decrease net Ca absorption in steers, as calculated by hepatic artery-portal vein difference (Johnson et al., 1987). There was no significant K x monensin interaction for serum Ca

TABLE 28. RUMINAL MAGNESIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH MONENSIN WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	ppm				
Overall[a]	93.56	76.11	98.01	97.35	6.28
Days					
0	82.42	41.30	74.63	63.46	17.27
28[a]	83.83	99.62	118.92	104.90	10.54
58	86.24	65.68	90.22	95.00	10.43
84	125.90	104.69	109.98	126.02	14.66

[a]K effect (P<.05).

TABLE 29. SERUM CALCIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mg/100 ml				
Overall[ab]	9.99	10.22	10.46	10.73	.12
Days					
0[a]	9.77	9.53	10.20	10.40	.13
28[c]	9.90	10.38	10.58	10.56	.19
58	9.90	10.48	10.50	10.97	.38
84	10.38	10.48	10.62	10.97	.22

[a]K effect ($P<.001$).
[b]Monensin effect ($P<.05$).
[c]K effect ($P<.05$).

levels in finishing steers, in the present experiment.

Feeding high K decreased (P<.05) soluble ruminal fluid Ca 33.2% at d 84, compared to feeding low K (Table 30). Monensin decreased (P<.05) ruminal Ca concentration 23.9%, overall. There was no K x monensin interaction for ruminal Ca concentration in finishing steers.

There was no effect of dietary K level upon serum inorganic P concentration in finishing steers (Table 31). Feeding monensin lowered serum inorganic P at d 0. Monensin and lasalocid supplementation have been shown to increase serum inorganic P in finishing steers (Edlin et al., 1984). Starnes et al. (1984) reported increased apparent absorption and retention of P in steers fed monensin and lasalocid. Kirk et al. (1985b) also found increased apparent absorption and retention of P when monensin was fed to sheep. Greene et al. (1986) have also found improved apparent absorption of P in sheep fed monensin, when averaged across ruminal infusions of 0, 7.6 and 31.6 g K/d. In the present study, there was an overall K x monensin interaction (P<.01) for serum inorganic P concentration in finishing steers (P<.01). Monensin addition to the diet containing low K increased serum inorganic P 3.6%, while monensin fed with high K depressed serum inorganic P 8.3%. This interaction was seen at d 0 (P<.10), with monensin fed with low K having no effect upon serum inorganic P, but monensin with high dietary K decreasing serum inorganic P 17.8%.

Feeding high K decreased (P<.001) soluble ruminal fluid inorganic P 12.9%, overall, compared to feeding low K (Table 32). High dietary K decreased (P<.05) ruminal inorganic P at d 28, 56 and 84. Feeding increased K has been shown to decrease P secretion into the preintestinal

TABLE 30. RUMINAL CALCIUM CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	ppm				
Overall[a]	97.48	67.82	80.92	67.89	10.80
Days					
0	104.98	35.74	49.68	29.42	25.32
28	78.87	93.23	148.26	92.00	26.35
58	95.49	33.56	58.16	52.45	17.35
84[b]	125.78	122.42	68.21	97.69	16.32

[a]Monensin effect (P<.05).
[b]K effect (P<.05).

TABLE 31. SERUM INORGANIC PHOSPHORUS CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				SE
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	
	mg/100 ml				
Overall[a]	6.93	7.18	7.31	6.70	.14
Days					
0[bc]	7.33	7.32	7.46	6.13	.34
28	6.47	6.72	6.55	6.51	.21
58	6.72	7.01	7.54	6.89	.32
84	7.19	7.69	7.70	7.28	.28

[a]K x monensin interaction (P<.01).
[b]K x monensin interaction (P<.10).
[c]Monensin effect (P<.10).

TABLE 32. RUMINAL INORGANIC PHOSPHORUS CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	mg/100 ml				
Overall[a]	1228	1206	1064	1056	10.0
Days					
0	1050	1026	924	918	6.4
28[b]	1196	1180	1034	1120	3.8
58[b]	1272	1216	1060	1108	5.0
84[b]	1408	1498	1218	1046	7.8

[a]K effect ($P<.001$).
[b]K effect ($P<.05$).

region in sheep (Greene et al., 1983b) and cattle (Green et al., 1983c). In the present study, feeding monensin had no effect upon ruminal inorganic P concentration in finishing steers. There was no K x monensin interaction for soluble ruminal fluid inorganic P concentrations in finishing steers.

Dietary K concentration had no effect upon serum Cu concentration in finishing steers (Table 33). Monensin increased ($P<.01$) serum Cu concentration 9.5%, overall, and increased ($P<.10$) serum Cu at d 28 and 84. Starnes et al. (1984) have reported increased serum Cu concentration in steers receiving 33 ppm monensin or lasalocid. Elsasser (1984) demonstrated increased liver Cu concentration in sheep fed monensin or lasalocid. In the present study, there was a significant K x monensin interaction ($P<.01$) for serum Cu concentration in finishing steers, overall. Monensin increased serum Cu 19.1% when fed with low K, but did not alter it when fed with high K. A K x monensin interaction ($P<.10$) was seen at d 28, with monensin increasing serum Cu when fed low K, but having no effect when fed with high K. There were no dietary effects upon soluble ruminal fluid Cu concentration in finishing steers (Table 34).

Dietary K level did not affect serum Fe (Table 35). Monensin decreased ($P<.05$) serum Fe concentration 8.1%, overall. A decrease ($P<.05$) in serum Fe was seen in steers fed monensin at d 28. Elsasser (1984) reported monensin decreased Fe uptake by chicken duodenal loops. Monensin has not been demonstrated to alter serum Fe levels in ruminants, but Spears and Harvey (1987) reported increased plasma Fe concentration in steers fed lasalocid. In the present study, there was no K x monensin interaction for serum Fe concentration in finishing steers.

TABLE 33. SERUM COPPER CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3%K + monensin	SE
	ug/100 ml				
Overall[ab]	80.4	95.7	84.0	84.4	.24
Days					
0	78.7	86.8	83.4	79.6	.50
28[cde]	91.2	107.5	91.2	91.8	.42
58	73.8	93.5	85.6	84.4	.58
84[e]	77.8	94.9	77.1	81.7	.58

[a]K x monensin interaction (P<.01).
[b]Monensin effect (P<.01).
[c]K x monensin interaction (P<.10).
[d]K effect (P<.10).
[e]Monensin effect (P<.10).

TABLE 34. RUMINAL COPPER CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	ppm				
Overall	.071	.081	.082	.071	.006
Days					
0	.002	.021	.004	.004	.006
28	.035	.061	.047	.043	.011
58	.142	.150	.154	.137	.016
84	.107	.092	.123	.099	.015

TABLE 35. SERUM IRON CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				SE
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	
	ug/100 ml				
Overall[a]	202	190	216	194	.78
Days					
0	142	130	166	143	1.60
28[a]	260	227	252	205	1.84
58	195	203	235	222	1.77
84	210	202	212	206	1.45

[a]Monensin effect ($P<.05$).

Feeding high K decreased (P<.05) soluble ruminal fluid Fe concentration 19.9%, overall, compared to feeding low K (Table 36). High dietary K decreased (P<.10) ruminal Fe at d 58. Feeding monensin had no effect upon ruminal Fe concentration finishing steers. There was an overall K x monensin interaction (P<.10) for ruminal Fe concentration in finishing steers. Monensin decreased ruminal Fe concentration slightly when fed with low K, but increased ruminal Fe 41.3% when fed with high K.

No effects of K or monensin were determined for serum Zn concentration in finishing steers (Table 37). Starnes et al. (1984) have reported that feeding monensin or lasalocid increased serum Zn concentration in steers. Kirk et al. (1985b) found monensin increased apparent absorption and retention of Zn when sheep were fed monensin. Costa et al. (1985) reported increased Zn retention in sheep receiving 125 mg monensin daily. An overall K x monensin interaction (P<.05) was demonstrated for serum Zn concentration in finishing steers. Monensin increased serum Zn 9.1% when fed with low K, but decreased serum Zn 4.3% when fed with high K.

Feeding high K decreased (P<.10) soluble ruminal fluid Zn content at d 0, compared to feeding low K (Table 38). Monensin decreased (P<.05) ruminal Zn concentration 17.8%, overall. A decrease (P<.10) in ruminal Zn was observed in steers fed monensin at d 1. Kirk et al. (1985b) reported increased ruminal fluid Zn in lambs fed monensin. Spears and Harvey (1987) reported that feeding lasalocid to finishing steers reduced ruminal Zn content. In the present study, there was no K x monensin interaction for soluble ruminal fluid Zn concentration in finishing

TABLE 36. RUMINAL IRON CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	ppm				
Overall[ab]	1.70	1.66	1.12	1.58	.12
Days					
0	1.13	1.25	.82	1.12	.17
28	1.33	1.32	1.25	1.64	.19
58[c]	2.75	2.13	1.38	1.90	.35
84	1.88	1.85	1.09	1.64	.30

[a]K x monensin interaction (P<.10).
[b]K effect (P<.05).
[c]K effect (P<.01).

TABLE 37. SERUM ZINC CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

	Dietary treatment				
Period	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	ug/100 ml				
Overall[a]	73.6	80.2	81.5	78.0	.22
Days					
0	55.9	63.6	72.7	61.5	.54
28	82.0	90.2	82.9	80.0	.38
58	73.3	80.6	86.7	87.6	.58
84	83.2	86.6	84.1	82.7	.30

[a]K x monensin interaction ($P<.05$).

TABLE 38. RUMINAL ZINC CONCENTRATION IN FINISHING STEERS FED LOW OR HIGH POTASSIUM WITH OR WITHOUT MONENSIN.

Period	Dietary treatment				
	.4% K	.4% K + monensin	2.3% K	2.3% K + monensin	SE
	ppm				
Overall[a]	.90	.77	.71	.65	.051
Days					
0[bc]	.88	.50	.52	.33	.122
28	1.03	.96	.92	.85	.114
58	.86	.66	.66	.65	.093
84	.86	.99	.79	.78	.076

[a]Monensin effect ($P<.05$).
[b]K effect ($P<.10$).
[c]Monensin effect ($P<.10$).

steers.

Conclusions. Changes in dietary K did not significantly alter the effects of monensin upon performance, carcass characteristics, or ruminal fluid VFA content of finishing steers. However, changes in mineral status of steers fed monensin were dependent upon K concentration in the diet. Serum Na, Mg, and inorganic P concentration increased when monensin was fed with low K, but decreased when the ionophore was fed with high K. Serum Cu increased when monensin was added to the low K diet, but monensin did not affect serum Cu when fed with high K. Addition of monensin to the diet containing low K increased serum Zn, but monensin fed with high K decreased serum Zn. Therefore, K concentration in the diet should be considered when examining effects of monensin upon mineral status of ruminants.

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JOURNAL ARTICLE NO. III.

EFFECTS OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF MINERALS IN SHEEP

ABSTRACT

Fifteen wethers, fitted with abomasal and ileal cannulae, were fed a 70% concentrate diet alone or supplemented with 23 ppm lasalocid or 23 ppm monensin, dry basis. Wethers were kept in metabolism stalls for total collection of feces and urine. They were fed 398 g DM twice daily during a 10-d preliminary period, a 10-d collection period, and a 6-d cannulae collection period. During the collection period, feed, feces, and urine were collected daily and composited. During the cannulae collection, abomasal and ileal digesta, and feces were collected at 12 h intervals, advancing 2 h each day, and composited. Apparent absorption of Mg increased ($P<.05$) 6% when lasalocid was fed, and 12% when monensin was fed. Lasalocid and monensin increased ($P<.05$) urinary Mg excretion 17% and 19%, respectively. Flow of Mg through the small intestine decreased ($P<.05$) 7% with lasalocid, and 10% with monensin. Large intestinal flow of Mg was decreased ($P<.05$) 4% with lasalocid and 11% with monensin. Lasalocid and monensin did not alter Ca and P metabolism. Feeding lasalocid decreased ($P<.05$) urinary excretion of Na 29%, while feeding monensin tended to increase it, compared to sheep fed the control diet. Apparent absorption of K increased ($P<.10$) slightly when lasalocid and monensin were fed. Apparent absorption of Fe increased ($P<.10$) 8% and 4%, respectively, when lasalocid and monensin were fed. For several variables, the effects of lasalocid and monensin were different, when compared to controls. Lasalocid and monensin were found to alter

absorption, retention, extent partial absorption and digestive tract flow of minerals in sheep.

(KEY WORDS: Lasalocid, Monensin, Minerals, Metabolism, Flow)

INTRODUCTION

Lasalocid and monensin are non-nutritive feed additives commonly supplemented to ruminants to increase feed efficiency and(or) rate of gain. The main effect of these ionophores is to promote an energetically favorable shift in ruminal fermentation, with decreased acetate and increased propionate production. However, the primary effect of these carboxylic ionophores is to alter the flow of cations across cell membranes (Pressman, 1976). Lasalocid is a divalent ionophore demonstrated to bind both divalent cations, Ca^{2+} and Mg^{2+}, as well as monovalent cations, Na^+ and K^+. Monensin is a monovalent ionophore binding only monovalent cations, such as Na^+ and K^+

Both lasalocid and monensin have been demonstrated to alter mineral metabolism in ruminants, presumably due to their ionophore action. Starnes et al. (1984) reported that feeding 33 ppm lasalocid or monensin in a high-concentrate diet to steers increased apparent absorption of Na, Mg and P, and increased Mg and P retention, compared to animals fed the same diet without ionophores. Kirk et al. (1985a,b) demonstrated increased apparent absorption of P, K and Zn, increased retention of P, Mg, K and Zn, and decreased Na retention in wethers fed a 70% concentrate diet with 20 ppm monensin, compared to no ionophore. Recently, Greene et al. (1988) examined changes in the site and extent of Mg and Ca absorption in steers, fitted with duodenal and ileal cannulas, fed with or without 25 ppm monensin. Feeding monensin increased absorption of Mg from the preintestinal region and increased Mg secretion into the small intestine. A previous report has indicated an increase in Zn availability but no significant change in partial absorption of Zn in duodenal and ileal cannulated steers (Greene et al., 1986b). Darden et al.(1985) reported increased Mg absorption from the stomach of ruminal and duodenal cannulated steers fed lasalocid, compared with monensin.

The present study was conducted to determine the effects of lasalocid and monensin upon the digestive tract flow and partial absorption of Ca, P, Mg, Na, K, Cu, Fe, and Zn in sheep.

EXPERIMENTAL PROCEDURE

Fifteen crossbred wethers (avg wt 45.0 kg), fitted with abomasal and ileal cannulas, were used in a randomized block design experiment. Sheep were blocked according to body weight, and randomly allotted to a basal diet (Table 39) fed alone, or supplemented with 23 ppm lasalocid, or 23 ppm monensin, dry basis. Chromic oxide was used as a marker to determine digestive tract flow and partial absorption of minerals. Sheep were maintained under continuous lighting in metabolism stalls, designed for the total collection of feces and urine, throughout the study. Animals were dewormed prior to placement in stalls. Animals were fed twice daily through a 17-d adjustment period to determine optimal feed intake. Wethers were fed 398 g DM at 0700 h and 1800 h daily (total of 796 g) during a 10-d preliminary period, a 10-d collection period, and a 6-d cannula collection period.

TABLE 39. INGREDIENT AND MINERAL COMPOSITION OF BASAL DIET

Item	Percent
Ingredient composition[a]	
Ground corn grain	66.83
Fescue hay	30.12
Corn gluten meal	2.00
Calcitic limestone	.50
Sodium chloride	.30
Chromic oxide	.25
Dry matter	90.45
Mineral composition[b]	
Calcium	.54
Phosphorus	.28
Magnesium	.13
Sodium	.21
Potassium	.65
Copper	.001
Iron	.007
Zinc	.001

[a]As-fed basis.
[b]Dry basis.

During the collection period feed, fecal and urine samples were collected to determine apparent absorption and retention of minerals. Feed samples were obtained at each feeding, 2 d before the start until 2 d after the end of collection. Total fecal material was collected daily and dried at 70 C in a forced-air oven for 48 h. Dried feces were composited daily in sealed containers, and sampled at the end of the trial. Urine was collected daily, diluted to constant volume with distilled, deionized water, and a 2% aliquot (by volume) composited, and frozen for subsequent analysis. Twenty-five milliliters of a 9M H_2SO_4 solution were added daily to urine collection containers.

During the cannula collection period, feed, abomasal and ileal digesta, and fecal samples were collected. Feed samples were taken at each feeding beginning 2 d before through d 4 of this period. Abomasal, ileal and fecal samples were collected simultaneously, twice daily, at 12-h intervals, advancing 2 h each day. Following the cannula collection period, an equal volume (25 ml) of abomasal digesta, an equal weight (15 g) of ileal digesta and an equal weight (25 g) of fecal material were composited for each animal. Abomasal and ileal digesta were dried in a 40 C forced-air oven for 72 h, and feces were dried in a 70 C forced-air oven for 48 h.

Feed, abomasal, ileal, and fecal samples were ground through a 1 mm screen, prior to wet digestion in nitric and perchloric acid (Hern, 1979). Urine samples were filtered through ashless filter paper. Chromium content was determined on composited feed, abomasal, ileal, and fecal samples, using the procedure of Hill and Anderson (1958). Feed, abomasal and ileal digesta, feces, and urine were analyzed for Na and K content by emission spectrophotometry, and Ca, Mg, Cu, Fe and Zn using atomic absorption spectrophotometry. Samples were diluted using 1.0% $LaCl_3$ for Ca and Mg determination. Phosphorus was determined by the colormetric procedure outlined by Fiske and Subarrow (1925).

Data were analyzed using the General Linear Model procedure of the Statistical Analysis System (SAS, 1986). Orthogonal contrasts were control- vs ionophore-fed sheep, and lasalocid- vs monensin-fed sheep. Examination of treatment means revealed opposite effects of lasalocid and monensin, compared to controls for several variables. In these instances, Dunnett's test for treatment mean comparison to control, was conducted (Zar, 1984).

RESULTS AND DISCUSSION

Chromium recovery exceeded 100% for all treatment groups, with an overall average of 104.9% of dietary Cr recovered in the feces. Apparent digestibility of DM during the metabolism study was 81.6, 82.7 and 81.6% for control-, lasalocid- and monensin-fed sheep, respectively. The addition of ionophores did not significantly alter digestive tract flow and partial absorption of DM (Table 40). Apparent DM digestion occurred primarily in the preintestinal region, followed in order by the small intestine and large intestine. Hogan and Phillipson (1960) reported that approximately 70% of DM absorption occurred in the stomach of sheep. In the present experiment, lasalocid decreased ($P<.10$) DM flow through the small intestine 10%, compared to monensin. Flow of DM through the large intestine was decreased ($P<.05$) for lasalocid vs monensin. Dry matter absorption from the small intestine, expressed as a percentage of DM

TABLE 40. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF DRY MATTER IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	796.0	796.0	796.0	---
Flow, g/d				
Preintestine	413.2	412.4	385.6	28.5
Small intestine[a]	163.2	154.4	172.6	4.98
Large intestine	129.7	126.5	136.8	2.44
Partial absorption				
Preintestine				
Grams per day	382.8	383.6	410.4	28.5
Percent of intake	48.08	48.19	51.56	3.58
Small intestine				
Grams per day	250.1	258.0	213.0	26.7
Percent of intake	31.41	32.41	26.76	3.35
Percent of entry[b]	60.34	61.82	55.05	2.34
Large intestine				
Grams per day	33.50	27.93	35.75	3.47
Percent of intake	4.21	3.51	4.49	.44
Percent of entry	20.25	17.81	20.69	1.58
Total				
Grams per day	666.3	669.5	659.2	2.44
Percent of intake	83.71	84.11	82.81	.30

[a]Lasalocid differs from monensin ($P<.05$).
[b]Lasalocid differs from monensin ($P<.10$).

entering this region, was higher (P<.10) for lasalocid-fed sheep, compared to monensin. The total absorption of DM determined during the cannula collection period was similar to that obtained during the total collection period.

The addition of ionophores to the basal diet did not alter Ca metabolism in sheep (Table 41). However, lasalocid tended to increase, while monensin tended to decrease apparent absorption and retention of Ca, compared to wethers fed no ionophores. Lasalocid decreased (P<.05) fecal Ca excretion 13%, increasing (P<.05) apparent absorption of Ca 37%, compared to monensin. Total Ca excretion decreased (P<.05) 10.1%, increasing (P<.05) Ca retention 36% for lasalocid, compared to monensin. Starnes et al. (1984) reported no change in apparent absorption or retention of Ca in steers fed 33 ppm lasalocid or monensin, although soluble Ca in the ruminal fluid decreased when the ionophores were fed. Kirk et al. (1985b) found no change in Ca metabolism in wethers receiving 20 ppm monensin, but urinary Ca excretion decreased 60% when monensin was fed. Contrary to these findings, Greene et al. (1986b) reported increased apparent absorption and retention of Ca in ruminally-cannulated wethers receiving 20 ppm monensin in a high-concentrate diet. Spears et al. (1987) found increased apparent absorption and retention of Ca in steers fed green-chopped, tall fescue with 200 mg monensin•hd^{-1}•d^{-1}

There were no significant differences between treatment groups for flow of Ca through the digestive tract, or partial absorption of Ca (Table 42). Greene et al. (1988) also reported no change in site or extent of Ca absorption in duodenal and ileal cannulated steers fed with 25 ppm monensin, although there was a tendency towards increased Ca absorption

TABLE 41. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF CALCIUM IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	4.31	4.31	4.31	--
Excretion, g/d				
Fecal[a]	3.09	2.80	3.21	.10
Urine	.19	.22	.15	.04
Total[a]	3.27	3.02	3.36	.07
Apparent absorption				
Grams per day[a]	1.22	1.51	1.10	.10
Percent of intake[a]	28.35	35.03	25.52	2.21
Retention				
Grams per day[a]	1.03	1.29	.95	.07
Percent of intake[a]	24.01	29.90	22.09	1.73
Percent of absorbed	84.30	86.19	86.44	2.80

[a]Lasalocid differs from monensin ($P<.05$).

TABLE 42. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF CALCIUM IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	4.17	4.17	4.17	--
Flow, g/d				
Preintestine	3.49	3.63	3.81	.14
Small intestine	3.13	3.08	3.25	.12
Large intestine	3.15	3.18	3.28	.08
Partial absorption				
Preintestine				
Grams per day	.68	.54	.37	.14
Percent of intake	16.34	12.95	8.78	3.45
Small intestine				
Grams per day	.36	.55	.55	.10
Percent of intake	8.61	13.20	13.27	2.51
Percent of entry	7.50	14.64	14.14	3.33
Large intestine				
Grams per day	-.02	-.09	-.03	.08
Percent of intake	--	--	--	--
Percent of entry	--	--	--	--
Total				
Grams per day	1.02	1.00	.89	.08
Percent of intake	24.48	23.89	21.31	2.05

in the preintestinal region when monensin was fed. O'Conner and Beede (1986) reported a linear decrease in bovine duodenal mucosal bath Ca with increased lasalocid concentration applied to the mucosal bath of a Ussing chamber. These results indicate an increase in Ca absorption in the presence of lasalocid.

The primary sites of Ca absorption in the present study were the preintestinal region and the small intestine. A trend was recorded for lower absorption preintestinally and higher absorption from the small intestine in sheep fed the ionophores. Greene et al. (1983) reported a net secretion of Ca into the preintestine and large intestine of sheep, with the small intestine as the primary site of Ca absorption. However, Grace et al. (1974) reported a net absorption of Ca from the preintestinal region, with Ca secretion into the small intestine. Rahnema and Fontenot (1982) reported net Ca absorption from the preintestine and large intestine of lambs, with a net secretion of Ca into the small intestine. Recently, Greene et al. (1988) suggested that the site of Ca absorption may vary widely in ruminants due to the form and solubility of Ca, and Ca x nutrient interactions in the digestive tract.

Feeding ionophores tended to increase apparent absorption and retention of P in sheep (Table 43). Starnes et al. (1984) found increased apparent absorption and retention of P in steers fed 33 ppm lasalocid or monensin compared to control steers. Kirk et al. (1985b) reported increased apparent absorption and retention of P in sheep fed 20 ppm monensin. Apparent absorption of P was increased in steers fed 200 $mg \cdot hd^{-1} \cdot d^{-1}$ monensin with green-chopped tall fescue (Spears et al., 1987).

TABLE 43. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF PHOSPHORUS IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	2.25	2.25	2.25	--
Excretion, g/d				
Fecal	1.79	1.58	1.69	.10
Urine	.04	.06	.07	.02
Total	1.83	1.64	1.76	.09
Apparent absorption				
Grams per day	.46	.67	.56	.10
Percent of intake	20.66	29.91	24.96	4.32
Retention				
Grams per day	.42	.61	.49	.09
Percent of intake	18.58	26.98	21.90	3.90
Percent of absorbed	88.84	90.3	88.06	2.70

Digestive tract flow and partial absorption of P was not altered by the addition of ionophores to the basal diet (Table 44). All treatment groups showed a net secretion of P into the preintestine. The small intestine was the primary site of P absorption. Although large intestinal absorption of P was minimal, there were differences between ionophore-fed groups. Absorption from this area was lower ($P<.05$) for sheep fed lasalocid, compared to monensin. Other researchers have reported a net secretion of P into the preintestine, with the small intestine as the primary site of absorption (Grace et al. 1974, Greene et al., 1983; Rahnema and Fontenot 1983; Wylie et al., 1985).

Apparent absorption of Mg was increased ($P<.10$) when ionophores were added to the basal diet (Table 45). The increases were 6% for lasalocid and 12% for monensin. This was due to a decrease ($P<.10$) in fecal Mg excretion of 5% and 12% for lasalocid and monensin, respectively. An increase ($P<.01$) in urinary Mg excretion of 17% for lasalocid, and 19% for monensin, compared to controls, prevented any significant change in Mg retention among ionophore-fed sheep. The increase in urinary Mg excretion was probably a reflection of increased absorption (Chicco et al., 1972). Starnes et al. (1984) found increased apparent absorption and retention of Mg when lasalocid and monensin were fed to steers. Kirk et al. (1985b) reported a 42% increase in Mg retention in sheep receiving 20 ppm monensin. Greene et al. (1986b) found monensin increased apparent absorption of Mg in ruminally cannulated wethers. Steers receiving 200 $mg \cdot hd^{-1} \cdot d^{-1}$ monensin with green-chopped tall fescue had increased apparent absorption of Mg (Spears et al., 1987). O'Conner and Beede (1986) reported a linear decrease in bovine duodenal mucosal bath Mg

TABLE 44. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF PHOSPHORUS IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	2.25	2.25	2.25	--
Flow, g/d				
Preintestine	7.12	6.94	7.17	.26
Small intestine	1.75	1.62	1.87	.08
Large intestine	1.60	1.55	1.60	.06
Partial absorption				
Preintestine				
Grams per day	-4.81	-4.63	-4.86	.26
Percent of intake	--	--	--	--
Small intestine				
Grams per day	5.37	5.32	5.30	.29
Percent of intake	232.44	230.21	229.34	12.40
Percent of entry	74.92	76.03	73.30	1.63
Large intestine				
Grams per day[a]	.15	.07	.27	.05
Percent of intake[a]	6.40	3.18	11.89	2.24
Percent of entry[a]	7.63	4.53	14.29	2.52
Total				
Grams per day	.71	.76	.71	.06
Percent of intake	30.62	32.81	30.71	2.80

[a]Lasalocid differs from monensin ($P<.05$).

TABLE 45. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF MAGNESIUM IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE[a]
Intake, g/d	1.07	1.07	1.07	--
Excretion, g/d				
Fecal[a]	.59	.56	.52	.02
Urine[b]	.42	.49	.50	.02
Total	1.01	1.05	1.03	.02
Apparent absorption				
Grams per day[a]	.48	.51	.54	.02
Percent of intake[a]	45.09	47.56	50.93	1.68
Retention				
Grams per day	.06	.02	.04	.02
Percent of intake	5.72	1.81	3.58	1.67
Percent of absorbed	12.46	3.64	6.62	3.22

[a]Control differs from ionophores ($P<.10$).
[b]Control differs from ionophores ($P<.01$).

concentrations with increased monensin addition to the mucosal side of the tissue in a Ussing chamber, indicating increased Mg absorption with monensin. Johnson et al. (1986) reported increased net absorption of Mg, by portal vein-hepatic artery difference in steers fed monensin.

Lasalocid and monensin decreased ($P<.05$) Mg flow through the small intestine 7% and 10%, respectively (Table 46). Large intestinal Mg flow decreased ($P<.10$) 4% with lasalocid, and 11% with monensin, compared to controls. Monensin decreased ($P<.10$) Mg flow through the large intestine 7%, compared to lasalocid. Despite changes in Mg flow through the sheep gut, ionophores did not alter Mg partial absorption. The preintestinal region was the primary site of Mg absorption, with a net secretion into the small intestine, and negligible absorption in the large intestine. A tendency toward increased absorption in the preintestinal region, and decreased secretion into the small intestine were observed when ionophores were fed. These trends resulted in an increase ($P<.10$) in total apparent absorption of Mg when ionophores were fed. Total absorption of Mg was 11.6% greater ($P<.10$) for lambs fed monensin, compared to lambs fed lasalocid. Previous reports have demonstrated the preintestinal region as the primary site of Mg apparent absorption, with a net secretion into the small intestine, and small absorption by the large intestine of sheep. (Grace et al. 1974; Giduck et al. 1981; Greene et al., 1983; and Rahenma and Fontenot 1983). However, Wylie et al. (1985) found that although the preintestine was the major region of Mg absorption, absorption also occurred in the small intestine, with a net Mg secretion into the large intestine. The reticulo-rumen has been

TABLE 46. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF MAGNESIUM IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	.98	.98	.98	--
Flow, g/d				
Preintestine	.49	.46	.45	.02
Small intestine[a]	.60	.56	.52	.02
Large intestine[bc]	.56	.54	.50	.02
Partial absorption				
Preintestine				
Grams per day	.49	.51	.53	.02
Percent of intake	50.10	52.71	54.01	2.38
Small intestine				
Grams per day	-.11	-.10	-.08	.01
Percent of intake	--	--	--	--
Percent of entry	--	--	--	--
Large intestine				
Grams per day	.03	.01	.03	.01
Percent of intake	3.53	1.49	2.84	.93
Percent of entry	5.57	2.25	5.35	1.51
Total				
Grams per day[bc]	.41	.43	.48	.02
Percent of intake[bc]	42.33	43.87	48.84	1.75

[a]Control differs from ionophores (P<.05).
[b]Control differs from ionophores (P<.10).
[c]Lasalocid differs from monensin (P<.10).

observed as the primary site of Mg absorption in sheep, with little absorption in the omasum and abomasum (Tomas and Potter, 1976).

In a recent report, Greene et al. (1988) found decreased Mg flow past duodenal cannulas in steers receiving monensin, indicating an increase in preintestinal Mg absorption. These results tend to agree with findings from sheep in the present study. However, Greene et al. (1988) reported larger amounts of Mg secreted into the small intestine of monensin-fed steers, possibly in response to increased ruminal Mg absorption. These authors suggest that monensin may be increasing Mg absorption from the stomach, by increasing activity of the Na^+-K^+ pump. Martens et al. (1978) have demonstrated that Mg is actively transported by an ouabain-inhibited active transport system in rumen epithelial tissue, indicating the necessity of Na^+-K^+-ATPase. Monensin has been shown to increase the activity of the Na^+-K^+ pump in cell membranes (Smith and Rozengurt, 1978; Mendoza et al., 1980). Since monensin has been found to be absorbed by the ruminant (Davison, 1983), this ionophore, and possibly lasalocid, may act within the rumen epithelium to increase Mg absorption, by increasing Na^+-K^+ pump activity. Darden et al. (1985) reported an increase in preintestinal Mg absorption for steers fed lasalocid compared to monensin.

Feeding ionophores did not alter apparent absorption or retention of Na, compared to feeding no ionophore (Table 47). Urinary Na excretion decreased ($P<.05$) 29% for sheep fed lasalocid, compared to those fed no ionophore. Monensin tended to increase urinary Na excretion, compared to controls. Urinary Na excretion decreased ($P<.01$) 40.4% for lasalocid-fed sheep, compared to monensin-fed sheep, resulting in a 19.0%

TABLE 47. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF SODIUM IN SHEEP.

	Ionophore			
Item	None	Lasalocid	Monensin	SE
Intake, g/d	1.65	1.65	1.65	--
Excretion, g/d				
Fecal	.33	.46	.32	.06
Urine[ab]	.79	.56	.94	.05
Total[b]	1.12	1.02	1.26	.04
Apparent absorption				
Grams per day	1.33	1.19	1.33	.06
Percent of intake	80.61	72.12	80.61	1.08
Retention				
Grams per day[b]	.53	.63	.39	.04
Percent of intake[b]	32.17	38.25	23.58	2.68
Percent of absorbed[a]	40.69	53.04	29.00	3.01

[a]Control differs from lasalocid ($P<.05$).
[b]Lasalocid differs from monensin ($P<.01$).

decrease (P<.01) in total Na excretion. Lasalocid increased (P<.01) Na retention 61.5.% in g/d, when compared to monensin. Starnes et al. (1984) found both lasalocid and monensin addition to the diet increased the apparent absorption of Na in steers. Kirk et al. (1985a) reported increased urinary Na excretion and decreased Na retention for sheep fed monensin, compared to sheep fed no ionophore. O'Conner and Beede (1986) found a linear decrease in bovine duodenum mucosal Na concentrations when increased monensin levels were added to the mucosal bath of tissue placed in a Ussing chamber, indicating an increase in Na absorption.

There were no changes in digestive tract flow or partial absorption of Na when ionophores were fed (Table 48). A net secretion of Na occurred in the preintestinal region, with absorption occurring in the small and large intestines. Grace et al. (1974) found the small intestine to be the primary site of Na absorption in sheep. Greene et al. (1983), Rahnema and Fontenot (1983), and Wylie et al. (1985) found a net secretion of Na into the preintestine, with the large intestine as the primary site of Na absorption and net absorption by the small intestine in sheep. Lasalocid decreased (P<.10) large intestinal absorption of Na, expressed as a percentage of Na entering this region, by 3%, compared to monensin.

Fecal K excretion was decreased (P<.10) 8% with lasalocid and 21% with monensin feeding (Table 49). The addition of lasalocid and monensin to the basal diet resulted in small increases (P<.10) in the apparent absorption of K. All treatment groups were in net negative K balance, due to the large loss of K through the urine. Kirk et al. (1985a) reported a 17% increase in K apparent absorption, and 53% increase in K retention, when monensin was fed to sheep. Starnes et al. (1984) found no changes

TABLE 48. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF SODIUM IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	1.39	1.39	1.39	--
Flow, g/d				
Preintestine	14.14	12.89	14.89	.83
Small intestine	8.12	7.32	8.26	.55
Large intestine	.29	.43	.27	.07
Partial absorption				
Preintestine				
Grams per day	-12.74	-11.50	-13.50	.83
Percent of intake	--	--	--	--
Small intestine				
Grams per day	6.01	5.56	6.63	1.02
Percent of intake	431.62	399.43	475.46	73.63
Percent of entry	42.01	41.76	43.42	5.61
Large intestine				
Grams per day	7.83	6.89	8.00	.56
Percent of intake	562.02	494.81	573.90	40.25
Percent of entry[a]	96.44	93.94	96.66	1.02
Total				
Grams per day	1.10	.96	1.12	.07
Percent of intake	79.12	69.16	80.76	4.83

[a]Lasalocid differs from monensin ($P<.10$).

TABLE 49. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF POTASSIUM IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, g/d	5.19	5.19	5.19	--
Excretion, g/d				
Fecal[a]	.48	.44	.38	.03
Urine	6.11	5.93	5.76	.14
Total	6.59	6.37	6.14	.15
Apparent absorption				
Grams per day[a]	4.70	4.75	4.81	.03
Percent of intake[a]	90.70	91.56	92.74	.62
Retention				
Grams per day	-1.40	-1.18	-.95	.15
Percent of intake	---	---	---	--
Percent of absorbed	---	---	---	--

[a]Control differs from ionophores (P<.10).

in K metabolism in steers receiving lasalocid or monensin, and Spears et al. (1987) reported increased K absorption and retention in steers fed green-chopped tall fescue with 200 mg monensin$\bullet$hd$^{-1}\bullet$d^{-1}. Greene et al. (1986) found monensin increased the apparent absorption of K in ruminally-cannulated sheep. Both lasalocid and monensin are known to carry K^+ across cell membranes (Pressman, 1976), and may directly alter the absorption of K, due to their ionophore activity.

There were no treatment differences in the digestive tract flow or partial absorption of K in these wethers (Table 50). All animals showed a net secretion of K into the preintestinal region, with the small intestine as the primary site of K apparent absorption. Greene et al. (1983) found the site of K absorption in sheep was altered by level of K in the diet. At .6% K, a dietary K level similar to the present study, these workers reported a net secretion of K into the preintestine, with K absorption occurring primarily in the small intestine. Increasing K concentration in the diet brought about net absorption from the preintestine. Similar findings were made by Wylie et al. (1985), although K level was altered by infusion of $KHCO_3$ into the rumen, abomasum or ileum. Rahnema and Fontenot (1983) found the small intestine was the primary site of K absorption, but whether K was absorbed or secreted into the preintestine was dependent upon Mg source in the diet. Other reports have consistently demonstrated the small intestine as the primary site of K absorption (Grace et al., 1974; Dillon et al., 1979).

Lasalocid and monensin did not alter the apparent absorption or retention of Cu in wethers when added to the basal diet (Table 51). Apparent absorption and retention of Cu was approximately 95% of intake

TABLE 50. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF POTASSIUM IN SHEEP.

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Intake, g/d	4.80	4.80	4.80	--
Flow, g/d				
Preintestine	8.60	8.83	8.41	.49
Small intestine	1.31	1.26	1.31	.11
Large intestine	.40	.42	.40	.04
Partial absorption				
Preintestine				
Grams per day	-3.81	-4.03	-3.62	.49
Percent of intake	--	--	--	--
Small intestine				
Grams per day	7.29	7.56	7.10	.49
Percent of intake	151.96	157.70	147.98	1.02
Percent of entry	84.84	85.66	83.99	1.41
Large intestine				
Grams per day	.92	.84	.91	.13
Percent of intake	19.09	17.54	18.97	2.79
Percent of entry	68.54	65.93	68.56	4.54
Total				
Grams per day	4.40	4.38	4.39	.04
Percent of intake	91.64	91.15	91.51	.74

TABLE 51. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF COPPER IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, mg/d	8.87	8.87	8.87	--
Excretion, mg/d				
Fecal	.40	.45	.49	.05
Urine	.05	.05	.05	0
Total	.45	.50	.54	.05
Apparent absorption				
Milligrams per day	8.47	8.42	8.38	.05
Percent of intake	95.54	94.93	94.44	.60
Retention				
Milligrams per day	8.42	8.37	8.32	.05
Percent of intake	94.97	94.34	93.88	.59
Percent of absorbed	99.41	99.38	99.40	.03

for all treatment groups. Little Cu was excreted in the urine. Previous research has suggested changes in Cu metabolism when ionophores are fed. Elsasser (1984) reported increased liver Cu in sheep receiving 30 mg/d of lasalocid or monensin in the diet. Sappington et al. (1987) also found increased liver Cu levels in sheep receiving 27.5 mg/kg lasalocid or monensin. The liver is the primary Cu storage tissue, and increased liver Cu levels suggest increased Cu retention. Starnes et al. (1984) reported increased serum Cu concentration in steers fed lasalocid or monensin.

Adding lasalocid or monensin to the basal diet did not alter site or extent of Cu apparent absorption in sheep (Table 52). However, lasalocid increased ($P<.05$) Cu absorbed from the small intestine, relative to Cu entering this region, by 42%, compared to monensin. Lasalocid decreased ($P<.05$) large intestinal apparent absorption of Cu 62%, compared to monensin. The preintestinal region was the primary site of apparent absorption of Cu, with approximately 90% of Cu ingested being absorbed in this area. Grace (1975) found no significant absorption or secretion of Cu in the stomach or small intestine of sheep, with net absorption occurring in the large intestine. Elsasser (1984) examined the effects of lasalocid and monensin upon ^{64}Cu uptake by chicken duodenal tissue. Lasalocid was found to increase mucosal ^{64}Cu counts, while monensin decreased counts, indicating increased Cu absorption with lasalocid, and decreased absorption with monensin. These findings are consistent with differences between the ionophores in Cu absorption in the small intestine, seen in the present study.

Apparent absorption of Fe was increased ($P<.10$) when lasalocid and monensin were added to the basal diet (Table 53). These findings resulted

TABLE 52. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF COPPER IN SHEEP.

	Ionophore			
Item	None	Lasalocid	Monensin	SE
Intake, mg/d	8.20	8.20	8.20	--
Flow, mg/d				
Preintestine	.70	.74	.89	.10
Small intestine	.46	.43	.63	.08
Large intestine	.38	.40	.54	.07
Partial absorption				
Preintestine				
Milligrams per day	7.50	7.46	7.31	.10
Percent of intake	91.50	90.99	89.18	1.23
Small intestine				
Milligrams per day	.23	.31	.26	.04
Percent of intake	2.84	3.80	3.14	.48
Percent of entry[a]	32.77	42.03	29.59	3.33
Large intestine				
Milligrams per day[a]	.08	.03	.08	.02
Percent of intake[a]	1.00	.35	1.03	.19
Percent of entry	16.82	7.42	12.87	3.38
Total				
Milligrams per day	7.82	7.80	7.66	.07
Percent of intake	95.34	95.14	93.36	.90

[a]Lasalocid differs from monensin ($P<.05$).

TABLE 53. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF IRON IN SHEEP.

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Intake, mg/d	55.69	55.69	55.69	--
Excretion, mg/d				
Fecal[a]	12.06	8.50	10.35	1.12
Urine	1.69	1.63	2.52	.44
Total	13.75	10.13	12.87	1.32
Apparent absorption				
Milligrams per day[a]	43.62	47.19	45.34	1.12
Percent of intake[a]	78.33	84.74	81.42	2.01
Retention				
Milligrams per day	41.94	45.55	42.82	1.32
Percent of intake	75.30	81.80	76.89	2.37
Percent of absorbed	96.03	96.51	94.47	1.01

[a]Control differs from ionophores ($P<.10$)

from a decrease ($P<.10$) in fecal Fe excretion of 30% in lasalocid-fed sheep, and 14% in monensin-fed sheep, compared to animals fed no ionophore. There were no differences between treatment groups for Fe retention. Few studies have examined the effects of ionophores upon Fe metabolism. However, Elsasser (1984) reported increased ^{59}Fe uptake by chicken duodenal tissue with lasalocid addition, and decreased ^{59}Fe uptake when monensin was added, indicating increased absorption of Fe with lasalocid and decreased absorption with monensin.

Feeding lasalocid and monensin did not alter the digestive tract flow or partial absorption of Fe in sheep (Table 54). Apparent absorption occurred primarily in the preintestinal region. Absorption of Fe occurred in the small intestine, with large intestinal secretion of Fe in control-fed sheep and some Fe absorption in the large intestine of sheep fed ionophores.

Lasalocid and monensin did not significantly alter apparent absorption and retention of Zn in sheep (Table 55). Dietary Zn was low, approximately 10 ppm,with the Zn requirement for lambs being 20 to 33 ppm (NRC, 1985). Urinary Zn excretion was relatively high, particularly for ionophore-fed sheep. Urinary Zn excretion is typically low in ruminants, although it may increase when chelating agents are fed (Powell et al., 1967). Kirk et al. (1985b) reported large increases in apparent absorption and retention of Zn in sheep receiving 20 ppm monensin. Greene et al. (1986) reported a 26.0% increase in apparent absorption of Zn by steers fed 25 ppm monensin. Costa et al. (1985) fed 125 mg monensin/d to steers receiving hay and straw in the diet, resulting in a 52.4%

TABLE 54. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF IRON IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, mg/d	47.30	47.30	47.30	---
Flow, mg/d				
Preintestine	17.63	16.02	16.20	4.18
Small intestine	10.29	9.56	10.13	1.24
Large intestine	10.97	9.50	9.54	1.07
Partial absorption				
Preintestine				
Milligrams per day	29.67	31.28	31.10	4.18
Percent of intake	62.73	66.12	65.74	8.83
Small intestine				
Milligrams per day	7.34	6.46	6.07	4.07
Percent of intake	15.52	13.66	12.84	8.60
Percent of entry	28.58	33.31	31.97	12.29
Large intestine				
Milligrams per day	-.68	.06	.06	.52
Percent of intake	---	.13	1.25	1.09
Percent of entry	---	1.56	5.86	5.39
Total				
Milligrams per day	36.33	37.80	37.76	1.07
Percent of intake	76.80	79.92	79.83	.23

TABLE 55. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON APPARENT ABSORPTION AND RETENTION OF ZINC IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, mg/d	7.95	7.95	7.95	--
Excretion, mg/d				
Fecal	2.06	2.08	2.15	.14
Urine	.41	1.29	1.09	.36
Total	2.47	3.37	3.24	.44
Apparent absorption				
Milligrams per day	5.90	5.87	5.80	.14
Percent of intake	74.14	73.81	72.92	1.74
Retention				
Milligrams per day	5.48	4.58	4.71	.44
Percent of intake	68.92	57.58	59.19	5.56
Percent of absorbed	92.83	77.19	81.33	6.57

increase in Zn retention, as measured by a Compton Whole Body Counter following $^{65}ZnCl_2$ oral dose.

Sheep fed ionophores had no significant changes in flow or partial absorption of Zn compared to sheep fed the control diet (Table 56). Lasalocid decreased ($P<.05$) flow of Zn through the small intestine 12%, compared with monensin. Apparent absorption of Zn occurred mainly in the preintestinal region, with slight absorption in the small intestine, and net Zn secretion into the large intestine of sheep. Lasalocid increased ($P<.01$) Zn secretion into the large intestine by 20%, compared with monensin. Grace (1975) has demonstrated a net secretion of Zn into the preintestinal region of sheep, with nearly equal Zn absorption from the small intestine and large intestine. However, Miller and Cragle (1965) reported as much as one-third daily Zn intake could be absorbed from the abomasum.

The results of this study indicate that lasalocid and monensin may alter the digestive tract flow and partial absorption of minerals in sheep, particularly for Mg. Although the site of Mg absorption was not changed by ionophore addition to the diet, the flow of Mg through segments of the gut was altered. Greater differences in flow and partial absorption of minerals were observed when comparing lasalocid and monensin. These differences may be due to differences in ion selectivity of these ionophores, with lasalocid capable of carrying both monovalent and divalent cations, and monensin an exclusively monovalent ionophore (Pressman, 1976).

TABLE 56. EFFECT OF FEEDING LASALOCID AND MONENSIN UPON DIGESTIVE TRACT FLOW AND PARTIAL ABSORPTION OF ZINC IN SHEEP.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Intake, mg/d	7.95	7.95	7.95	--
Flow, mg/d				
Preintestine	1.88	1.86	1.83	.16
Small intestine[a]	1.83	1.76	1.99	.07
Large intestine	1.92	1.88	2.00	.06
Partial absorption				
Preintestine				
Milligrams per day	6.52	6.54	6.57	.16
Percent of intake	77.62	77.80	78.17	1.88
Small intestine				
Milligrams per day	.05	.11	.15	.11
Percent of intake	.57	1.26	1.84	1.27
Percent of entry	1.01	3.28	9.95	5.36
Large intestine				
Milligrams per day[b]	-.09	-.12	-.10	.02
Percent of intake	---	---	---	---
Percent of entry	---	---	---	---
Total				
Milligrams per day	6.48	6.52	6.40	.06
Percent of intake	77.14	77.60	76.21	.74

[a]Lasalocid differs from monensin ($P<.05$).
[b]Lasalocid differs from monensin ($P<.01$).

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JOURNAL ARTICLE NO. IV.

EFFECTS OF FEEDING LASALOCID AND MONENSIN UPON SERUM MINERAL CONCENTRATION AND RENAL HANDLING OF MINERALS IN SHEEP.

ABSTRACT

Twelve ewe lambs were used in a randomized block design experiment to determine the effects of feeding lasalocid and monensin upon serum mineral concentration and renal handling of minerals in sheep. Sheep were blocked by weight and allotted to three dietary treatments consisting of a control diet with the the following additives: none, 23 ppm lasalocid, or 23 ppm monensin, dry basis. Sheep were kept in stainless steel metabolism crates, with _ad libitum_ access to deionized water. The experiment consisted of a 5-d adjustment period, a 7-d preliminary period, and a 9-d experimental period. Serial collections of blood and urine were made during d 1 and d 5 of the experimental period to determine renal function and renal handling of minerals. Urine was collected using bladder catheters at 0, 30, 60, 120, 180, 240, 300, and 360 min after feeding. Blood was collected via in-dwelling jugular catheters at -30, 15, 45, 90, 150, 210, 270, and 330 min after feeding. Serum clearance of creatinine was used to determine glomerular filtration rate (GFR). During d 1, monensin reduced ($P<.01$) urine flow rate, compared to lasalocid. Urine flow rate at d 5 was reduced in sheep fed monensin, compared to those fed no ionophore ($P<.01$), or lasalocid ($P<.05$). There were no differences in GFR during d 1 or 5. Feeding ionophores increased serum inorganic P concentration on d 1 ($P<.10$), and during d 1 through 9

($P<.05$). Dietary ionophores reduced serum K ($P<.05$) and Ca ($P<.10$) during d 1 through 9. Serum clearance and urinary excretion rates of Na and K, during d 5, were lower in sheep fed monensin, than those fed no ionophore ($P<.01$), or lasalocid ($P<.05$). During d 1, serum clearance rate and urinary excretion rates of Ca were reduced ($P<.05$) when ionophores were fed. During d 1, sheep fed monensin had lower ($P<.05$) serum clearance rates of Cu than those fed no ionophore or lasalocid. Sheep fed lasalocid had greater ($P<.01$) Cu clearance rates than those fed no ionophore. Urinary Cu excretion was reduced ($P<.01$) during d 1 in sheep fed monensin vs. those fed no ionophore or lasalocid. During d 1, serum clearance and urinary excretion rates of Fe were lower ($P<.05$) in sheep fed monensin than those fed lasalocid. During d 5, urinary excretion rates of Zn were greater ($P<.05$) when lasalocid was fed, compared to feeding no ionophore or monensin. Feeding monensin lowered ($P<.01$) urinary Zn excretion during d 5, compared to feeding no ionophore. Differences in these variables attributed to dietary treatments may be partly the result of differences in DM and mineral intake. Differences in effects of ionophores between d 1 and d 5 may indicate physiological adaptation to dietary lasalocid and monensin. These results indicate that dietary ionophores alter renal handling of minerals in sheep, which may be responsible for some of the changes in mineral metabolism seen when these are fed to ruminants.
(KEY WORDS: Lasalocid, Monensin, Sheep, Minerals, Renal Clearance)

INTRODUCTION

Lasalocid and monensin are ionophores commonly supplemented to ruminants to increase feed efficiency and(or) rate of gain. The main effect of these ionophores is to promote an energetically favorable shift in ruminal fermentation, with decreased acetate and increased propionate production. However, the primary action of these compounds is to alter the flow of cations across cell membranes (Pressman, 1976). Presumably due to this effect, both lasalocid and monensin have been demonstrated to alter mineral metabolism in cattle (Starnes et al., 1984) and sheep (Kirk et al., 1985a,b; Greene et al., 1986).

These ionophores have been shown to alter urinary excretion of minerals in ruminants. Starnes et al. (1984) reported a trend towards decreased urinary excretion of Na and K in steers receiving 33 ppm dietary lasalocid or monensin. Kirk et al. (1985a) reported an increase in urinary excretion of Na in sheep fed 20 ppm monensin. Feeding monensin was shown to reduce urinary Ca excretion, and tended to reduce urinary Mg excretion in sheep (Kirk et al., 1985b).

The present study was conducted to determine the effects of feeding ionophores upon renal function and renal handling of minerals in sheep. The experiment also examined the possible changes in serum mineral concentrations over time, following ionophore feeding. Previous research has examined serum mineral concentration changes over time in response to an i.v. dose of ionophores (Armstrong and Spears, 1988).

EXPERIMENTAL PROCEDURE

Twelve ewe lambs (avg wt 38.6 kg) were used in a randomized block design experiment to determine the effects of feeding lasalocid and monensin upon serum mineral concentration and renal handling of minerals in sheep. Sheep were blocked according to weight, and allotted to dietary treatments consisting of a control diet alone or supplemented with 23 ppm lasalocid, or 23 ppm monensin, dry basis (Table 57). Sheep were kept in stainless steel metabolism crates, designed for the total collection of feces and urine. Deionized water was available ad libitum.

One block of animals was placed on experiment each day (3 sheep/day), each animal receiving a different dietary treatment. The experiment began with a 5-d period for adjustment to metabolism crates and diets. Sheep then received 690 g DM of the control diet for a 7-d preliminary period. A 9-d experimental period began following the preliminary period. Throughout this period, sheep were fed 291 g (dry basis) of the diets at 0800 h and 0900 h each day (total of 582 g), advancing 5 min for each animal. Animals first received their experimental diets at 0800 h on d 1 of the experimental period. Ionophores were provided only during the first feeding of each day. These diets contained no ionophore, 46 ppm lasalocid, or 46 ppm monensin. During the second daily feeding, all sheep were fed the diet containing no ionophore. There were no refusals of feed containing ionophores during the study, although, at times, there were refusals following the second daily feeding.

Jugular catheters were installed 12 h prior to feeding at d 1, and were maintained with heparinized saline. Sheep were fitted with 12 F Foley bladder catheters 2 h before beginning the collection. Bladder catheters were flushed with 30 ml sterile, deionized water 60 min before feeding, urine and water were discarded, and urine collection initiated. Renal clearance collection was conducted at d 1. Urine was collected at 0, 30, 60, 120, 180, 240, 300 and 360 min after feeding. At the end of

TABLE 57. INGREDIENT AND MINERAL COMPOSITION OF DIET.

Item	Concentration
Ingredient composition[a]	
Orchardgrass hay	53.0
Corn grain	38.2
Molasses	5.0
Soybean meal	3.0
Trace mineralized salt	.5
Limestone	.3
Mineral concentration[b]	
Calcium, %	1.65
Phosphorus, %	.391
Magnesium, %	.186
Sodium, %	.246
Potassium, %	1.54
Copper, ppm	8.28
Iron, ppm	18.4
Zinc, ppm	3.85

[a]Percent as-fed basis.
[b]Dry basis.

each collection period, bladder catheters were flushed with 30 ml sterile, deionized water, urine volume measured, and urine was sampled. Urine was immediately placed on ice and later frozen for subsequent analysis. Blood samples were collected 30 min before the first feeding and at the midpoint of each urine collection period (15, 45, 90, 150, 210, 270 and 330 min). Blood was immediately placed on ice, and later centrifuged, serum removed and frozen for subsequent analysis.

A renal clearance collection was repeated at d 5 of the experimental period. Sample collection was identical to that at d 1. Blood samples were also collected 330 min after feeding at d 2, 3, 4 and 9 of the study.

Feed and orts samples were ground through a 1 mm screen in a stainless steel Wiley mill, prior to wet digestion in nitric and perchloric acid (Sandel, 1950). Feed, orts, serum and urine were analyzed for Na, K, Mg, Ca, Cu, Fe and Zn using atomic absorption spectrophotometry. Inorganic phosphorus was determined using a colorimetric procedure (Fiske and Subarrow, 1925). Serum and urine were also analyzed for creatinine concentration[1]. Serum aldosterone was determined by radioimmunoassay[2].

Clearance rates of creatinine and minerals were determined by the formula (Pitts, 1974): $(Ux * V)/Px$ where:

[1]Sigma kit #555 creatinine procedure. Sigma Chemical Company, St. Louis, MO 63178-9916

[2]Coat-a-count aldosterone kit. Diagnostics Products Corportation, Los Angeles, CA 90045

Ux = urinary concentration of compound(mg/100 ml)

V = urine excreted/min(ml/min)

Px = plasma concentration of compound(mg/100ml)

Clearance rate refers to milliliters of plasma completely cleared of compound in 1 min. Since creatinine is freely filtered through the glomerular capillary membranes, and is neither secreted nor reabsorbed by the renal tubules, the clearance rate of creatinine can be used to estimate GFR (the ml of plasma filtered by the glomerulus/min). Filtered plasma load of minerals (mg filtered by glomerulus/min) was calculated by multiplying the serum concentration of mineral by the GFR. Net tubular reabsorption of minerals was calculated by subtracting the urinary excretion rate of the mineral from the filtered load of the mineral.

Data were analyzed using the General Linear Model procedure of the Statistical Analysis System (SAS, 1986). The model used for the renal clearance data was: Y = u + a (block) + b(treatment) + c(animal[block x treatment]) + d(time) + e(treatment x time) + experimental error. Block and treatment effects were tested using animal(block x treatment) effects as the error term. Data obtained at initial sampling times was excluded from the statistical analysis. Orthogonal contrasts were control vs. ionophore-fed sheep, and lasalocid- vs. monensin-fed sheep. Dunnett's test for mean comparison to controls was conducted when the effects of ionophores were opposite in relation to those of the control (Zar, 1984).

RESULTS AND DISCUSSION

During d 1, urine flow rate was lower in sheep fed monensin than in sheep fed lasalocid ($P<.01$), and tended to be lower than those fed no ionophore (Table 58). On d 5, sheep fed monensin had lower urine flow rates than sheep fed no ionophore ($P<.01$) or lasalocid ($P<.05$). Glomerular filtration rate was not altered by feeding ionophores during d 1 or d 5. Values for GFR were lower than those reported previously in sheep (Ergene and Pickering, 1978a,b; DeGregorio et al., 1981). Altered urine flow with no change in GFR may indicate altered reabsorption of water in sheep fed monensin. Serum creatinine concentration was not different in sheep fed with or without ionophores, during d 1 or d 5. During d 1 urinary creatinine excretion was greater ($P<.05$) in sheep fed lasalocid than those fed monensin, during d 1.

Serum Na concentration was not affected by feeding lasalocid or monensin on d 1 or d 5, or during d 1 through 9 (Table 59). In previous studies no changes were found in serum Na concentration in ruminants fed ionophores, although _in vivo_ and _in vitro_ research indicates that ionophores increase digestive tract absorption of Na (Starnes et al., 1984; O'Conner and Beede, 1986). In the present study, sheep fed lasalocid had greater ($P<.10$) Na intake on d 1 and 5 than those fed monensin, and Na intake tended to be greater for sheep fed lasalocid vs. no ionophore (Appendix Table 16).

Serum K concentration tended to be lower in sheep after feeding lasalocid or monensin, compared to no ionophore, during d 1 and d 5 (Table 60). Serum K concentration was lower ($P<.05$) in sheep fed ionophores during d 1 through d 9. Armstrong and Spears (1988) reported that serum K concentration was reduced in cattle following i.v. administration of monensin, but lasalocid had no effect. Spears and Harvey (1984) found that feeding lasalocid decreased serum K at 112 d in grazing cattle.

TABLE 58. URINE FLOW RATES, GLOMERULAR FILTRATION RATES, AND SERUM AND URINE CREATININE CONCENTRATION IN SHEEP FED WITH OR WITHOUT IONOPHORES

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Urine flow, ml/min				
Day 1[a]	1.12	1.14	.85	.04
Day 5[ab]	.90	.99	.69	.03
Glomerular filtration rate, ml/min				
Day 1	59.41	67.61	59.38	2.99
Day 5	56.23	54.53	54.69	2.38
Serum creatinine, mg/100 ml				
Day 1	.951	.883	.918	.014
Day 5	1.033	.978	1.004	.009
Urinary creatinine, mg/min				
Day 1[a]	55.94	57.49	52.09	2.09
Day 5	57.58	51.93	53.94	2.04

[a]Lasalocid differs from monensin ($P<.05$).
[b]Control differs from monensin ($P<.01$).

TABLE 59. SERUM SODIUM CONCENTRATION AND RENAL HANDLING OF SODIUM IN SHEEP FED A BASAL DIET WITH OR WITHOUT IONOPHORES

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Serum Na, mg/100 ml				
Day 1	298.0	297.2	299.2	1.8
Day 5	312.6	316.7	304.6	3.9
Day 1-9	313.5	307.6	309.1	3.7
Serum Na clearance, ml/min				
Day 1	.759	.524	.306	.087
Day 5[abc]	.294	.294	.090	.040
Urinary Na excretion, mg/min				
Day 1	2.396	1.661	.941	.248
Day 5[abc]	.960	.940	.275	.130
Na filtered load, mg/min				
Day 1	178.9	205.3	174.5	9.5
Day 5	177.5	172.8	166.2	7.6
Na net tubular reabsorption, mg/min				
Day 1	176.6	203.8	173.7	9.5
Day 5	176.6	172.3	163.5	7.4

[a]Time x treatment interaction ($P<.10$).
[b]Control differs from monensin ($P<.01$).
[c]Lasalocid differs from monensin ($P<.05$).

TABLE 60. SERUM POTASSIUM CONCENTRATION AND RENAL HANDLING OF POTASSIUM IN SHEEP FED A BASAL DIET WITH OR WITHOUT IONOPHORES

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Serum K, mg/100 ml				
Day 1	20.70	20.45	19.46	.24
Day 5	22.16	21.80	20.45	.37
Day 1-9[a]	22.30	21.06	21.46	.38
Serum K clearance, ml/min				
Day 1	28.52	34.21	26.73	1.69
Day 5[bcd]	33.08	33.68	21.79	1.51
Urinary K excretion, mg/min				
Day 1	5.872	6.707	5.347	.300
Day 5[cde]	7.282	7.330	4.456	.307
K filtered load, mg/min				
Day 1[d]	12.33	14.09	11.30	.61
Day 5	12.62	11.95	11.03	.52
K net tubular reabsorption, mg/min				
Day 1	6.571	7.028	5.976	.684
Day 5	5.260	4.726	6.571	.534

[a]Control differs from ionophores (P<.05).
[b]Time x treatment interaction (P<.10).
[c]Control differs from monensin (P<.01).
[d]Lasalocid differs from monensin (P<.05).
[e]Time x treatment interaction (P<.01).

Edlin et al. (1984) also reported decreased serum K in finishing steers fed lasalocid or monensin. Kirk et al. (1985a) found monensin increased apparent absorption and retention of K in wethers. Greene et al. (1986) also reported increased apparent absorption of K in sheep fed monensin. In the present study, sheep fed ionophores had greater ($P<.10$) K intake than controls on d 1 (Appendix Table 16). Sheep fed lasalocid had greater K intake than those fed monensin on d 1 ($P<.05$), and d 5 ($P<.05$).

Sodium and K homeostasis is maintained through the action of aldosterone (Blair-West et al., 1963). Serum aldosterone concentration tended to be higher in sheep fed ionophores than those fed no ionophore during d 1 and d 5 (Table 61). A trend toward increased serum aldosterone in sheep fed ionophores was also seen in samples obtained on d 1 through 5. Serum aldosterone increases in ruminants in response to decreased serum Na (Blair-West et al., 1971; Bell et al., 1981) and increased serum K (Blair-West et al., 1963). Aldosterone has been demonstrated to decrease urinary Na excretion in ruminants (Kumar and Singh, 1981; Riad et al., 1981), but has not been shown to alter serum Na or K concentration (Safwate et al., 1982; Riad et al., 1986).

During d 1, serum clearance rate and urinary excretion rate of Na tended to be lower when ionophores were fed (Table 59). During d 5, sheep fed monensin had lower serum clearance and urinary excretion rates of Na than those fed no ionophore ($P<.01$) or lasalocid ($P<.05$). There were significant time x treatment interactions ($P<.10$) for serum clearance and urinary excretion rates of Na during d 5 (Fig. 2 and 3). Serum clearance and urinary excretion rates of Na were similar for all treatment groups until 180 min after feeding, after which sheep fed no ionophore or

TABLE 61. SERUM ALDOSTERONE CONCENTRATION IN SHEEP FED WITH OR WITHOUT IONOPHORES

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Serum aldosterone, ng/100 ml				
Day 1	6.64	11.17	8.65	.67
Day 5	12.41	15.16	16.35	1.00
Day 1-5	8.22	9.80	9.41	.91

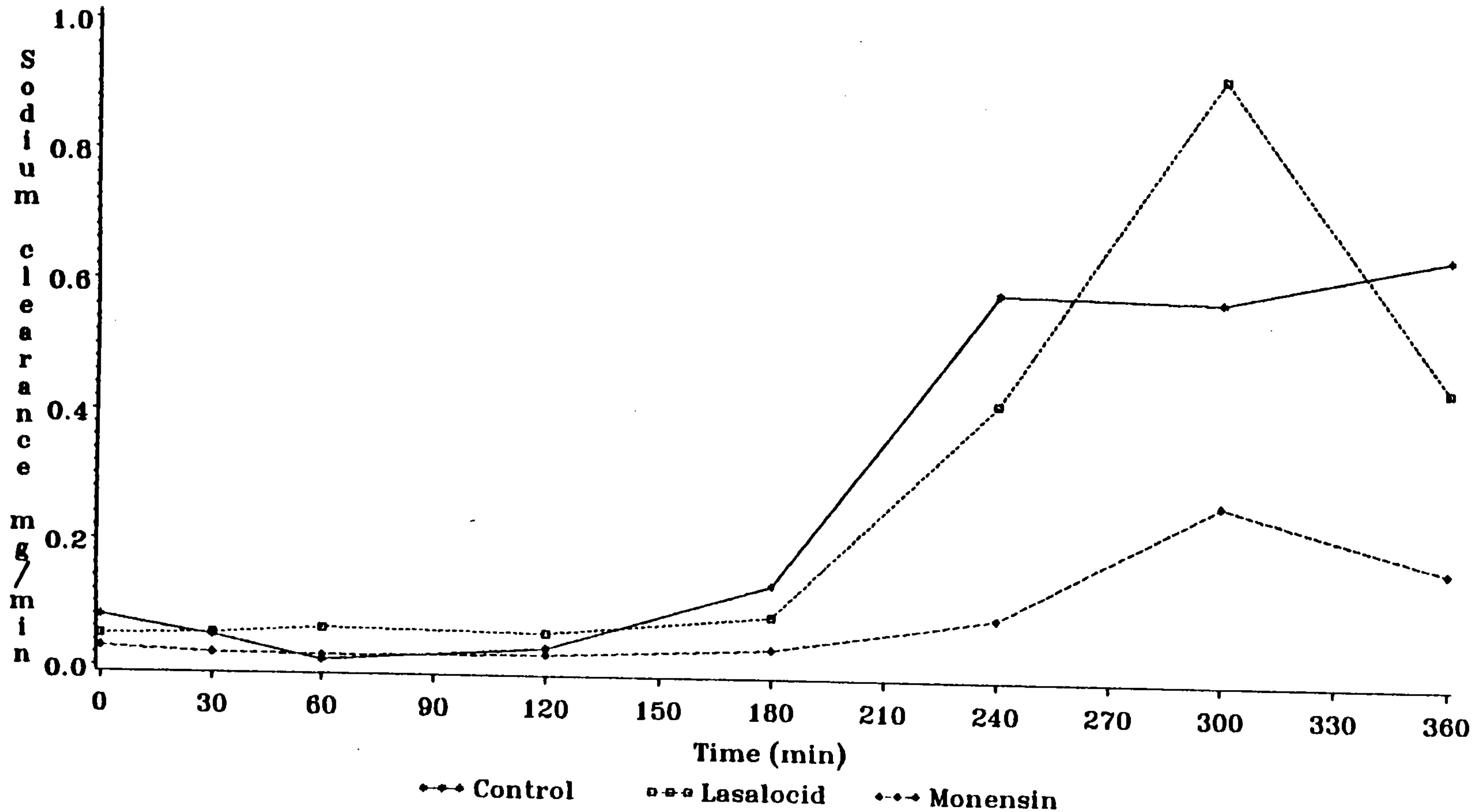

Figure 2. Serum clearance of sodium in sheep (day 5). Time x treatment interaction ($P<.10$). Control differs from monensin ($P<.01$). Lasalocid differs from monensin ($P<.05$). SE=.040.

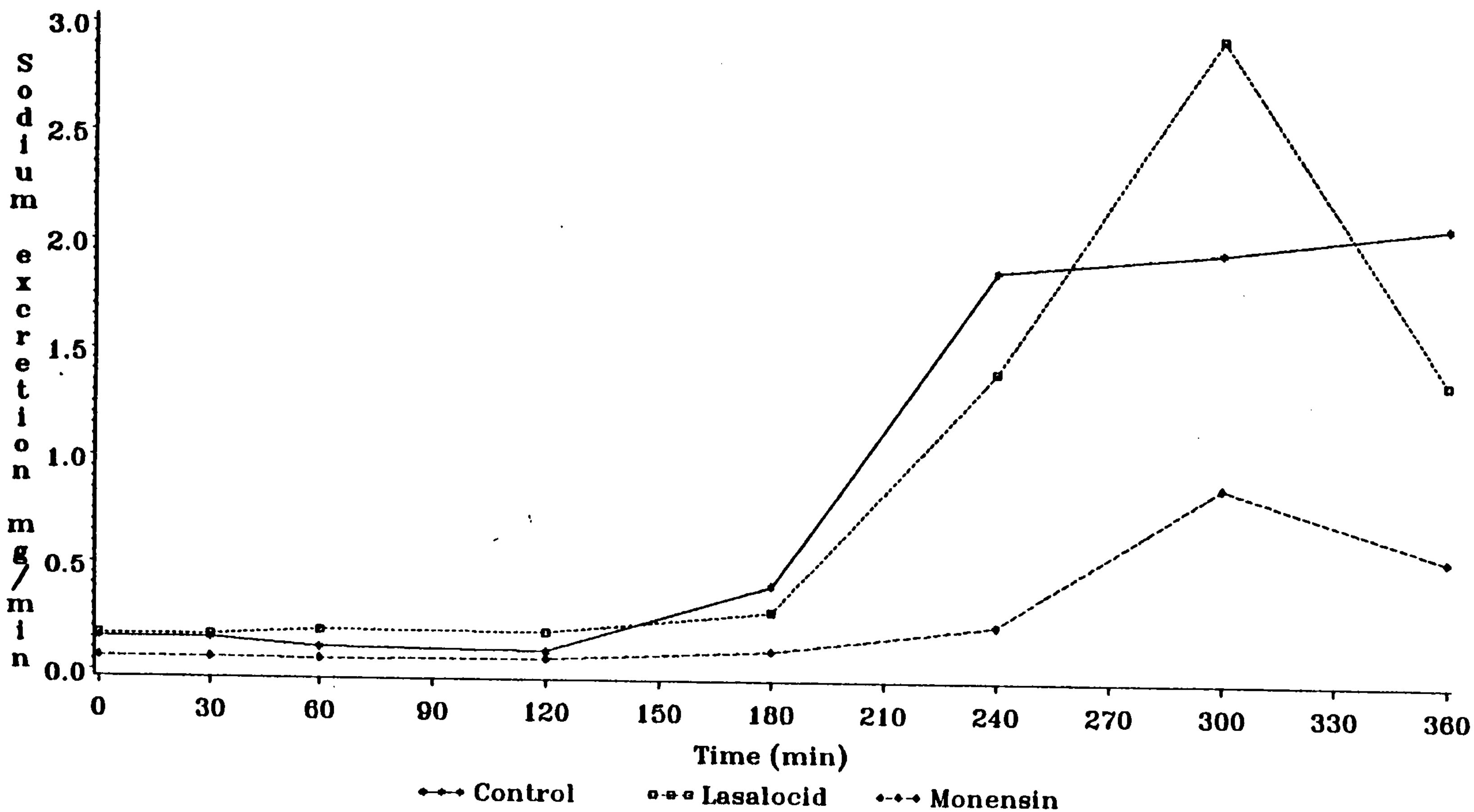

Figure 3. Urinary sodium excretion rate in sheep (day 5). Time x treatment interaction (P<.10). Control differs from monensin (P<.01). Lasalocid differs from monensin (P<.05). SE=.130

lasalocid had much greater increases in these variables than those fed monensin. Decreased values for these variables when monensin was fed may be related to increased serum aldosterone demonstrated when feeding ionophores. Kumar and Singh (1981) reported a significant depression of urinary Na concentration 60 min after i.v. aldosterone administration in buffalo calves. A decrease in serum clearance rate of Na would indicate reduced renal efficiency in clearing the blood of Na^+ (a problem during high Na intake), or an improved ability to conserve Na^+ (an advantage during depressed Na status). In the present study, there were no changes in the filtered load or net tubular reabsorption of Na during d 1 or d 5 (Table 59).

Serum clearance rate of K tended to be greater in sheep fed lasalocid than those fed no ionophore or monensin, during d 1 (Table 60). During d 5, serum clearance of K was reduced in monensin-fed sheep, compared to those fed no ionophore ($P<.01$) or lasalocid ($P<.05$). These changes correspond, in part, with changes in urinary excretion of K. During d 1, sheep fed lasalocid tended to have greater urinary excretion of K than those fed no ionophore or monensin. During d 5, sheep fed monensin had lower urinary excretion rates of K than those fed no ionophore ($P<.01$) or lasalocid ($P<.05$). There was a time x treatment interaction for K clearance rate ($P<.10$) (Fig. 4), and urinary excretion rate of K ($P<.01$) (Fig. 5), during d 5. Although sheep fed monensin had lower K clearance and excretion rates throughout sampling, the greatest differences were seen after 120 min.

Filtered load of K was reduced ($P<.05$) when monensin was fed, compared to feeding lasalocid, during d 1 (Table 60). There were no

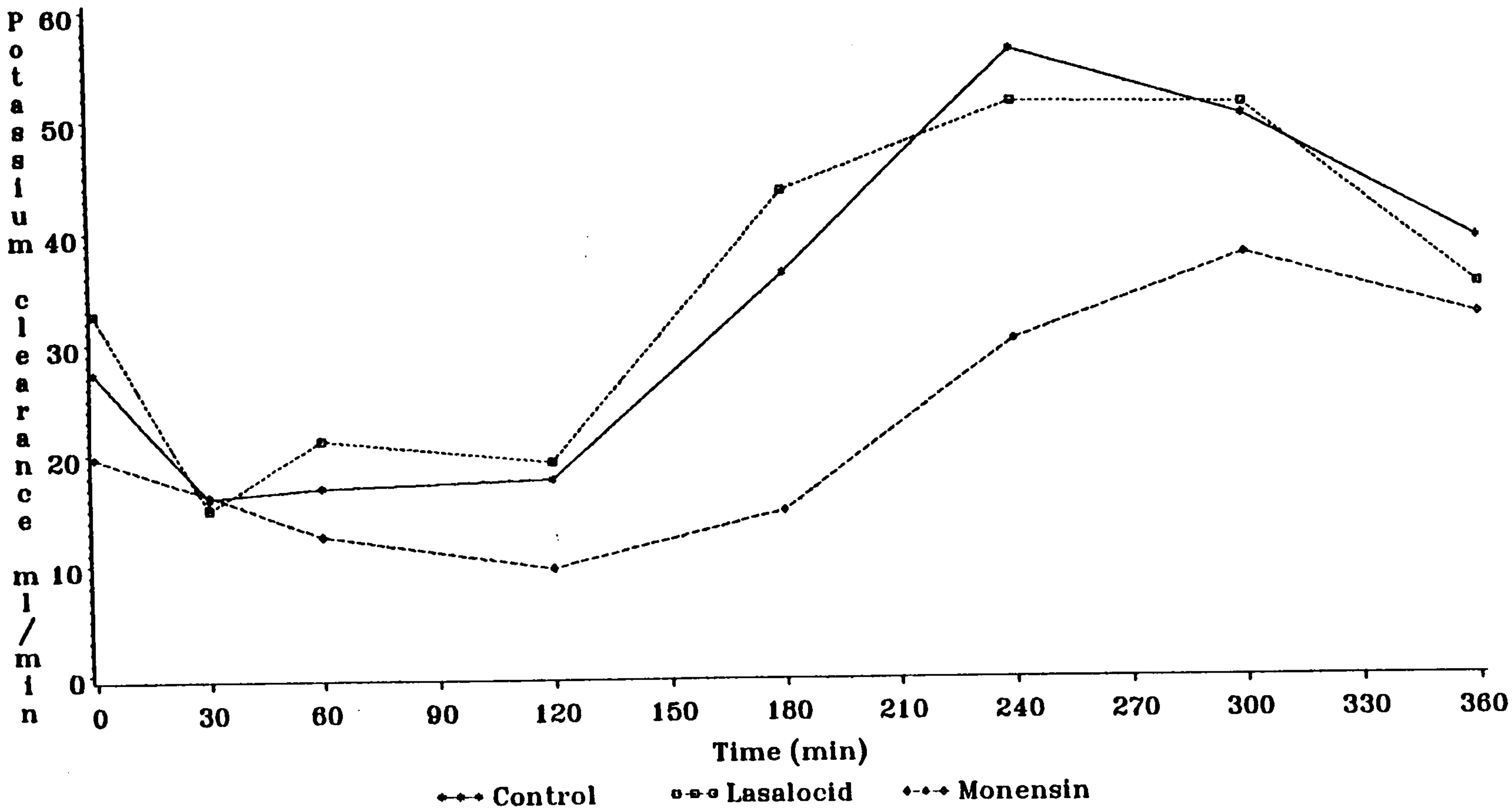

Figure 4. Serum clearance rate of potassium in sheep (day 5). Time x treatment interaction (P<.10). Control differs from monensin (P<.01). Lasalocid differs from monensin (P<.05). SE=1.51.

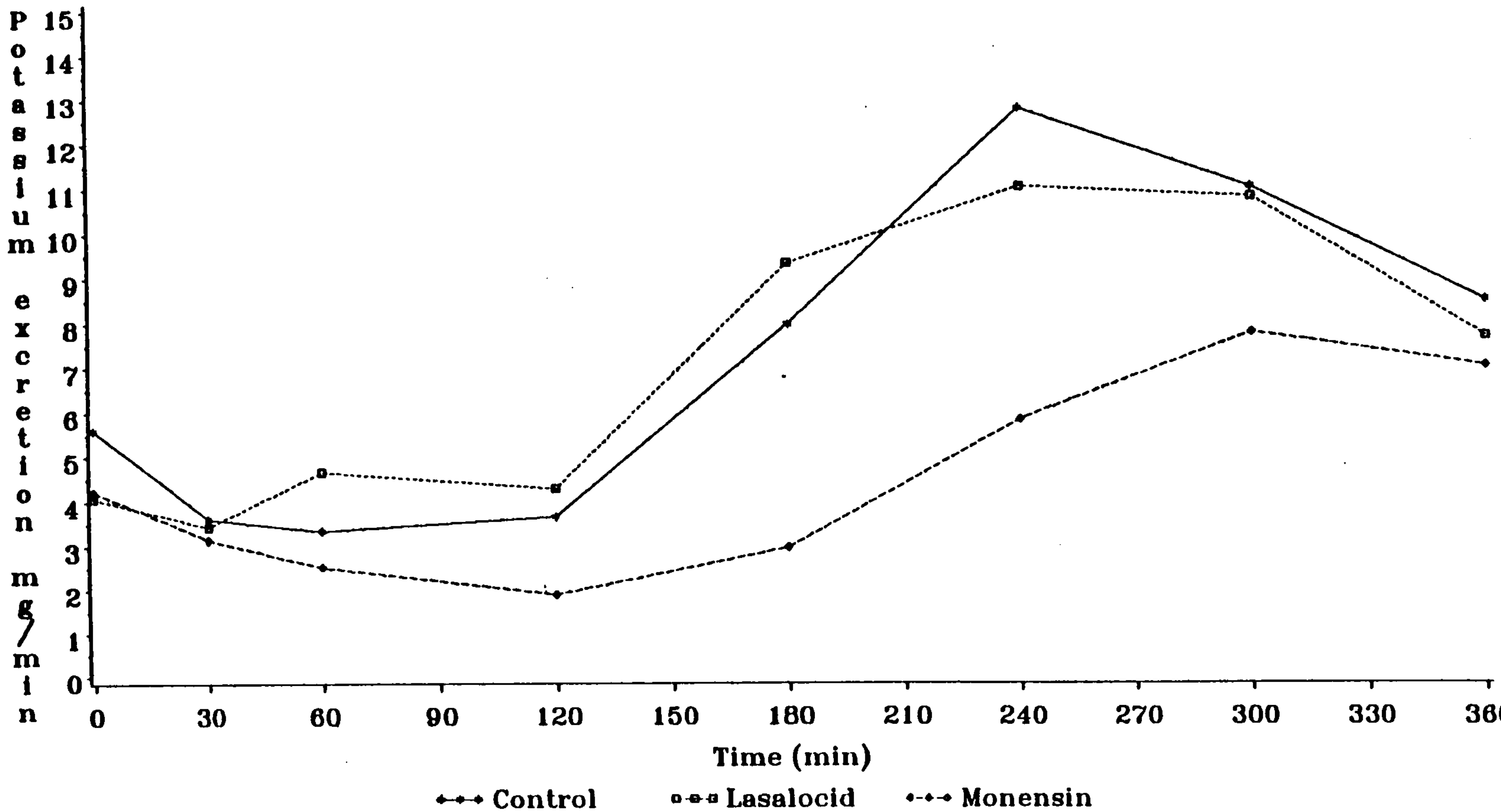

Figure 5. Urinary excretion rate of potassium in sheep (day 5). Time x treatment interaction ($P<.01$). Control differs from monensin ($P<.01$). Lasalocid differs from monensin ($P<.05$). SE=.307.

differences in filtered load of K at d 5, or in net tubular reabsorption of K at d 1 or d 5. Riad et al. (1986) have reported no change in urinary K excretion following aldosterone administration to cattle, and suggested that the effects of the hormone in increasing tubular Na^+ reabsorption are not associated with inceased K excretion. However, Kumar and Singh (1981) reported increased urinary K concentration in buffalo calves following i.v. aldosterone administration. Kelley and Preston (1986) found monensin decreased endogenous urinary K in cattle.

Serum Mg concentration was not altered when ionophores were fed on d 1 or d 5, or during d 1 through 9 (Table 62). Mean values were slightly lower when ionophores were fed. There was a time x treatment interaction for serum Mg concentration during d 5 (Fig. 6). Sheep fed monensin had slightly lower serum Mg concentration than controls throughout the collection. Sheep fed lasalocid had serum Mg levels greater than or equal to controls at 15, 90, 210, and 330 min after feeding, but lower values at 45, 150, and 270 min. Spears and Harvey (1984) found lasalocid decreased serum Mg concentration in grazing steers. Grings and Males (1988) reported that feeding monensin with MgO supplements to pregnant cows, fed wheat straw diets, depressed serum Mg concentration, but monensin fed with $MgSO_4$, increased serum Mg. Chirase et al. (1988) found 200 mg lasalocid•hd^{-1}•d^{-1}, fed to grazing beef cows, reduced the rate of decline of serum Mg, prior to parturition. Intravenous monensin administration reduced serum Mg in heifers and steers, but i.v. lasalocid did not alter it (Armstrong and Spears, 1988). Most research has indicated an increase in apparent absorption and(or) retention of Mg by ruminants receiving ionophores in high-concentrate (Starnes et al., 1984;

TABLE 62. SERUM MAGNESIUM CONCENTRATION AND RENAL HANDLING OF MAGNESIUM IN SHEEP FED A BASAL DIET WITH OR WITHOUT IONOPHORES

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Serum Mg, mg/100 ml				
Day 1	2.17	2.10	2.12	.02
Day 5[a]	2.12	2.08	2.08	.01
Day 1-9	2.13	2.12	2.10	.03
Serum Mg clearance, ml/min				
Day 1	.844	.873	.730	.036
Day 5	.810	.978	.807	.039
Urinary Mg excretion, mg/min				
Day 1	.184	.184	.149	.007
Day 5	.176	.201	.169	.008
Mg filtered load, mg/min				
Day 1	1.295	1.441	1.240	.064
Day 5	1.203	1.140	1.125	.048
Mg net tubular reabsorption, mg/min				
Day 1	1.111	1.253	1.084	.063
Day 5	1.030	.937	.956	.045

[a]Time x treatment interaction (P<.10).

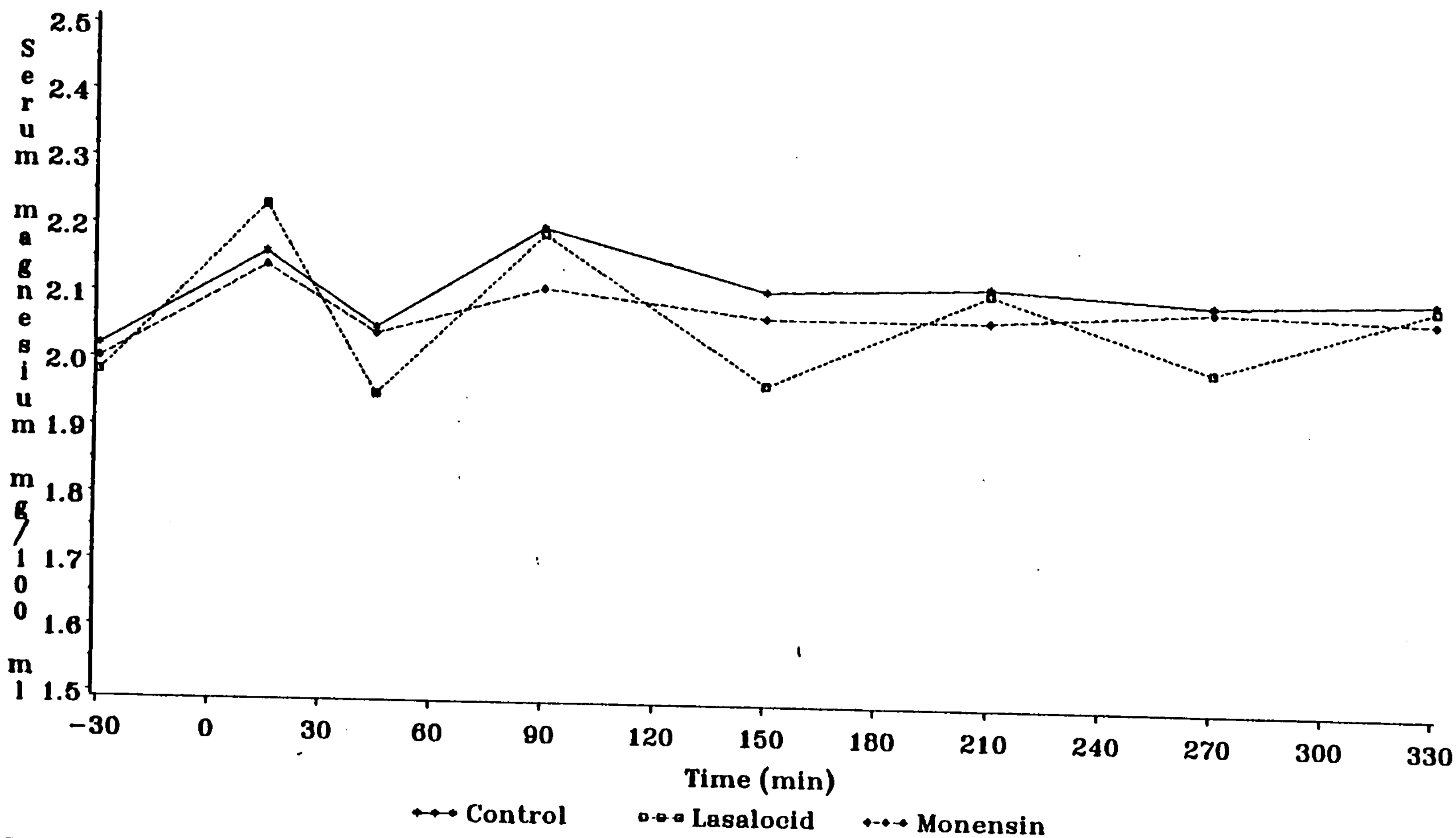

Figure 6. Serum magnesium concentration in sheep (day 5). Time x treatment interaction (P<.10). SE = .01.

Kirk et al., 1985b; Greene et al., 1986), and high-forage (Spears et al., 1987) diets. In the present study, serum clearance and urinary excretion rates of Mg were not different between treatment groups, during d 1 or d 5 (Table 62). Filtered load and net tubular reabsorption of Mg were not altered by feeding ionophores. Sheep fed lasalocid had greater ($P<.10$) Mg intake than those fed monensin on d 1 and 5 (Appendix Table 16).

Serum Ca concentration was not altered following a meal containing lasalocid or monensin on d 1, but tended to be lower in sheep fed ionophores on d 5 (Table 63). Serum Ca was reduced ($P<.10$) when ionophores were fed during d 1 through 9, but the differences were small. Elsasser (1984) reported that 10 to 30 ppm monensin decreased serum Ca in sheep fed high-concentrate diets. Chirase et al., (1988) found lasalocid supplemented to grazing beef cows increased serum Ca during mid-lactation. Monensin has been demonstrated to increase the apparent absorption and retention of Ca in sheep (Greene et al., 1986) and cattle (Spears et al., 1987).

During d 1, serum clearance rate of Ca was reduced ($P<.05$) for sheep fed ionophores, compared to those fed no ionophore. Serum clearance of Ca was not altered by dietary treatment during d 5. Urinary Ca excretion rate was lower ($P<.05$) in sheep fed ionophores during d 1, but was not altered by feeding ionophores at d 5. There were no differences in filtered load or net tubular reabsorption of Ca during d 1 or d 5. Kirk et al. (1985b) reported decreased urinary Ca excretion by sheep fed monensin. In the present study, sheep fed ionophores had greater ($P<.10$) Ca intake than those fed no ionophore (Appendix Table 16).

TABLE 63. SERUM CALCIUM CONCENTRATION AND RENAL HANDLING OF CALCIUM IN SHEEP FED A BASAL DIET WITH OR WITHOUT IONOPHORES

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Serum Ca, mg/100 ml				
Day 1	10.36	10.21	10.70	.22
Day 5	11.05	10.87	10.61	.07
Day 1-9[a]	10.81	10.65	10.44	.12
Serum Ca clearance, ml/min				
Day 1[b]	1.264	.504	.852	.104
Day 5	1.177	1.052	1.106	.096
Urinary Ca excretion, mg/min				
Day 1[b]	.123	.053	.080	.010
Day 5	.130	.115	.118	.010
Ca filtered load, mg/min				
Day 1	6.107	7.039	6.325	.395
Day 5	6.177	5.945	5.774	
Ca net tubular reabsorption, mg/min				
Day 1	5.904	7.071	6.236	.401
Day 5	6.047	5.828	5.636	.256

[a]Control differs from ionophores ($P<.10$).
[b]Control differs from ionophores ($P<.05$).

On d 1, serum inorganic P concentration was greater (P<.10) in sheep fed lasalocid or monensin (Table 64). During d 5, however, feeding ionophores did not alter serum inorganic P. Serum inorganic P in samples obtained from d 1 to 9 was greater (P<.05) in sheep fed ionophores. Elsasser (1984) reported increased serum inorganic P concentration in sheep fed 10 or 30 ppm monensin. Edlin et al. (1984) also found lasalocid and monensin increased serum inorganic P in finishing cattle. Serum inorganic P was reduced after monensin was administered i.v. to cattle, but lasalocid had no effect (Armstrong and Spears, 1988). Starnes et al. (1984) reported that lasalocid and monensin increased apparent absorption and retention of P in steers. Kirk et al. (1985b) found feeding monensin increased apparent absorption and retention of P in sheep. In the present study, feeding ionophores increased (P<.05) the filtered load of P during d 1, but had no effect during d 5. No P was detected in the urine.

Serum Cu concentration was not different between treatment groups on d 1 or d 5, or during d 1 through 9 (Table 65). Starnes et al. (1984) reported increased serum Cu in steers fed lasalocid or monensin. Dietary lasalocid and monensin were shown to increase liver Cu in sheep (Elsasser, 1984; Sappington et al., 1987).

Serum clearance of Cu was increased in sheep fed lasalocid, compared to those fed no ionophore (P<.01) or monensin (P<.05), during d 1. Sheep fed monensin had lower (P<.05) Cu clearance rates, compared to controls. Urinary excretion rate of Cu was reduced (P<.01) in sheep fed monensin compared to those fed no ionophore or lasalocid, during d 1. There was a time x treatment interaction (P<.01) for urinary excretion of Cu during d 1 (Fig. 7). Sheep fed monensin had greater urinary excretion rates of

TABLE 64. SERUM PHOSPHORUS CONCENTRATION IN SHEEP FED WITH OR WITHOUT IONOPHORES

Item	Ionophore None	Ionophore Lasalocid	Ionophore Monensin	SE
Serum inorganic P, mg/100 ml				
Day 1[a]	5.77	6.58	6.72	.12
Day 5	5.88	5.91	5.90	.12
Day 1-9[b]	5.90	6.37	6.38	.18
Inorganic P filtered load, mg/min				
Day 1	3.40	4.47	4.00	.22
Day 5	3.34	3.29	3.12	.13

[a]Control differs from ionophores ($P<.10$).
[b]Control differs from ionophores ($P<.05$).

TABLE 65. SERUM COPPER CONCENTRATION AND RENAL HANDLING OF COPPER IN SHEEP FED WITH OR WITHOUT IONOPHORES

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Serum Cu, ug/100 ml				
Day 1	45.5	39.0	48.4	1.2
Day 5	40.2	39.0	39.7	.7
Day 1-9	73.8	70.1	76.7	2.6
Serum Cu clearance, ml/min				
Day 1[abc]	.135	.184	.104	.011
Day 5	.140	.119	.093	.006
Urinary Cu, ug/min				
Day 1[def]	.059	.061	.046	.002
Day 5	.053	.046	.035	.002
Cu filtered load, ug/min				
Day 1[g]	27.2	27.6	33.8	1.1
Day 5	23.6	21.8	21.9	1.2
Cu net tubular reabsorption, ug/min				
Day 1[g]	27.1	27.5	33.7	1.1
Day 5	23.5	21.8	21.9	1.2

[a]Control differs from lasalocid ($P<.01$).
[b]Lasalocid differs from monensin ($P<.05$).
[c]Control differs from monensin ($P<.05$).
[d]Control differs from monensin ($P<.01$).
[e]Lasalocid differs from monensin ($P<.01$).
[f]Time x treatment interaction ($P<.01$).
[g]Time x treatment interaction ($P<.10$).

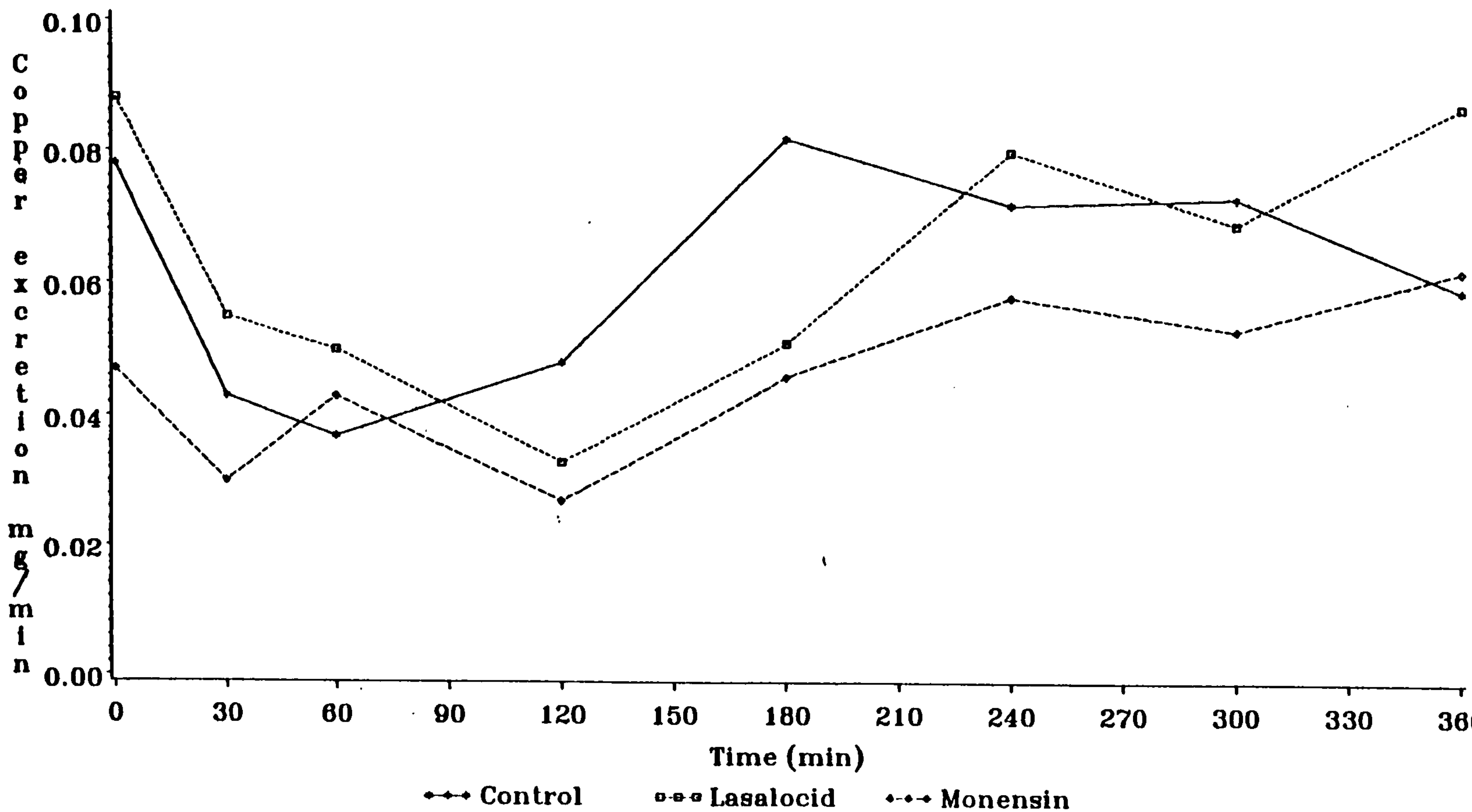

Figure 7. Urinary excretion rate of copper in sheep (day 1). Time x treatment interaction (P<.01). Control differs from monensin (P<.01). Lasalocid differs from monensin (P<.01). SE=.002.

Cu early in the collection, but had lower values beginning 120 min after feeding, compared to sheep fed no ionophore or lasalocid. During d 5, there were no changes in serum clearance or urinary excretion rates of Cu due to treatment. There were no overall differences between treatments for filtered load or net tubular reabsorption of Cu during d 1 or d 5. Time x treatment interactions (P<.10) were observed for filtered load (Fig. 8) and net tubular reabsorption (Fig. 9) of Cu, during d 1. Sheep fed lasalocid had greater filtered load and net tubular reabsorption of Cu at 60 min after feeding, but lower values at 300 min, compared to sheep fed no ionophore or monensin. Sheep fed lasalocid had greater (P<.10) Cu intake than those fed monensin on d 1 and 5 (Appendix Table 16).

There were no differences between treatment groups in serum Fe concentration on d 1 or d 5, or during d 1 through 9 (Table 66). Elsasser (1984) reported increased ^{59}Fe uptake by chicken duodenal tissue with lasalocid addition and decreased ^{59}Fe uptake with monensin addition, indicating increased absorption of Fe with lasalocid and decreased absorption with monensin.

On d 1, serum clearance rate of Fe was lower (P<.05) after monensin was fed, compared to feeding lasalocid, and tended to be lower than when no ionophores were fed. During d 5, serum clearance of Fe tended to be lower when ionophores were fed. There was a time x treatment interaction (P<.05) for Fe clearance during d 5 (Fig. 10). Differences seemed to be greatest between 120 min to 240 min after feeding the ionophores. During d 1, urinary Fe excretion was reduced (P<.05) when monensin was fed, compared to feeding lasalocid, and tended to be lower compared to controls. During d 5, both lasalocid and monensin tended to reduce

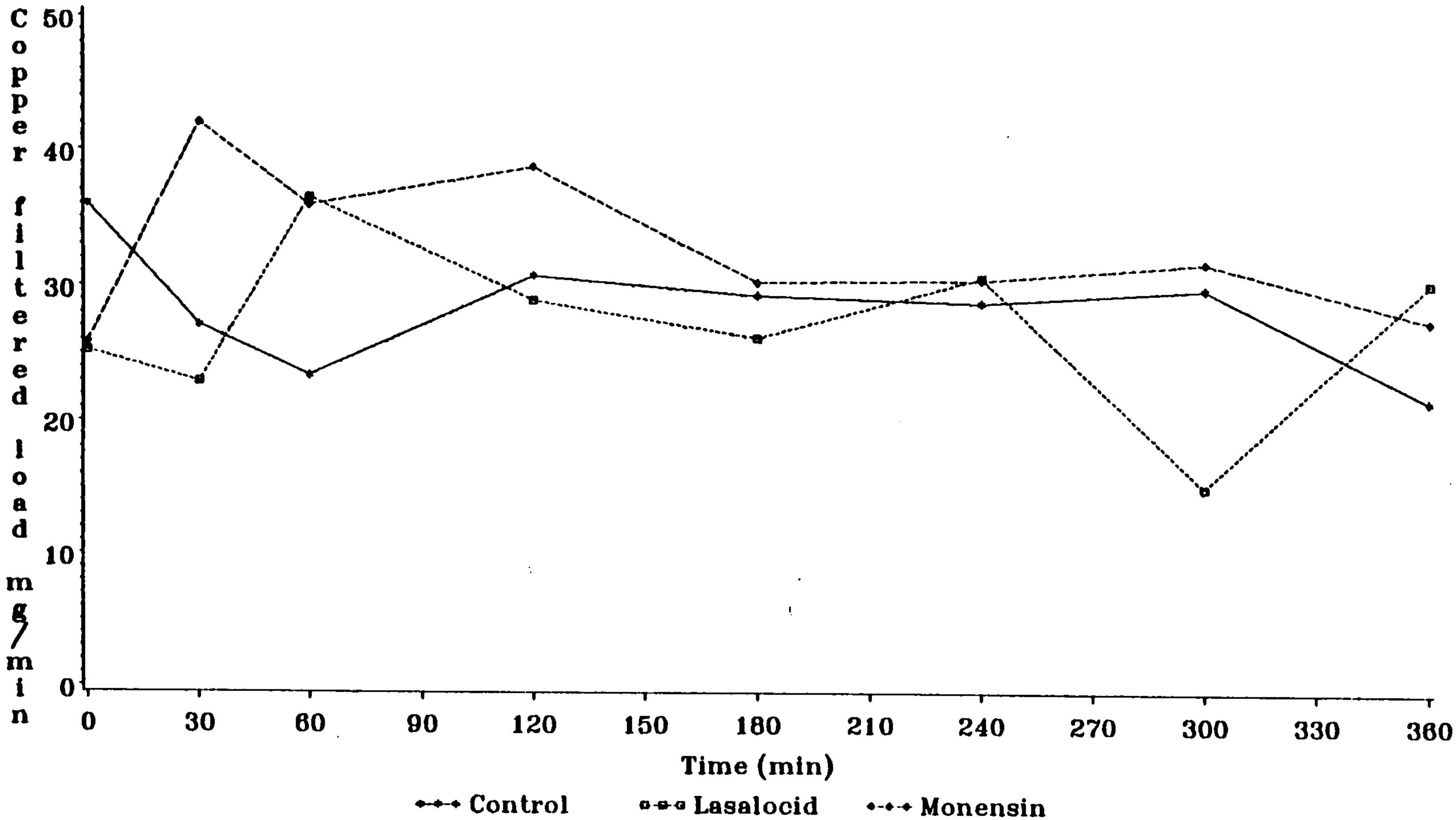

Figure 8. Filtered load of copper in sheep (day 1). Time x treatment interaction (P<.01). SE=1.1.

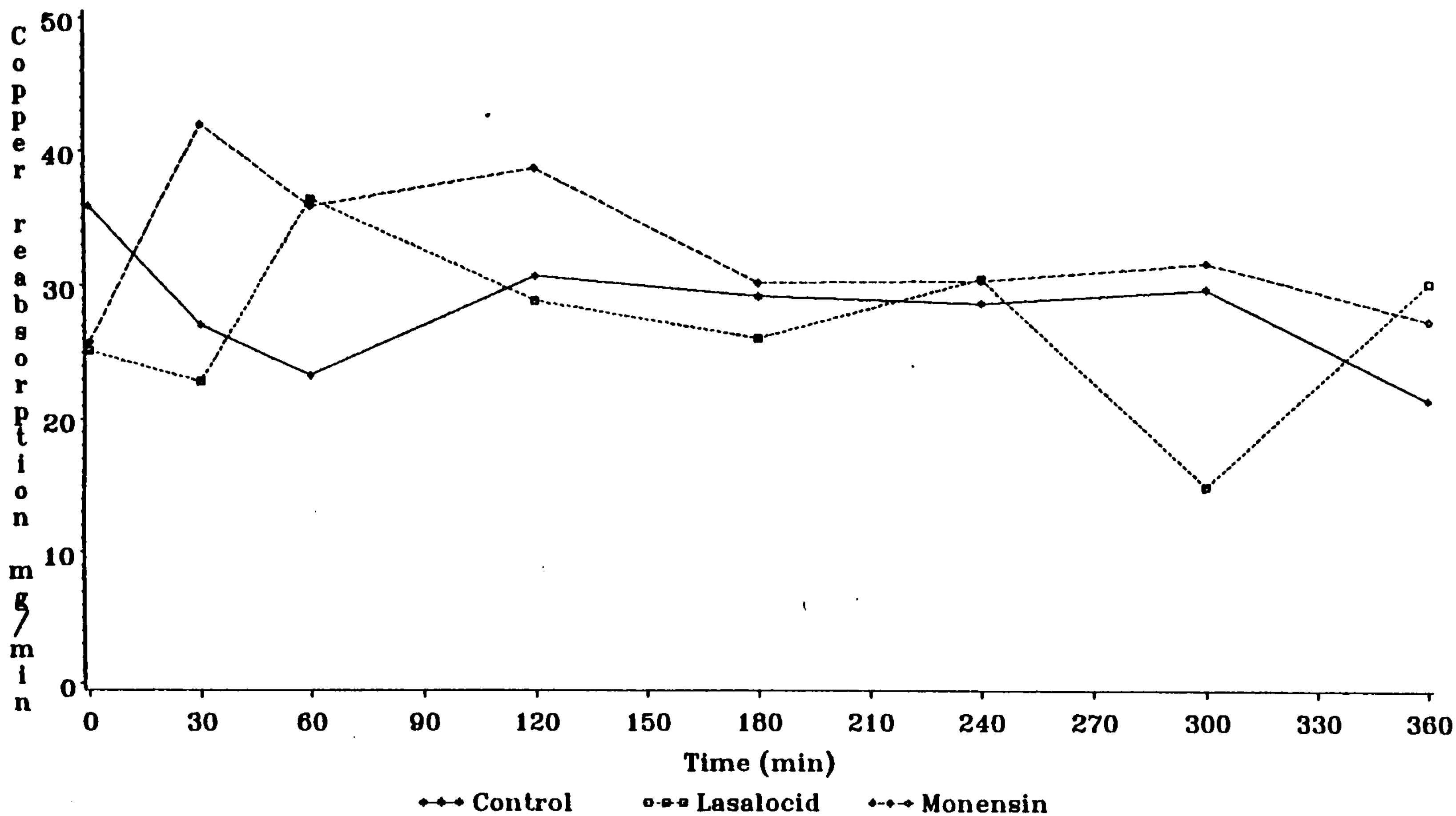

Figure 9. Net tubular reabsorption of copper in sheep (day 1). Time x treatment interaction (P<.10). SE=1.1.

TABLE 66. SERUM IRON CONCENTRATION AND RENAL HANDLING OF IRON IN SHEEP FED WITH OR WITHOUT IONOPHORES

Item	Ionophore			SE
	None	Lasalocid	Monensin	
Serum Fe, ug/100 ml				
Day 1	62.8	63.0	72.9	3.8
Day 5	75.9	72.6	64.4	3.2
Day 1-9	122.8	141.2	123.2	9.8
Serum Fe clearance, ml/min				
Day 1[a]	.556	.856	.374	.078
Day 5[b]	.718	.473	.369	.024
Urinary Fe, ug/min				
Day 1[a]	.234	.260	.163	.010
Day 5	.266	.185	.136	.008
Fe filtered load, ug/min				
Day 1	38.3	47.1	50.9	3.0
Day 5	43.2	41.9	34.2	2.3
Fe net tubular reabsorption, ug/min				
Day 1	38.0	46.8	50.7	3.0
Day 5	42.9	41.8	34.0	2.3

[a]Lasalocid differs from monensin ($P<.05$).
[b]Time x treatment interaction ($P<.05$).

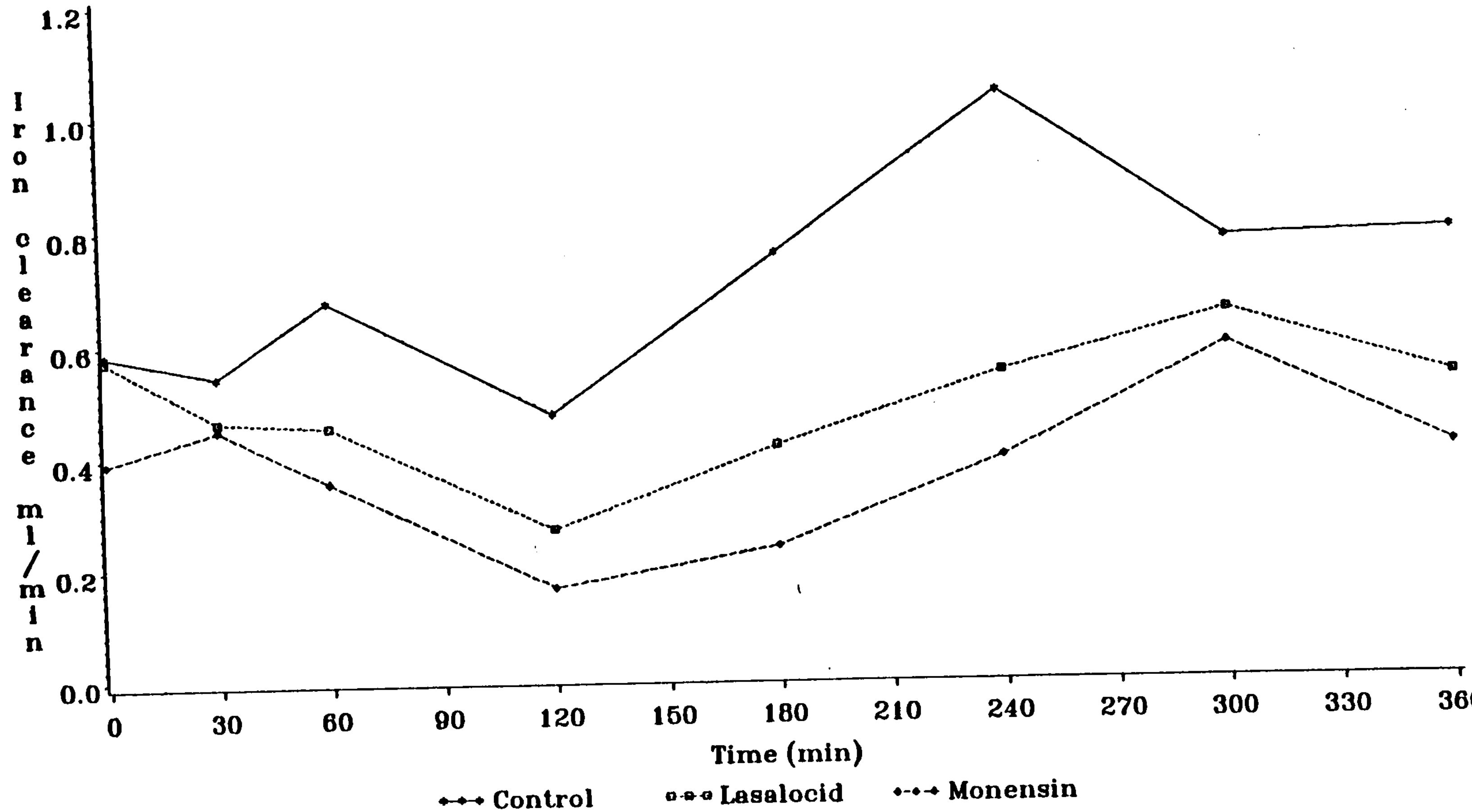

Figure 10. Serum clearance rate of iron in sheep (day 5). Time x treatment interaction (P<.05). SE=.024.

urinary Fe excretion, compared to controls. There were no differences between treatment groups for filtered load or net tubular reabsorption of Fe in sheep. Sheep fed lasalocid had greater (P<.10) Fe intake than those fed monensin on d 5 (Appendix Table 16).

Serum Zn concentration was not significantly altered by feeding ionophores on d 1 or d 5, or during d 1 through 9 (Table 67). Kirk et al. (1985b) reported large increases in apparent absorption and retention of Zn in sheep fed 20 ppm monensin. Greene et al. (1986) also reported increased apparent absorption of Zn in steers fed monensin. Costa et al. (1985) found that feeding monensin increased Zn retention in steers. In the present study, serum clearance of Zn tended to be higher when lasalocid was fed, compared to feeding no ionophore or monensin, during d 1 and d 5. There was a time x treatment interaction (P<.10) for Zn clearance during d 5 (Fig. 11). The differences appeared to be greater toward the end of the sampling period. Urinary excretion of Zn tended to be greater when lasalocid was fed, during d 1. There was a time x treatment interaction (P<.05) for urinary Zn excretion rate during d 1 (Fig. 12). The values were much greater for the lasalocid-fed sheep through 120 min, and decreased at 180 min. During d 5, feeding lasalocid increased (P<.05) urinary Zn excretion compared to feeding no ionophore or monensin. Feeding monensin reduced (P<.01) urinary Zn excretion vs. controls. Filtered load and net tubular reabsorption of Zn were not different between treatment groups.

Although sheep consumed all feed presented to them during the first feeding (containing ionophores), animals refused some feed during the

TABLE 67. SERUM ZINC CONCENTRATION AND RENAL HANDLING OF ZINC IN SHEEP FED WITH OR WITHOUT IONOPHORES

Item	Ionophore			
	None	Lasalocid	Monensin	SE
Serum Zn, ug/100 ml				
Day 1	59.7	57.9	59.5	1.7
Day 5	63.6	62.5	59.6	1.2
Day 1-9	61.1	58.8	58.6	2.5
Serum Zn clearance, ml/min				
Day 1	.298	.378	.308	.040
Day 5[a]	.140	.160	.131	.007
Urinary Zn, ug/min				
Day 1[a]	.179	.210	.167	.015
Day 5[bcd]	.088	.102	.071	.004
Zn filtered load, ug/min				
Day 1	35.7	41.0	35.5	1.6
Day 5	35.9	35.2	31.7	1.4
Zn net tubular reabsorption, mg/min				
Day 1	35.5	40.8	35.4	1.7
Day 5	35.8	35.1	31.8	1.4

[a]Time x treatment interaction (P<.10).
[b]Control differs from lasalocid (P<.05).
[c]Lasalocid differs from monensin (P<.05).
[d]Control differs from monensin (P<.01).

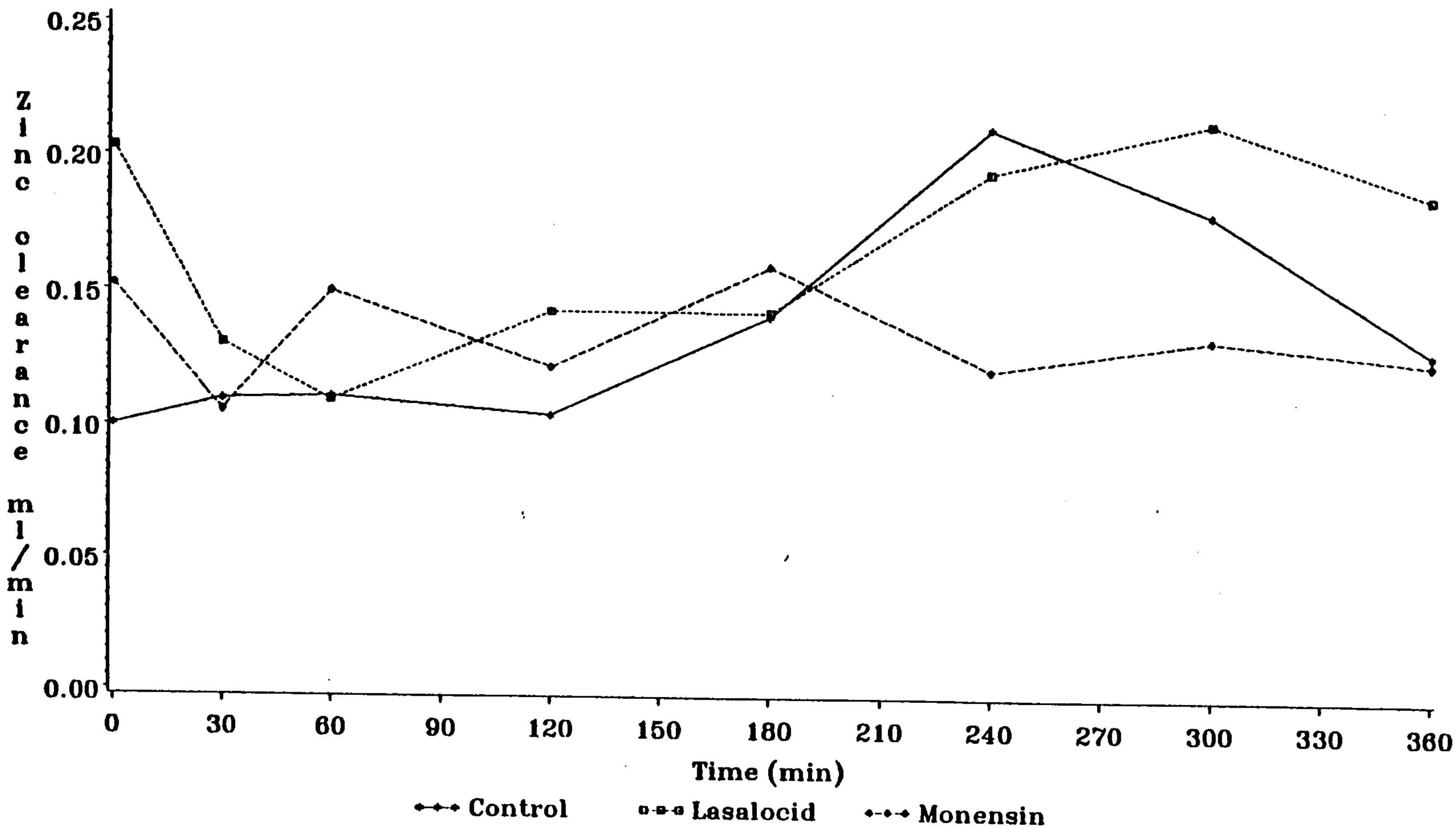

Figure 11. Serum clearance rate of zinc in sheep (day 5). Time x treatment interaction ($P<.10$). SE=.007.

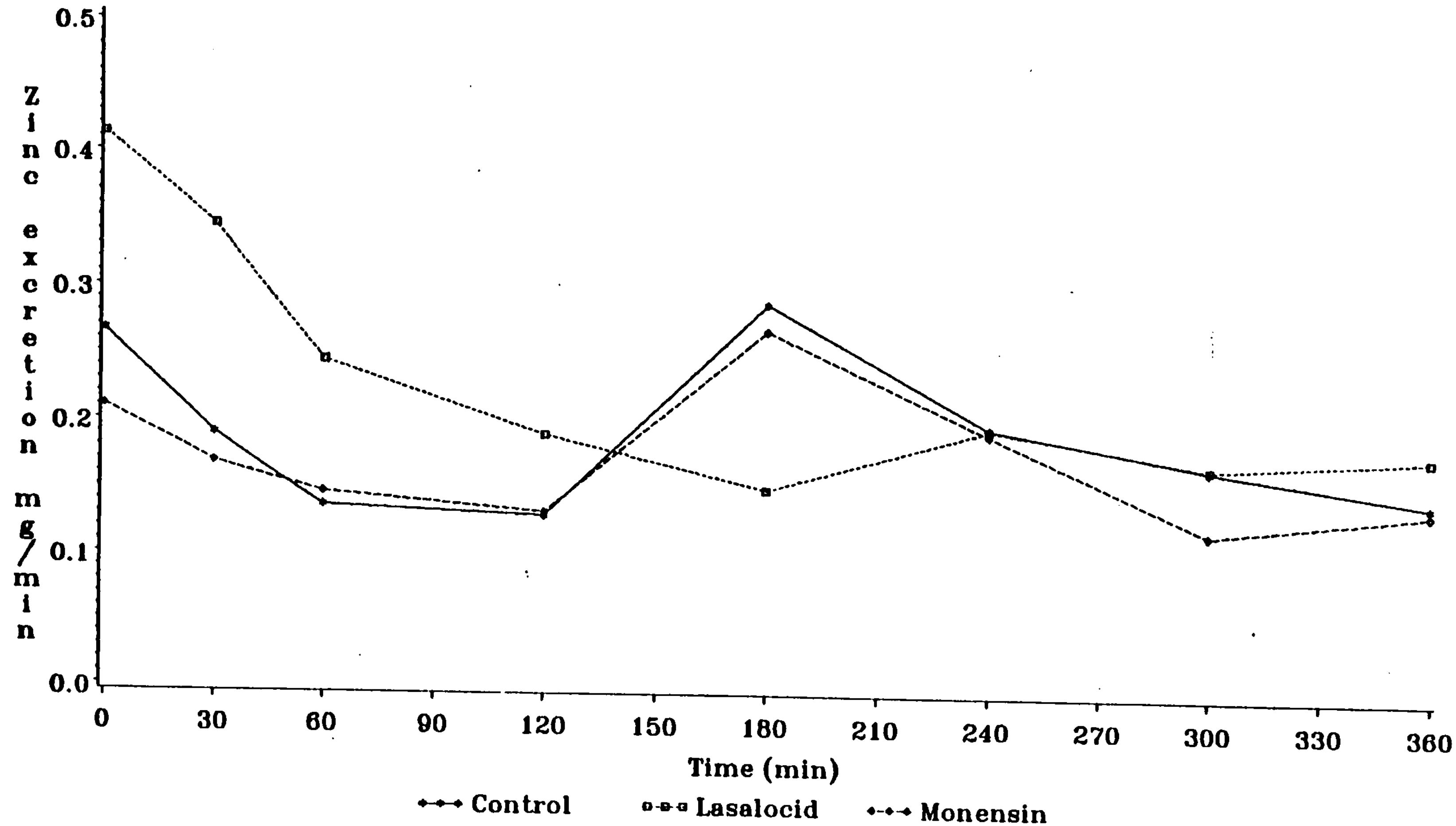

Figure 12. Urinary excretion rate of zinc in sheep (day 1). Time x treatment interaction (P<.10). SE=.015.

second feeding (containing no ionophores) during both d 1 and d 5. This resulted in differences in mineral intake between treatment groups. Dry matter intake also differed between treatments (Appendix Table 16). Therefore, differences due to treatments for serum mineral and aldosterone concentration, renal function, and renal handling of minerals may have been due, at least partly, to changes in intake.

Serum clearance and urinary excretion rates of minerals were highly correlated with urinary mineral concentration, but were poorly correlated with urine flow or serum mineral concentration (Appendix Table 17). This would suggest that changes in clearance and excretion of minerals due to feeding ionophores are the result of altered tubular reabsorption of minerals. Net tubular reabsorption of minerals was not associated with urinary excretion rates of the minerals, but was highly correlated with the filtered load of mineral and GFR (Appendix Table 18). Since the amount of mineral excreted in the urine is relatively small in relation to the filtered load, statistically significant differences in net tubular reabsorption may be masked.

The different effects of ionophores upon serum mineral concentration and renal handling of minerals in sheep when an ionophore is first fed, and after several days feeding, may indicate physiological adaptations to ionophores. Results from this study indicate that dietary ionophores alter the renal handing of minerals in sheep, and these changes may be responsible for some of the differences in mineral metabolism seen when ruminants are fed ionophores.

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GENERAL DISCUSSION

Sodium. Both lasalocid and monensin transport Na^+ across cell membranes (Pressman, 1976). During the two grazing trials, there were no consistent changes in serum Na concentration in steers fed supplemental lasalocid or monensin. However, during the finishing study, feeding monensin to steers increased serum Na when fed with low dietary K, and decreased it when fed with high K. Changes in serum Na were numerically small. Although previous studies have shown no change in serum Na when ionophores were fed, *in vivo* (Starnes et al., 1984) and *in vitro* (O'Conner and Beede), research suggests that ionophores increase digestive tract absorption of Na. In the present study, with multi-cannulated sheep no changes were seen in apparent absorption, digestive tract flow, or partial absorption of Na when lasalocid or monensin was fed.

Urine serves as the primary route of Na excretion in ruminants (Devlin and Roberts, 1965; Perry et al., 1966), with urinary Na excretion increasing following elevated Na intake (Tomas et al., 1973). Therefore, Na status may be maintained through the action of the kidney. During the two grazing trials, urinary excretion of Na by steers was greater when ionophores were supplemented. Multi-cannulated sheep displayed lower urinary Na excretion when fed lasalocid, and greater urinary Na excretion when fed monensin. During the renal clearance study, feeding monensin to sheep decreased serum clearance and urinary excretion rates of Na at d 1 and d 5. Feeding lasalocid had no effect upon serum clearance and urinary excretion rates of Na in sheep. Previously, Kirk et al. (1985a) reported increased urinary excretion of Na and decreased Na retention in sheep fed monensin. Changes in urinary Na excretion and renal handling of Na when ionophores are fed may prevent changes in serum Na concentration in these animals.

Potassium. Lasalocid and monensin have also been shown to bind and transport K^+ (Pressman, 1976). There were no consistent changes in serum K concentration when ionophores were fed to grazing or finishing steers in the present study. However, during the second grazing trial, steers fed lasalocid had less K accumulation in the liver than controls, and those fed monensin had K loss from the liver. Since K is primarily an intracellular ion, decreased liver K may indicate depressed K status. Feeding lasalocid or monensin lowered serum K concentration in sheep during d 1 through 9 of the renal clearance study. Previously, dietary ionophores have been demonstrated to lower serum K concentration in grazing (Spears and Harvey, 1984) and finishing cattle (Edlin et al., 1984). However, apparent absorption of K has been shown to increase in sheep fed monensin (Kirk et al., 1985a; Greene et al., 1986). In the present study, feeding lasalocid or monensin to multi-cannulated sheep increased apparent absorption of K, although there were no changes in digestive tract flow or partial absorption of K.

Excretion of K occurs primarily through the urine in ruminants (Elam and Autry, 1961; Devlin and Roberts, 1963), with increased urinary K excretion during elevated K intake (Newton et al., 1972). In the present study, urinary excretion of K was not altered in grazing steers fed supplemental lasalocid or monensin. Sheep fed monensin during the renal clearance study had lower serum clearance and urinary excretion rates of K during d 5. Feeding lasalocid did not significantly alter serum clearance or urinary excretion rates of K in sheep.

Aldosterone. The effects of ionophores upon Na and K status of ruminants may be mediated by the actions of aldosterone. Dietary lasalocid and monensin tended to increase serum aldosterone concentration in sheep during the renal clearance study, at d 1 and d 5, and during d 1 through 5. During the finishing study, steers fed monensin also displayed a trend toward increased serum aldosterone. Serum aldosterone increases in ruminants in response to decreased serum Na (Blair-West et al., 1963). Aldosterone has been shown to decrease urinary Na excretion in ruminants (Kumar and Singh, 1981; Riad et al., 1986), but not to alter serum Na or K concentration (Safwate et al., 1982; Riad et al., 1986). Changes in serum Na and renal handling of Na in sheep, following increased serum aldosterone in ionophore-fed sheep in the present study, would agree with those reports.

Magnesium. Previous research indicated an increase in apparent absorption and(or) retention of Mg in ruminants receiving ionophores in high-concentrate (Starnes et al., 1984; Kirk et al., 1985b; Greene et al., 1986) and high-forage (Spears et al., 1987) diets. It has been suggested that feeding ionophores may be useful in preventing grass tetany (hypomagnesemia) (Greene et al., 1986). Chirase et al. (1988) found lasalocid supplementation reduced the rate of decline of serum Mg in grazing beef cows, prior to parturition. However, Spears and Harvey (1984) found lasalocid decreased serum Mg in grazing steers. Grings and Males (1988) reported that feeding monensin with MgO in supplements for pregnant cows fed a wheat-straw based diet, also depressed serum Mg. In the present study, grazing steers fed monensin had lower serum Mg during trial 1, and steers fed either lasalocid or monensin had lower serum Mg during trial 2. However, feeding lasalocid or monensin to multi-cannulated sheep increased apparent absorption of Mg, with a tendency for greater absorption of Mg from the preintestinal region, and less Mg secretion from the small intestine when lasalocid or monensin was fed. Greene et al. (1988) reported increased preintestinal absorption of Mg when monensin was fed to multicannulated steers. Small intestinal secretion of Mg increased when monensin was fed.

Results from the present finishing study indicate that the effects of ionophores upon Mg absorption may be dependent upon dietary K concentration. Steers had increased serum Mg when monensin was fed with low dietary K, but decreased serum Mg when monensin was fed with high K. Previously, improvements in Mg status of ruminants fed ionophores have been shown primarily in animals fed high-concentrate diets. These diets are typically low in K, while forages are normally high in K. Elevated K concentration in forages may explain why ionophores depressed serum Mg concentrations in grazing ruminants fed ionophores. Since hypomagnesemia is often associated with intake of high-K diets (Fontenot et al., 1969; Newton et al., 1972), ionophores may exacerbate hypomagnesemia if supplemented to cattle under these conditions.

The K-induced reduction in ruminal Na:K ratio, seen during the finishing study, may play an important role in altering Mg absorption and therefore serum Mg concentration. Martens et al. (1987) found intraruminal Na infusion in sheep increased Mg absorption, compared with no Na infusion. Na infusion brought about an increase in salivary and ruminal K levels, with a resultant decrease in ruminal Na:K ratio. Martens and Rayssiguier (1980) reported that high dietary K was ineffective in depressing Mg absorption in sheep, unless accompanied by low dietary Na. However, Poe et al. (1985) found a greater decrease in Mg availability when high K and high Na were fed to sheep, than when high K and low Na were fed. Martens and Blume (1986) found that ruminal Na concentration did not influence Mg absorption in the washed sheep rumen, but an increase in potential difference following ruminal K infusion, played a major role in lowering Mg availability. Martens et al. (1987) reported a relationship between increased potential difference and decreased Mg absorption. In the present study, there was a significant correlation for ruminal Na:K ratio and serum Mg in finishing steers ($r=.4808$; $P<.001$).

Martens et al. (1978) reported Mg absorption in the sheep stomach takes place through the action of a ouabain-inhibited active transport system, indicating the involvement of the Na^{+}-K^{+} pump. Monensin has been demonstrated to increase Na^{+}-K^{+} pump activity in cell membranes (Smith and Rozengurt, 1978; Mendoza et al., 1980). It has been suggested that monensin may increase Mg absorption by increasing activity of the Na^{+}-K^{+} pump in the rumen (Greene et al., 1988). Monensin preferentially binds Na^{+} verses K^{+}, 10 to 1 (Pressman, 1976). However, Russell (1987) has demonstrated, using isolated Streptococcus bovis cultures, that monensin may carry K^{+} rather than Na^{+} when the K^{+}-gradient greatly exceeds the Na^{+} gradient. In the present study, monensin increased serum Mg when fed with .4% K, but decreased serum Mg when fed with 2.3% K. The K-induced decrease in ruminal Na:K ratio may have altered the Na^{+}- and K^{+}-gradients enough to allow monensin to preferentially carry K^{+}, rather than Na^{+}, when the ionophore was fed with high K. This would result in decreased Na^{+}-K^{+}

pump activity and a secondary reduction in Mg absorption, thereby reducing serum Mg when monensin was fed with high K. Such action could explain why feeding supplemental ionophores depressed serum Mg in cattle grazing higher K-containing pasture, as reported in the present study and by Spears and Harvey (1984).

Calcium. In the present study, serum Ca concentration was greater in grazing and finishing steers when ionophores were fed. Chirase et al. (1988) reported increased serum Ca in grazing cows fed supplemental lasalocid, but Elsasser (1984) found dietary monensin decreased serum Ca in sheep. Dietary ionophores have been shown to increase the apparent absorption and retention of Ca in cattle (Spears et al., 1987) and sheep (Greene et al., 1986). In the present study, no change in apparent absorption or retention of Ca were obtained when multi-cannulated sheep were fed ionophores, but Ca absorption and retention were greater for lasalocid-fed vs monensin-fed sheep. There were no changes in digestive tract flow or partial absorption of Ca due to feeding ionophores. Greene et al. (1988) found no change in partial absorption of Ca in multi-cannulated steers fed monensin.

Absorbed Ca is primarily excreted through the feces in ruminants (Hansard et al., 1952). However, renal tubular reabsorption of Ca, mediated by the action of parathyroid hormone, is involved in maintaining Ca homeostasis (Agus, 1977). In the present study, urinary Ca excretion was greater when grazing steers were fed supplemental ionophores during the first, but not in the second trial. During the renal clearance study, sheep fed ionophores showed no change in serum Ca at d 1, but serum clearance and urinary excretion rate of Ca were depressed. At d 5, serum

Ca concentration tended to be lower in sheep fed ionophores, but renal handling of Ca was not changed. Ramburg et al. (1976) has suggested that a decrease in Ca clearance seen in older cows brings about reduced serum Ca concentration. Similar effects may have occurred in the present study. Therefore, the effects of ionophores upon serum Ca may be secondary to altered renal handling of Ca.

Phosphorus. Serum inorganic P concentration was greater in grazing steers receiving supplemental lasalocid or monensin during trial 2, but not trial 1. During the renal clearance study, sheep fed ionophores had greater serum inorganic P at d 1, but not d 5. Finishing steers fed monensin with low K had increased serum inorganic P, but those fed monensin with high K had decreased serum inorganic P. Previous research has shown increased serum inorganic P when ionophores were fed to cattle (Edlin et al., 1984) and sheep (Elsasser, 1984). Dietary ionophores have been demonstrated to increase apparent absorption and retention of P in cattle (Starnes et al., 1984) and sheep (Kirk et al., 1985b). In the present study, multi-cannulated sheep fed lasalocid or monensin tended to have greater absorption and retention of P, but with no change in digestive tract flow or partial absorption of P. Improved absorption of P may be beneficial in the prevention of P deficiency and the resultant hypophosphatemia, anorexia, pica, decreased fertility and milk production, poor appearance and listlessness (Thompson, 1978).

Copper. Liver Cu concentration is often used to determine Cu status in ruminants. There were no significant effects of feeding ionophores upon the change in liver Cu during the two grazing trials. Elsasser (1984) and Sappington et al. (1987) reported increased liver Cu concentration in sheep fed lasalocid or monensin, suggesting improved Cu absorption. However, some research has indicated that liver Cu concentration is poorly related to Cu deficiency in ruminants (Mylrea, 1958; Smith and Coup, 1973; Kellaway, 1978).

Serum Cu concentration was lower in steers fed monensin than those fed lasalocid during the second grazing trial. Finishing steers fed monensin with low K had increased serum Cu, but those fed monensin with high K had no change in serum Cu concentration. Serum Cu concentration was not altered after ionophores were fed to sheep during the renal clearance study. Starnes et al. (1984) reported increased serum Cu in steers fed lasalocid or monensin.

In the present study, there was no change in apparent absorption or retention of Cu in multi-cannulated sheep fed ionophores. Absorption of Cu from the small intestine was greater for sheep fed lasalocid than those fed monensin, while large intestinal Cu absorption was less for lasalocid than monensin. Increased absorption of Cu in ruminants fed ionophores, suggested by previous research, should be considered when sheep are fed, since these animals are particularly susceptible to Cu toxicosis. Feeding ionophores, particularly lasalocid (which is often fed a a coccidiostat to sheep), with high copper-containing feedstuffs (such as broiler litter) may be detrimental.

Urinary Cu accounts for only 1 to 2% of total Cu excreted in sheep (Smith et al., 1968). In the present study, changes in renal handling of Cu were seen when ionophores were fed to sheep. At d 1 of the renal clearance study, lasalocid increased serum clearance rate of Cu, and monensin decreased it, compared to controls. Urinary Cu excretion rate was lower when monensin was fed, compared to feeding no ionophore or lasalocid, at d 1. During the second grazing trial, urinary Cu excretion by steers was reduced when ionophores were fed. These changes in urinary Cu excretion would have little impact upon Cu homeostasis, but may be a reflection of changes in Cu status of ruminants fed ionophores.

Iron. Serum Fe concentration was lower in steers fed monensin than those fed lasalocid during the second grazing trial. Finishing steers fed monensin had lower serum Fe concentration. Serum Fe concentration was not altered in grazing steers or in sheep used in the renal clearance study, when lasalocid or monensin was fed. In the present study, feeding ionophores to multi-cannulated sheep increased apparent absorption of Fe, although digestive tract flow and partial absorption of Fe were not affected. Elsasser (1984) found ^{59}Fe absorption in chicken duodenal loops increased when lasalocid was added but decreased when monensin was added.

Sheep fed lasalocid had greater serum clearance rate of Fe than those fed monensin during the first day of the renal clearance study, but Fe clearance tended to be lower when either lasalocid or monensin was fed at d 5. Urinary excretion rate of Fe was reduced in sheep fed monensin, compared to those fed lasalocid at d 1, and tended to be reduced by either lasalocid or monensin at d 5. Urinary Fe excretion in steers was decreased when ionophores were fed during the second grazing trial. Changes in urinary excretion would have little impact upon Fe homeostasis, since relatively little Fe is excreted through the urine (Underwood, 1977). However, changes in renal handling of Fe may reflect altered Fe status in ruminants fed ionophores.

Zinc. Serum Zn concentration was not altered by feeding ionophores to steers during grazing trials or sheep during the renal clearance study. Feeding monensin with low K increased serum Zn concentration in finishing steers, but feeding monensin with high K decreased serum Zn. Previous research has shown increased apparent absorption of Zn in sheep (Kirk et al., 1985b; Greene et al., 1986) and increased Zn retention in sheep (Kirk et al., 1985b) and cattle (Costa et al., 1985). In the present study, multi-cannulated sheep displayed no change in apparent absorption or retention of Zn when ionophores were fed. Digestive tract flow and partial absorption of Zn were not altered when ionophores were fed. Greene et al. (1988) reported no change in partial absorption of Zn in multi-cannulated steers fed monensin.

Renal handling of Zn was altered when ionophores were fed to sheep. During d 5 of the renal clearance study, sheep fed lasalocid had greater urinary excretion rates of Zn, while those fed monensin had lower urinary Zn excretion rates, compared to controls. During the first grazing trial, feeding ionophores increased urinary Zn excretion in steers, but during the second trial, feeding ionophores reduced urinary Zn excretion. Little absorbed Zn is excreted through the urine of ruminants (Hansard and Mohammed, 1968). Therefore, changes in urinary Zn excretion by ruminants fed ionophores would be expected to have little impact upon Zn homeostasis of ruminants, but may be an indication of altered Zn status.

Results from this study indicate that feeding lasalocid or monensin can alter mineral status of ruminants fed either high-forage or high-concentrate diets. However, the effects of these ionophores may differ. Changes in mineral metabolism when lasalocid or monensin is fed to ruminants appear to be due to altered extent of partial absorption within the digestive tract, and altered renal handling of minerals. The effects of ionophores upon mineral metabolism in ruminants have been demonstrated to be dependent upon levels in the diet.

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APPENDIX

APPENDIX TABLE 1. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM CALCIUM CONCENTRATION IN GRAZING STEERS.

Trial	Day	Ionophore			SE
		None	Lasalocid	Monensin	
		mg/100 ml			
1	0	9.00	8.33	8.68	.31
	14	9.61	9.50	9.20	.47
	28	9.60	10.26	10.04	.80
	42	9.35	9.39	9.38	.51
	56[a]	8.88	8.98	9.52	.64
	70	9.34	9.63	9.68	.40
	84	9.40	9.74	9.52	.63
2	0	11.34	11.19	11.71	.18
	14	11.48	11.68	11.38	.14
	28	10.73	10.66	10.89	.13
	42	10.85	10.78	10.54	.17
	56	12.92	13.53	13.81	1.33
	70	10.90	10.81	10.73	.19
	84	10.49	10.60	10.92	.17

[a]Lasalocid differs from monensin (P<.10).

APPENDIX TABLE 2. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY CALCIUM EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore None	Ionophore Lasalocid	Ionophore Monensin	SE
		------mg/100 mg creatinine-------			
1	0	4.67	1.66	7.52	3.17
	14	2.76	2.39	4.16	.75
	28[a]	2.00	3.37	1.26	.79
	42[b]	75	1.56	.65	.24
	56[b]	1.40	2.47	1.28	.37
	70[c]	.93	3.32	2.51	.59
	84	2.03	2.93	1.85	.58
2	0	2.50	2.25	2.99	.46
	14	3.46	2.74	1.77	1.07
	28[c]	2.72	1.00	4.18	.88
	42	3.82	2.82	3.92	.75
	56	4.34	3.07	1.82	2.13
	70[c]	3.58	3.70	1.96	.52
	84[b]	2.96	5.16	2.09	1.18

[a]Control differs from ionophores (P<.05).
[b]Lasalocid differs from monensin (P<.10).
[c]Lasalocid differs from monensin (P<.05).

APPENDIX TABLE 3. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM INORGANIC PHOSPHORUS CONCENTRATION IN GRAZING STEERS.

		Ionophore			
Trial	Day	None	Lasalocid	Monensin	SE
		mg/100 ml			
1	0	7.92	7.56	8.16	.32
	14	8.87	9.56	9.18	.40
	28	9.57	9.38	9.21	.48
	42[ab]	8.05	6.26	7.96	.28
	56	7.70	7.83	7.42	.47
	70	7.78	7.59	8.31	.49
	84	9.10	8.85	8.48	.47
2	0	7.09	7.32	7.28	.24
	14	7.57	7.45	7.28	.23
	28	7.43	7.24	7.05	.22
	42[a]	8.29	9.03	9.03	.23
	56	6.09	6.38	6.41	.30
	70[a]	7.19	7.68	7.69	.21
	84	6.55	6.74	6.93	.22

[a]Control differs from ionophores ($P<.05$).
[b]Lasalocid differs from monensin ($P<.001$).

APPENDIX TABLE 4. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM MAGNESIUM CONCENTRATION IN GRAZING STEERS.

Trial	Day	Ionophore None	Lasalocid	Monensin	SE
		mg/100 ml			
1	0	1.97	1.86	1.86	.57
	14	2.08	2.09	2.08	.19
	28	1.81	1.88	1.93	.24
	42	1.80	1.76	1.67	.19
	56[ab]	1.75	1.75	1.44	.19
	70[a]	1.80	1.82	1.60	.23
	84	1.61	1.72	1.59	.25
2	0	2.06	2.06	2.09	.06
	14[c]	2.07	2.02	2.17	.05
	28	2.10	2.06	2.12	.06
	42[d]	2.34	2.19	2.22	.05
	56	2.26	2.16	2.15	.06
	70	2.17	2.07	2.07	.05
	84	2.24	2.21	2.24	.04

[a]Lasalocid differs from monensin ($P<.01$).
[b]Control differs from monensin ($P<.05$).
[c]Lasalocid differs from monensin ($P<.10$).
[d]Control differs from ionophores ($P<.05$).

APPENDIX TABLE 5. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY MAGNESIUM EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore None	Lasalocid	Monensin	SE
		------mg/100 mg creatinine-------			
1	0	34.11	26.51	84.78	38.28
	14	26.47	31.31	43.36	10.40
	28	11.25	11.72	10.68	4.17
	42	4.73	3.96	4.39	1.62
	56	5.02	7.88	2.87	2.06
	70	1.84	2.93	3.35	1.12
	84	1.76	5.18	2.40	1.15
2	0	40.87	40.34	48.95	8.79
	14[a]	31.19	22.37	74.99	18.66
	28	18.34	23.57	43.71	8.80
	42[b]	17.86	10.34	37.46	4.80
	56[a]	30.57	17.42	36.18	6.27
	70	47.66	44.04	31.93	5.12
	84	46.7	29.16	43.59	5.88

[a]Lasalocid differs from monensin (P<.10).
[b]Lasalocid differs from monensin (P<.01).

APPENDIX TABLE 6. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM SODIUM CONCENTRATION IN GRAZING STEERS.

Trial	Day	Ionophore			SE
		None	Lasalocid	Monensin	
		mg/100 ml			
1	0	314.2	308.8	288.8	15.9
	14	226.5	228.5	229.2	5.4
	28	226.5	225.8	232.0	9.6
	42	220.9	222.0	222.5	5.0
	56	290.1	316.1	314.0	5.6
	70[a]	247.5	290.8	304.4	5.9
	84[a]	276.9	255.6	255.3	2.9
2					
	0	360.9	357.9	376.4	11.3
	14	336.4	343.7	344.8	3.8
	28[a]	297.2	301.4	310.6	3.9
	42	343.2	340.2	334.3	4.3
	56	342.0	335.0	340.9	4.0
	70	342.0	347.7	347.4	6.6
	84	343.7	351.5	352.6	5.9

[a]Control differs from ionophores (P<.10).

APPENDIX TABLE 7. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY SODIUM EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore None	Ionophore Lasalocid	Ionophore Monensin	SE
		------mg/100 mg creatinine------			
1	0	271.76	263.27	556.24	180.59
	14[a]	-13.26	233.99	240.23	100.72
	28[a]	-4.14	71.60	122.64	35.96
	42[b]	2.70	32.31	30.61	8.97
	56[a]	26.93	143.88	68.84	34.83
	70	15.88	100.99	39.58	25.32
	84	171.35	113.99	110.35	99.20
2	0	818.9	452.0	613.3	267.7
	14	574.3	323.5	332.4	186.6
	28	145.7	185.5	316.9	65.1
	42[a]	222.5	374.6	417.1	69.8
	56	297.7	405.6	286.2	77.2
	70[c]	88.9	338.9	371.4	76.5
	84	357.6	396.3	354.9	87.6

[a]Control differs from ionophores ($P<.10$).
[b]Control differs from ionophores ($P<.05$).

APPENDIX TABLE 8. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM POTASSIUM CONCENTRATION IN GRAZING STEERS

Trial	Day	Ionophore None	Lasalocid	Monensin	SE
		mg/100 ml			
1	0	24.56	25.60	25.10	12.90
	14	24.18	25.83	24.02	6.13
	28[a]	19.46	18.10	21.98	4.44
	42	17.82	15.27	15.62	3.34
	56[a]	14.31	13.86	15.32	1.86
	70	13.19	13.73	15.08	3.11
	84[b]	12.88	14.52	12.45	1.82
2	0	32.11	32.04	31.21	1.52
	14	31.74	33.33	32.85	1.68
	28	26.54	25.49	24.89	.65
	42[b]	19.73	17.51	14.98	1.02
	56	16.08	14.99	16.05	.68
	70	18.01	18.60	18.00	.66
	84[a]	16.15	16.54	18.35	.72

[a]Lasalocid differs from monensin ($P<.10$).
[b]Lasalocid differs from monensin ($P<.05$).

APPENDIX TABLE 9. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY POTASSIUM EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore None	Lasalocid	Monensin	SE
		------mg/100 mg creatinine-------			
1	0	2653	2953	5171	1859
	14	2873	2073	3190	669
	28	1446	2470	1287	506
	42	2238	1946	835	826
	56	1385	1226	1394	286
	70	2265	2284	1919	197
	84	304	2747	2589	559
2	0	3877	4268	5560	458
	14[a]	10982	5740	5233	2104
	28	4301	4021	4427	289
	42	4793	4263	4627	358
	56	4593	4851	3996	353
	70	4814	5590	7088	1187
	84	2924	2807	3060	254

[a]Control differs from ionophores (P<.05).

APPENDIX TABLE 10. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM COPPER CONCENTRATION IN GRAZING STEERS.

Trial	Day	Ionophore: None	Lasalocid	Monensin	SE
		ug/100 ml			
1	0	56.8	61.0	56.0	1.2
	14	59.0	62.6	56.6	.6
	28	61.0	72.9	63.8	.6
	42	69.2	74.2	72.2	.5
	56	72.1	74.6	69.8	.4
	70	73.3	74.2	69.8	.4
	84	91.7	84.0	91.8	.8
2	0	73.1	70.2	70.1	2.5
	14[a]	87.8	93.7	84.0	3.9
	28	67.8	72.9	70.9	2.5
	42	75.1	76.4	71.0	2.8
	56	66.6	74.1	66.7	3.3
	70	62.7	68.6	61.4	6.0
	84	66.2	70.8	69.1	2.9

[a]Lasalocid differs from monensin (P<.10).

APPENDIX TABLE 11. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY COPPER EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore			SE
		None	Lasalocid	Monensin	
		------ug/100 mg creatinine-------			
1	0	54.1	33.6	51.2	21.6
	14	26.0	20.7	26.6	5.6
	28	12.5	32.0	15.0	7.1
	42[a]	11.4	19.9	9.5	2.4
	56	10.8	17.6	14.9	3.9
	70	1.6	9.0	104.0	40.9
	84	26.9	22.7	23.1	5.1
2	0	37.0	40.1	56.3	6.1
	14[b]	534.4	99.8	151.6	180.3
	28	54.4	43.6	44.1	6.1
	42	69.2	64.4	66.5	12.0
	56[c]	43.3	56.2	40.3	5.0
	70	77.6	99.0	78.4	21.2
	84	58.0	37.9	43.8	10.4

[a]Control differs from ionophores (P<.05).
[b]Control differs from ionophores (P<.10).
[c]Lasalocid differs from monensin (P<.05).

APPENDIX TABLE 12. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM IRON CONCENTRATION IN GRAZING STEERS.

Trial	Day	Ionophore None	Ionophore Lasalocid	Ionophore Monensin	SE
		ug/100 ml			
1	0	200.8	190.0	197.7	4.82
	14	170.0	185.1	182.1	1.26
	28[a]	186.4	186.6	207.6	.70
	42	209.9	221.2	224.6	1.68
	56	197.2	219.9	209.8	1.57
	70	246.1	179.5	201.5	4.05
	84[b]	181.1	202.7	222.8	1.18
2	0	175.6	185.2	172.8	9.18
	14	207.3	216.7	194.8	9.28
	28	222.2	221.0	225.0	13.23
	42	199.6	225.9	223.3	12.48
	56	181.7	197.5	170.5	11.43
	70	162.6	179.6	167.2	6.95
	84	186.9	175.3	172.9	8.29

[a]Lasalocid differs from monensin (P=.05).
[b]Control differs from ionophores (P<.10).

APPENDIX TABLE 13. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY IRON EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore			SE
		None	Lasalocid	Monensin	
		------ug/100 mg creatinine-------			
1	0	188	164	240	70.9
	14	110	134	174	37.1
	28	58	154	74	36.2
	42[a]	44	126	42	22.3
	56	46	139	1070	597.7
	70	97	166	202	36.7
	84	237	264	230	63.3
2	0	188.0	118.8	293.2	66.9
	14	1299.8	451.5	364.6	408.9
	28	365.2	205.4	240.5	68.1
	42	315.6	220.7	266.3	66.9
	56	258.6	239.8	191.3	55.3
	70[b]	431.5	565.2	312.9	87.0
	84	244.7	173.1	299.3	52.5

[a]Lasalocid differs from monensin (P<.05).
[b]Lasalocid differs from monensin (P<.10).

APPENDIX TABLE 14. EFFECTS OF LASALOCID AND MONENSIN UPON SERUM ZINC CONCENTRATION IN GRAZING STEERS.

Trial	Day	Ionophore			SE
		None	Lasalocid	Monensin	
		ug/100 ml			
1	0	81.4	84.4	90.2	1.28
	14	98.0	85.5	88.6	.67
	28	90.9	86.8	92.6	.52
	42	90.3	82.8	101.6	.96
	56	85.2	85.0	87.8	.38
	70	99.1	92.6	90.1	.45
	84	84.1	95.2	94.4	.51
2	0	87.3	87.4	85.9	2.84
	14	99.1	98.0	89.2	4.41
	28	88.2	87.1	85.5	2.29
	42	84.9	86.6	83.4	3.40
	56	76.9	78.6	74.6	2.56
	70[a]	79.1	86.7	78.6	2.73
	84	90.3	86.4	87.6	3.02

[a]Lasalocid differs from monensin (P<.05).

APPENDIX TABLE 15. EFFECTS OF LASALOCID AND MONENSIN UPON URINARY ZINC EXCRETION IN GRAZING STEERS.

Trial	Day	Ionophore			SE
		None	Lasalocid	Monensin	
		------ug/100 mg creatinine-------			
1	0	75.4	63.7	106.3	34.2
	14[a]	47.0	36.8	77.6	11.8
	28	10.2	75.4	17.2	27.3
	42[a]	13.8	38.5	12.0	6.2
	56	21.1	34.0	43.6	10.7
	70[b]	28.1	50.1	97.1	18.8
	84	58.6	68.7	53.1	11.6
2	0	70.0	61.9	82.6	13.6
	14	436.8	169.6	213.5	154.3
	28[c]	109.4	53.8	61.0	15.9
	42[c]	130.5	69.2	71.0	21.3
	56	75.4	73.2	52.9	11.3
	70	89.1	143.0	99.7	29.5
	84	65.3	65.7	66.	10.7

[a]Lasalocid differs from monensin ($P<.05$).
[b]Control differs from ionophores ($P<.10$).
[c]Control differs from ionophores ($P<.05$).

APPENDIX TABLE 16. DRY MATTER AND MINERAL INTAKE BY SHEEP FED WITH OR WITHOUT IONOPHORES.

Item	Ionophore			
	None	Lasalocid	Monensin	SE
	g/d			
DM intake				
Day 1[a]	407.5	543.0	423.0	39.4
Day 5[a]	504.0	582.0	444.8	46.1
Na intake				
Day 1[a]	.415	.646	.442	.069
Day 5[a]	.572	.721	.460	.079
K intake				
Day 1[bc]	3.07	4.32	3.27	.28
Day 5[a]	3.82	4.54	3.47	.34
Mg intake				
Day 1[a]	.352	.500	.383	.042
Day 5[a]	.454	.540	.396	.050
Ca intake				
Day 1[b]	3.17	4.23	3.98	.34
Day 5	3.94	4.55	4.02	.39
P intake				
Day 1	.782	.978	.838	.096
Day 5	.98	1.07	.89	.10
Cu intake				
Day 1[a]	1.60	2.27	1.70	.18
Day 5[a]	2.01	2.45	1.77	.20
Fe intake				
Day 1	34.2	49.0	39.2	4.5
Day 5[a]	44.5	53.0	39.5	4.6
Zn intake				
Day 1	7.31	10.36	8.16	.87
Day 5	9.31	11.20	8.59	1.02

[a]Lasalocid differs from monensin ($P<.10$).
[b]Control differs from ionophores ($P<.10$).
[c]Lasalocid differs from monensin ($P<.05$).

APPENDIX TABLE 17. CORRELATION COEFFICIENTS FOR SERUM CLEARANCE AND URINARY EXCRETION RATE OF MINERALS IN SHEEP.

Item	Mineral						
	Na	K	Mg	Ca	Cu	Fe	Zn
	Serum Clearance Rate						
Trial 1							
Urine flow rate	.304[c]	.249[b]	-.183[a]	-.254[b]	.462[d]	.235[a]	.300[c]
Serum mineral concentration	.135	-.433[d]	.156	-.039	-.682[d]	.063	-.462
Urine mineral concentration	.968[d]	.691[d]	.770[d]	.927[d]	.386[c]	.547[d]	.730[d]
Trial 2							
Urine flow rate	.371[d]	.335[c]	.048	.012	.304[c]	.213[b]	.357[d]
Serum minerals concentration	.245[b]	.019	.134	.020	-.531[d]	.035	-.464[d]
Urine mineral concentration	.947[d]	.814[d]	.753[d]	.902[d]	.634[d]	.806[d]	.451[d]
	Urinary Excretion Rate						
Trial 1							
Urine flow rate	.244[b]	.270[c]	-.110	-.188[a]	.596[d]	.245[b]	.497[d]
Serum mineral concentration	.152	-.270[b]	.288	.038	-.218[a]	.056	-.189
Urine mineral concentration	.968[d]	.740[d]	.751[d]	.924[d]	.493[d]	.792[d]	.641[d]
Trial 2							
Urine flow rate	.374	.341	.045	.017	.402[d]	.289[c]	.486[d]
Serum mineral concentration	.270[b]	.189[a]	.311[c]	.090	-.092	.036	.124
Urine mineral concentration	.945[d]	.833[d]	.779[d]	.898[d]	.662[d]	.826[d]	.536[d]

[a]$P<.10$.
[b]$P<.05$.
[c]$P<.01$.
[d]$P<.001$.

APPENDIX TABLE 18. CORRELATION COEFFICIENTS FOR NET TUBULAR REABSORPTION OF MINERALS IN SHEEP.

Item	Mineral						
	Na	K	Mg	Ca	Cu	Fe	Zn
Trial 1							
Glomerular filtration rate	.990[d]	.619[d]	.964[d]	.899[d]	.646[d]	.624[d]	.672[d]
Filtered load of mineral	.999[d]	.736[d]	.980[d]	.991[d]	1.00[d]	1.00[d]	1.00[d]
Urinary excretion rate of mineral	-.244	.591	.066	0.00	.017	.233	-.075
Trial 2							
Glomerular filtration rate	.961[d]	.570[d]	.949[d]	.973[d]	.876[d]	.355[c]	.760[d]
Filtered load of mineral	1.00[d]	.560[d]	.967[d]	.998[d]	1.00[d]	1.00[d]	1.00[d]
Urinary excretion rate of mineral	.124	-.635[d]	-.031	.107	.265[b]	.098	.369[d]

[a]$P<.10$.
[b]$P<.05$.
[c]$P<.01$.
[d]$P<.001$.

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