

SUPPLEMENTAL POTASSIUM IN FEEDLOT RATIOS: INFLUENCE OF
SOURCE AND LEVEL ON ANIMAL PERFORMANCE, DIGESTIBILITY,
AND IONOPHORE ACTION

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PREFACE

This thesis is dedicated to my belated father and the vast number of cattle feeders who have applied basic principles of nutrition in an effort to produce a high quality product at maximum economic returns. The work represented in this publication was performed with one major goal in mind--that the results and knowledge obtained herein would be applicable and beneficial to the cattle feeding industry. From this perspective, the objective was met.

Scientifically, this research may have raised as many questions as it addressed. Reviewing the past 2 1/2 years of work, there are several changes I would make in the design of the experiments. Conducting the performance trial in the warm season months may have offered some insight into the issue of heat stress and potassium needs. Monitoring water intake might have provided more information on water turnover, rumen volumes, and the effect of potassium level. In the last trial, stalling animals in pens, rather than the metabolism crates, might have allowed for greater feed intakes, which would more closely parallel feedlot conditions.

This kind of endeavor would not have been possible had there not been the support of a lot of people. I would like to thank my committee members, Drs. F.N. Owens, my adviser, D.R. Gill, C.A. Hibberd and J.R. Kropp, for their direction and input. My gratitude is also extended to: Dr. Robert Totusek for the provision of an elite faculty and plentiful resources to undertake this kind of research, Dr. Leland F. Richardson

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There are three groups of people who deserve much credit. The first group--Casey Fussell, Randy Pirtle, Curtis Smith, and Ken Poling, supervisor--provided superior daily care and feed to the experimental animals. The second group, consisting of Riza Karimi, Becky Marks, Jim Trembley, John Wagner, and Kim Wilson, offered their friendship, skills, and expertise in various laboratory analyses. The animal science staff, especially Carolyn Gray and Gwen Stogsdill, are gratefully acknowledged for their cooperation and support in preparing my manuscripts.

Of the many individuals I've had contact with at Oklahoma State University, two have contributed significantly to my educational and professional development. Dr. A.L. Goetsch, serving as adviser pro-tem, offered friendship, encouragement, and invaluable advice in the planning and execution of basic research. Dr. F.N. Owens, my adviser, was extremely supportive and challenged me to integrate basic scientific research with current field practices and problems.

My greatest appreciation and thank you goes to my family. My father instilled in me the values of professionalism and a love for agriculture. My mother and brother, Gene, encouraged me to specialize in nutrition and supported me faithfully. Through the most difficult and pleasant times, it's their confidence which helped me to persevere.

Finally, it may seem unusual, but I owe special recognition to the animals participating in this research. While intensively working with them, I developed a marked respect and warmth for them. James Herriot (1977) best captured these feelings in the following quote:

All things bright and beautiful,
All creatures great and small,
All things wise and wonderful,
The Lord God made them all.

Cecil Frances Alexander 1818-1895

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CHAPTER I

INTRODUCTION

Little information about the potassium (K) requirements of feedlot cattle has been available until recently. In the past, roughages were thought to provide adequate amounts of K. However, diets based largely on milo, corn, or corn and cob meal, together with low-K hays and silages, may not provide sufficient K for fast-growing or high-producing animals. Therefore, the need for and use of K supplementation has increased.

Currently, most midwestern feedlots supplement K to attain diet dry matter concentrations between .5 and .8%. Values of the K requirements have been variable depending on whether the levels reflected maintenance, maintenance plus optimal growth, or additional effects of K on digestive function.

The majority of feedlots supplement with K from potassium chloride (KCl) or potassium bicarbonate (KHCO_3). Potassium chloride is preferred because of its lower cost per unit of K. The maximum price spread for one pound of K among eight different K supplements was \$2.69. The following list shows the variation among these supplements in percentage K and price per unit of K (Anonymous, 1985).

Source of K	Percent K	Price/lb	Price/lb K from Source
Potassium Acetate ($KC_2H_3O_2$)	39.84	\$1.11	\$2.79
Potassium Bicarbonate ($KHCO_3$)	39.05	.14	.36
Potassium Carbonate (K_2CO_3)	56.58	.43	.76
Potassium Chloride (KCl)	52.44	.05	.10
Potassium Citrate ($K_3C_6H_5O_7 \cdot H_2O$)	36.15	.94	2.60
Potassium Hydroxide (KOH)	69.68	1.08	1.55
Potassium-Magnesium Sulfate (40% K_2SO_4 and 55% $MgSO_4$)	17.95	.03	.17
Potassium Sulfate (K_2SO_4)	44.87	.86	1.92

Relative value of various K sources is unknown. The first section of this thesis deals with comparison of two sources of supplemental K each fed at two levels of supplementation.

While some literature is available concerning dietary K requirements, relatively little information exists concerning the effects of K on digestion, absorption, or rate of passage of digesta. The second section of this thesis deals with the influence of K level on digestibility and rate of passage of fluid and particulate matter from the rumen of feedlot steers.

Recent in vitro research has revealed an interaction between dietary K level and ionophores which can affect the microbial population in the rumen. The third trial of this thesis investigated the interaction of a fixed K level with three different ionophores--monensin, lasalocid, and salinomycin. Using incubations with ruminal fluid, effects of K and ionophores on ammonia levels, VFA levels, gas production, digestibility, and microbial counts were measured. The last trial, an in situ trial, explored the K-monensin interaction on the disappearance of soybean meal and ground corn from dacron bags suspended in the rumen as well as on concentrations of ruminal ammonia and volatile fatty acids and rate of passage kinetics.

Present K research has dealt primarily with ideal forms and concentrations of K. Future research must explore how K affects the microbial population of the gut, the total animal, and the economic return from feeding cattle.

CHAPTER II

REVIEW OF LITERATURE

Biochemical Functions and Regulation

Osmotic Balance Between Cells and Extracellular Fluids

The osmotic pressure within an organism dictates the migration of water and soluble substances into or out of tissues (Georgievskii, 1982b). Potassium, the third most abundant element in the body, has its major effect on cellular osmotic balance (Ammerman and Goodrich, 1983). The major cation of body cells, K serves as the osmotic pressure regulator within cells similar to the effect of sodium (Na) in extracellular fluids (Hays and Swenson, 1984).

Electrolyte concentrations differ markedly between the intra- and extracellular spaces. Levels of protein anions in the extracellular fluid are relatively low. Sodium (Na^+) and chloride (Cl^-) are largely extracellular while most of the K^+ , phosphate, and sulfate is intracellular (International Minerals and Chemical Corporation, Technical Service Department, 1981). The intracellular-extracellular separation of K and Na cations is maintained by active transport of K^+ and Na^+ across the cell membrane (Ammerman and Goodrich, 1983).

Since Na comprises more than 90% of the total osmotically active bases of extracellular fluids, changes in osmotic pressure are dependent primarily upon the Na concentration (Hays and Swenson, 1984). Under

stress conditions, Na is lost, which is compensated by an increase in extracellular K; but the organism is limited in its capacity to substitute bases, and large losses of Na lead to reduced osmotic pressure and, consequently, loss of water or dehydration.

Maintenance of Acid-Base Equilibrium

Active transport of K and Na across the cell membrane is important for some K-dependent phenomenon, including regulation of electrolyte and water distribution between body fluid compartments, intra- and extracellular pH, cellular respiration, gastrointestinal function, and urine formation (International Minerals and Chemical Corporation, Technical Services Department, 1981). Giduck et al. (1981) observed that with either a 3 or 23% soluble carbohydrate diet, addition of K to a dietary level of 4% (40,000 ppm) increased the pH of sheep ruminal fluid. No details were available on the diet composition of these treatments.

The pH of extracellular and intracellular fluids remains fairly constant, despite the formation of acidic and basic products during metabolism. Acid-base equilibrium must be maintained for the life of the organism. The acid-base equilibrium is regulated by exchange of H^+ ions with intracellular components capable of donating or accepting H^+ ions (Georgievskii, 1982b).

Certain strong cation and anions do not directly affect the acid-base equilibrium. Na^+ and K^+ ions themselves do not alter the H^+ ion concentration since they are neither acid nor base, but these cations are indispensable for regulating the acid-base equilibrium. Substitution of Na^+ or K^+ for H^+ by the kidneys or vice versa

regulates extracellular pH (Georgievskii, 1982b).

Ruminant animals have a special capacity to conserve body Na and, thereby, water and urea by action of the kidney. Yet, excretion of K by cattle, both in the feces and urine, is obligatory. Some K excretion appears necessary to prevent alkalosis, as K ions are exchanged for hydrogen ions in the kidney (Ward, 1966). But with a K deficiency and alkalosis, the concentration of basic amino acids in tissue fluids and cellular Na levels increase as a means of maintaining the cation-anion balance (Hays and Swenson, 1984).

Ionic Balance Controls Cellular Excitability and Activity

Sodium and K control the electrical activity of nerve and muscle cells (Ammerman and Goodrich, 1982). When a nerve fiber is stimulated, Na^+ ions enter the nerve cell while K^+ ions exit. This migration establishes a bio-electric impulse. Various enzyme systems trigger these reactions (International Minerals and Chemical Corporation, Technical Services Department, 1981). When extracellular K is low, the electrochemical gradient is increased, transmission of nerve impulses is impaired, and muscular paralysis can develop (Hays and Swenson, 1984).

Potassium also functions in the contraction of muscle (Fregley, 1984). Potassium influences the contractibility of smooth, skeletal, and cardiac muscle by antagonizing the effects of the calcium ion. Under conditions of salt restriction, calcium becomes very important to help maintain the K content of living tissue (Hays and Swenson, 1984).

One of the most essential muscular activities, heart beat, requires a proper balance between calcium and K to maintain rhythmicity of contractions. Excess calcium or calcium without K lengthens systole at

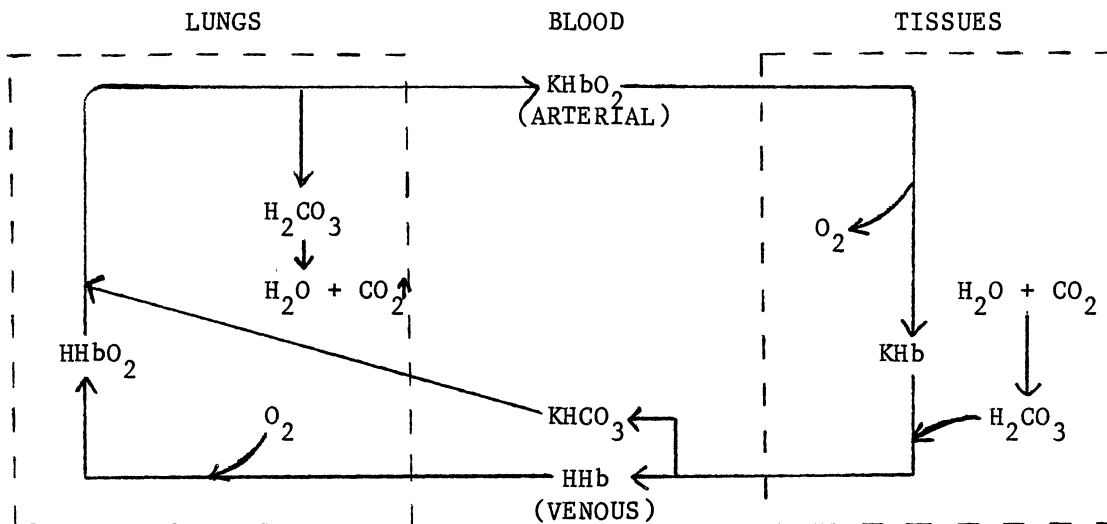
the expense of diastole, and leads to calcium rigor in which the heart stops beating in a fully contracted state. Excess K, or K unbalanced by calcium, causes the reverse reaction. Potassium excess increases diastole until the heart stops in a completely relaxed state (International Minerals and Chemical Corporation, Technical Services Department, 1981).

Acting as a calcium antagonist, K acts as a brake to suppress heart flutter and regulate heart beat. Potassium also helps prevent tetany, convulsions, and an unsteady gait (International Minerals and Chemical Corporation, Technical Services Department, 1981).

Oxygen and Carbon Dioxide Transport in the Blood

Potassium is important in the transport of oxygen and CO_2 by hemoglobin (Beede et al., 1983). The principal protein buffering agent in blood is the K salt of oxyhemoglobin (KHbO_2) contained in the erythrocytes. In the capillaries, oxygen is liberated from KHbO_2 , the reduced hemoglobin combines with carbonic acid, and KHCO_3 is formed. This is accompanied by a partial penetration of the bicarbonate ion into the plasma, while the hydrogen ion is bound by the hemoglobin.

HHb acquires oxygen in the lungs forming HHbO_2 which has the properties of a strong acid. HHbO_2 displaces the K^+ ion from the bicarbonate which diffuses from the plasma into the erythrocytes and is balanced by diffusion of Cl^- ions in the opposite direction. K^+ displaces H^+ forming KHbO_2 and releasing H_2CO_3 which in turn dissociates to CO_2 and H_2O . This total cycle is shown below:



Following the above transformations, KHbO_2 accounts for more than 70% of the buffering capacity of the blood (Georgievskii, 1982b).

Maintaining Proper Water Balance

Potassium also functions to maintain water balance in the body. Maintenance of this balance depends on both the acid-base equilibrium and osmotic pressure (Hays and Swenson, 1984).

Potassium together with other cations also acts in the rumen fluid to maintain a desirable medium for bacterial fermentation (Ward, 1966). Addition of K increases the osmotic pressure of the rumen fluid which increases the movement of water into the rumen, and thus increases moisture content of digesta in the rumen. Since K is absorbed slowly from the rumen, it is more effective than Na to cause water influx. Similarly, divalent cations which are poorly absorbed cause increased rumen moisture content (Parthasarathy and Phillipson, 1953). Balch and Johnson (1950) reported that rate of cellulose digestion by dairy cows was greater with a higher moisture content in the rumen and suggested that increased moisture content also may increase extent of cellulose

digestion in the colon.

Potassium is essential for normal physiological and biochemical reactions. With high-concentrate rations for beef and dairy cattle and sheep, dietary K intake is low. Potassium intake from high-roughage diets may be fourfold that with concentrate diets. Although tissue K deficiency is seldom a concern, osmotic differences in rumen fluid can explain some of the differences in rumen function between high-concentrate and high-roughage diets (Ward, 1966).

Cofactor In Enzyme Systems

Many enzyme systems have a specific requirement for K. Acting with other ions, such as Na, magnesium, and calcium, K also influences enzyme activity. Inside the cell, K is further concerned with most metabolic reactions which involve phosphate (Underwood, 1981).

In the glycolytic pathway, K is needed for activity of pyruvic kinase, which catalyzes the conversion of phosphoenolpyruvate and ADP to enolpyruvate and ATP. Since two moles of ATP formed per mole of glucose are the sole net profit from the glycolytic process, and since the glycolytic system appears to be universal in living cells, it can be presumed K is required universally as a catalyst (Beerstecher, 1977).

Potassium is reported (International Minerals and Chemical Corporation, Technical Services Department, 1981) to activate adenosinetriphosphatase, hexokinase, carbonic anhydrase, cholinesterase, and galactosidase. Other enzymes activated by K include salivary amylase, acetate activating enzyme, homoserine dehydrase, pyruvate kinase, phosphotransacetylase, and fructokinase.

Potassium also seems to be directly involved in the processes of

protein synthesis (Georgievskii, 1982a). Potassium influences the uptake of amino acids by cells which may be the basis for the influence of K on growth (International Minerals and Chemical Corporation, Technical Services Department, 1981). The requirement for K is affected by the growth rate of the animal and the protein level of the diet. Hence, rapidly growing animals have a higher requirement for K. Increasing the protein level increases the K requirement (Hays and Swenson, 1984), but no reason was given as to how protein intake affects the K requirement.

Regulation

Maintenance of proper K levels in the body is controlled primarily by the kidney (Clanton, 1980). Its regulation involves the mineral corticoids--aldosterone and deoxycorticosterone. Georgievskii (1982a) concluded that these hormones alter excretion of K^+ only indirectly as their prime effect is on resorption of Na^+ ions in the renal tubules. Clanton (1980) reported that hormone levels in turn are controlled by osmotic and electrolyte concentrations. Secretion of aldosterone is stimulated when the Na^+ levels in the plasma decrease and K^+ levels increase simultaneously. By eliminating excess K^+ ions through the kidneys, aldosterone maintains a constant Na:K ratio in extracellular fluids. Excretion of K^+ pulls water along, so loss of K from cells into extracellular and plasma fluids causes cellular dehydration.

Potassium deficiencies may be caused by inadequate K intake, high NaCl consumption, gastrointestinal losses, and stress conditions (Ammerman and Goodrich, 1983). Animal management systems often subject the animal to increased stress levels and thereby increase the K

requirement. Stress conditions increase the activity of the adrenal gland. As concentrations of aldosterone in blood increase, the kidneys tend to conserve Na, but increase excretion of K. This causes loss of K from the body and loss of intracellular water. While the normal kidney functions well to remove excessive amounts of K from the plasma, it has little capacity to conserve K under K deficient conditions. Renal excretion of K continues even when body K levels are dangerously low (International Minerals and Chemical Corporation, Technical Services Department, 1981).

Rumen Function

The function of K in the rumen is to maintain specific buffering and moisture conditions to produce an optimum milieu for bacterial fermentation. Potassium is indispensable for normal microflora activity including cellulose digestion (Georgievskii, 1982a).

One of the first studies of ruminal K concentrations was an in vitro test of mineral effects upon urea utilization and cellulose digestion (Burroughs et al., 1951). Potassium as KCl was added at 75, 150 and 300 ppm (in addition to the amounts contained in artificial saliva) to an in vitro flask containing a washed rumen suspension and nine grams of filter paper. Potassium additions of 75, 150, and 300 ppm did not alter cellulose digestion or urea utilization but K was already present in the mineral mixture resembling saliva at a level of 1967 ppm. In contrast, Phillipson (1953) found that cellulose digestion in the rumen was highest with K in rumen fluid between 990 and 1750 ppm.

Telle et al. (1964) observed similar trends with sheep. With dietary K levels of 3000 ppm and 6200 ppm of dry matter, ruminal K

and 1176 ppm, respectively. Ruminal Na concentrations, in contrast, decreased with K supplementation from a mixture of KHCO_3 and K_2CO_3 from 2800 ppm to 2360 ppm. Added potassium increased cellulose digestion and/or the number of rumen microorganisms, increased animal growth, and increased length of the rumen papillae. Whether the response was due to K concentration or the changes in the K:Na ratio is not certain.

Hubert et al. (1958) used a washed suspension of rumen microorganisms to study the requirements of rumen microflora for Na, K, rubidium, lithium, and cesium for cellulose digestion. Potassium was essential for in vitro cellulose digestion, whereas Na had no effect in the absence of K. Addition of Na to a fermentation medium containing 50 ppm K tended to depress cellulose digestion. With a K concentration of 100 to 400 ppm, addition of Na increased cellulose digestion. It was concluded that a minimum K concentration of 100 ppm in the fermentation medium was essential for in vitro cellulose digestion. A Na to K ratio of 5.3:1 was optimum for cellulose digestion.

Passage of Na and K ions from the rumen is a function of ruminal and blood concentrations of these two elements. Parthasarathy and Phillipson (1953) studied movement of K, Na, Cl, and water across the rumen epithelium of sheep. They discovered that Na and K were absorbed from the rumen when their ruminal concentrations exceeded concentrations in blood which are normally 3000 to 3500 and 150 to 220 ppm, respectively. Influx occurred when the rumen concentrations were lower than the blood concentrations. Potassium disappearance from the rumen was influenced by the tonicity of the solution. Potassium disappearance of .2-.4 g/hr from isotonic solutions in the rumen occurred when the K

concentration was 1330 to 2200 ppm. Sodium disappearance was .2-.4 g/hr when the rumen Na concentration was 3600-3850 ppm.

Dobson (1959) studied concentration changes and absorption from 1.5 liters of fluid in isolated reticulo-ruminal sacs. Water uptake was 100 ml. Initial concentrations of Na and K were 3427 and 919 ppm respectively. Concentrations 91 minutes later were 3082 and 868 ppm reflecting a Na uptake of 842 mg and a K uptake of 164 mg.

Various reports of ruminal concentrations of K and Na exist, and their values vary depending on the species and the type of diet. Parthasarathy and Phillipson (1953) reported K concentrations in rumen fluid were usually less than 90 meq/l (3519 ppm) in sheep but the dietary K level was not mentioned. Sellers and Dobson (1960) measured Na and K concentrations in rumen fluid of sheep fed various diets. Results are presented as follows:

Diet	Na meq	Na ppm	K meq	K ppm	Na + K meq	Na/K meq basis
Hay	66-95	1518-2185	34-71	1329-2776	133	1.53
Hay and meal	91-97	2093-2231	37-44	1447-1720	135	2.32
Fresh grass	25	575	100	3910	125	.25

The basal diet contained 100 meq/l (2300 ppm) Na and 30 meq/l (1173 ppm) K; the grass diet contained 25 meq/l (575 ppm) Na and 100 meq/l (3910 ppm) K. Emery et al. (1960) examined various hay-grain rations for beef cattle and noted the following ruminal levels of Na and K:

Diet	Sodium meq/l	Sodium ppm	K meq/l	K ppm	Na + K meq	Na/K meq basis
80% grain:20% hay	57-106	1311-2438	41-59	1603-2307	132	1.63
60% grain:40% hay	70-120	1610-2760	30-37	1173-1447	129	2.84
40% grain:60% hay	101-117	2323-2691	30-40	1173-1564	144	3.11
20% grain:80% hay	101-120	2323-2760	28-37	1095-1447	143	3.40
no grain:100% hay	108-120	2484-2760	23-44	899-1720	148	3.40

Scott (1966) noted that rumen concentrations changed as the ratio of Na to K entering the rumen changed. However, the sum of the concentrations of Na and K in sheep rumen fluid (meq/l) remained near 140 meq/l despite considerable variation in the ratios. Chow and Walker (1964) fed Na:K ratios of 4:1 (lucerne diet), 3:1 (maize diet), and 2:1 (wheat diet) to sheep. Ruminal concentrations (meq/l) of Na and K were 88-104 (2024-2392 ppm) and 23-27 (899-1056 ppm) for the lucerne diet; 77-93 (1771-2139 ppm) and 36-42 (1407-1642 ppm) for the wheat diet; and 57-77 (1311-1711 ppm) and 18-23 (704-899 ppm) for the maize diet for total meq being 121, 124 and 88 and Na/K ratios of 3.84, 2.18 and 3.27.

The predominant rumen bacteria have enzymes which require K^+ for activity. Potassium is essential for both protein synthesis and glycolysis. The K content of bacteria is high: 40-49 g/kg DM for *Bacillus* and 16g/kg for *Aerobacter aerogenes*. Sodium also is present in substantial amounts in resting bacteria, but almost absent from bacteria growing in a medium of moderate salinity (Durand and Kawashima, 1979). The workers did not specify what they considered "moderate".

Bryant et al. (1959) demonstrated the essentiality of PO_4^{-3} , NH_4^+ , Ca^{++} , K^+ , and Na^+ and established the levels required for *Bacteroides succinogenes* strain S85. Best growth occurred with 1955

ppm, 390 ppm, and 117 ppm K^+ at 1310 ppm, 1932 ppm, and 2552 ppm Na^+ levels, respectively. At lower levels of Na, higher levels of K were necessary for best growth and vice versa.

Warner and Stacy (1965) stated the culture media for rumen ciliate protozoa is most successful when the media is hypotonic. The rumen would be hypotonic only if deprived of feed. Feeding makes the rumen hypertonic with the major contributors to the rumen osmotic pressure being Na and K salts. After feeding, ruminal concentrations of Na and K in sheep were 63-139 meq/l (1449-3197 ppm) and 18-60 meq/l (704-2346 ppm), respectively.

Caldwell and Hudson (1974) ascertained that K^+ and Na^+ are required for growth of rumen bacteria. For most species, K^+ could be replaced by Rb^+ . In media containing excess Na and K as the major monovalent cations, addition of K^+ to the media containing excess Na^+ increased growth yields. Optical density doubling times of microorganisms (used as an index of growth rate) increased with increasing K concentrations. The concentration of the K^+ range that supported rapid and abundant growth of most rumen bacteria was substantially lower than the corresponding range for Na^+ , although the quantitative requirements of Na^+ and K^+ for *S. ruminantium* were similar.

It can be concluded that rather high levels (500 and 1500 ppm) of available K and Na are required for optimum fermentation in the rumen, however, quantitative requirements and physiochemical reasons for the requirement are unknown (Durand and Kawashima, 1980).

Potassium Absorption, Metabolism, and Excretion

Absorption

There are no body reserves of K other than in muscle and nerve cells. Since K is a very mobile element, it must be supplied in the ration daily (International Minerals and Chemical Corporation, Technical Services Department, 1981). Potassium carbonate, chloride, and K salts of organic acids in vegetable feeds are readily soluble and are easily extracted from feeds in the digestive tract (Georgievskii, 1982a). High absorption was noted by Ward (1966) who found that as K intake increased, K excretion in urine and in milk of lactating cows increased. Urinary K accounted for 86% of total K output of nonlactating cows and 75% for lactating cows. Milk accounted for 12% leaving 13% in feces.

Potassium is absorbed by all segments of the digestive tract, probably by diffusion, due to the low concentration of K in blood or due to an electrochemical gradient. Georgievskii (1982a) reported that K recycling via saliva is small. He cited evidence where the salivary K flux into the rumen of sheep grazing on spring pastures was 3.4 meq/l (133 ppm). The K intake from feed was 35 meq/l (1368 ppm). Bailey (1961) investigated a variety of beef cattle diets and found salivary K levels ranged from 4-7 meq (156-274 ppm) which is similar to its concentration in plasma.

The concentration of K in ruminant saliva varies strongly with the dietary intake of Na and K. With a Na deficiency, urinary Na declines to extremely low levels and fecal Na is also reduced. Sodium concentration of blood falls, and the K concentration of parotid saliva increases causing a reduction in the Na:K ratio. Replacement of Na by K

in saliva secreted by ruminants is caused by an increased aldosterone secretion by the adrenal glands together with an increase in the sensitivity of the parotid gland to aldosterone during Na deficiency (Underwood, 1981).

Bailey (1961) discovered that the concentration of K in rumen fluid was proportional to, but invariably higher than, the salivary concentration of K. Potassium is absorbed from the rumen and abomasum when its concentration exceeds that of blood (blood plasma is normally 242 ppm and whole blood is 375 ppm), but Parthasarathy (1952) found an influx of Na and K into the rumen when concentrations were below those of blood. Potassium supplementation causes the K concentration in the ruminal fluid to increase which increases the electro-chemical gradient between the blood and rumen contents (House and Van Campen, 1971).

Georgievskii (1982a) reported that in ruminants with ruminal K concentrations of 25-75 meq (977-2932 ppm), feed is the primary source of K. Potassium concentration reached a maximum 3-4 hours after feeding. In contrast, Bailey (1961) found that ruminal K concentration was maximum one hour post-feeding and decreased thereafter to its lowest value 14 hours after the meal. As K intake increases, K concentration in rumen fluid and K absorption from the rumen increase proportionally (Scott, 1975).

When the concentration of K in the diets of cows was increased from 174 to 425 g/day, rate of digestion and passage increased. The amount of chyme per kg of dry matter increased due to increased secretion of digestive juices. The concentration of K in body water increased while the concentration of Na decreased. The overall quantity of chyme passing through the intestine in 24 hours increased by 150%

(Georgievskii, 1982a). No details were available as to the K concentration of the diet dry matter or water intake.

Since the omasum receives digesta from the reticulum, omasal K concentrations should be similar to those in the reticulo-rumen. Of the K that entered the omasum from the reticulum, absorption was 9% by sheep and 8.1% by goats. The mean K absorption rate was about one-fifth (4 mmole/h) the absorption rate from the reticulo-rumen. Low absorption of K in the omasum was previously suggested from acute experiments with ligated omasa of sheep; no significant decrease in the K concentration was observed as long as concentration in the omasal fluid was kept within the physiological range. Studies on K transport across the isolated forestomach epithelium bathed in Ringer solution suggest that the omasal epithelium may function similarly to the ruminal epithelium, though the two tissues show certain quantitative differences (Engelhardt and Hauffe, 1975).

NRC (1980) states that most K is absorbed from the upper small intestine, but some absorption occurs in the lower small intestine and the large intestine as well. Giduck et al. (1981) and Greene et al. (1981) found in wether lambs and steers fed low levels of K (6000 ppm), the primary site of K absorption was the small intestine, but at higher levels [4% (40000 ppm) or 4.8% (48000 ppm)] in the diet, the preintestinal region was also important. According to Ward (1966), tonicity of the intestinal contents change from hypertonic in the small intestine to a hypotonic condition in the colon and fecal matter. The change in tonicity is a result of absorption of Na and some organic ions. Concurrently, the K concentration becomes progressively greater reaching a maximum in the fecal matter. Regardless of the K level in

the diet, only a small quantity of K was absorbed in the large intestine of wether lambs, and absorption decreased as dietary K intake increased (Greene et al., 1983a).

Annenkov (1982a) studied K metabolism of three month old calves (150 kg live weight) fed three types of diets--a semi-purified diet, a concentrate diet, and a concentrate + hay diet. Potassium concentration in the forestomach was lower than the dietary concentration indicating absorption had occurred. Potassium was secreted into the first third of the small intestine. In later segments of the small and in the large intestine, more K was absorbed than excreted. Only 1-26% of the K fed reached the lower part of the colon. The rate of mineral metabolism was greatest when the calves were fed concentrates and lowest when they were fed the semi-purified diets.

Metabolism

Increasing the K concentration in the diet from .6 to 4.8 % (6000-48000 ppm) resulted in a linear increase in K retention by sheep fed a corn grain-corn cob diet. Amounts of K retained were 1.7, 3.8, 6.7, and 13.4 g/day when .6 (6000 ppm), 1.2 (12,000 ppm), 2.4 (24,000 ppm), and 4.8% (48,000 ppm) K were fed, respectively (Greene et al., 1983b).

The bulk of the assimilated K enters the blood stream and passes from blood into the body tissues. A new equilibrium of K between the intracellular and extracellular phases becomes established within 48 hours. Potassium metabolism is rapid in muscles, kidney, liver, and brain. For lactating animals, K ions enter the milk against a concentration gradient as the K concentration in milk is several times

higher than that of blood (Georgievskii, 1982a).

Excretion

Both K and Na are excreted with urine, feces, and scint (scurf). The endogenous losses depend on the ratio of these two elements, the intake of these elements with the feeds, the water intake by the animals, and environmental temperature and humidity (Annenkov, 1982b).

Despite the relatively high concentration of K in the large intestine, feces is not the primary route of excretion for the ruminant as it represents only about 13% of the total K excretion by lactating cows. Although the contribution of endogenous K to fecal K is uncertain, extensive absorption of K suggests that fecal K arises primarily from endogenous sources (Ward, 1966).

Since dietary K is practically 100% absorbed, all animals eliminate K principally through the kidneys. Of the total K excreted, the amount excreted in the urine is 75 to 86% for cows, 85 to 88% for sheep, and 90% for pigs. Neither the amount of K consumed nor feed intake substantially affects the amounts of K eliminated by the two routes (Georgievskii, 1982a). High levels of K can be tolerated by cattle as they rapidly excrete the element (Scotto et al., 1971). As K intake increases, the amount of K excreted via both the kidneys and intestine increases.

In the urine, K and Na occur as inorganic salts (phosphates, sulphates) and organic salts (urates). In contrast with carnivores, ruminants exchange large amounts of K^+ ions for H^+ ions in the kidneys (Georgievskii, 1982a).

Differences Among Supplemental Potassium Sources

Bioavailability

The availability of an element for an organism is called its bioavailability. Feed ingredients supply energy, amino acids, and many essential mineral elements. Often these elements are in complex forms that may not be bioavailable. This makes selection of mineral supplements important (International Minerals and Chemical Corporation, Technical Services Department, 1981).

Apart from economics, bioavailability is a major factor influencing the selection of supplemental forms of minerals. Some of the factors which influence bioavailability include age of the animal, levels of interacting elements, and particle size (International Minerals and Chemical Corporation, Technical Service Department, 1981).

Definitive information of K bioavailability from various K sources is lacking as shown below. However, it appears that the chloride, biphosphate, acetate, sulfate, and citrate salts of K are similarly high in K bioavailability (Miller, 1983). Carbonate and bicarbonate salts should be high, as well.

Potassium Source	RBV ^a		
	Poultry ^b	Swine ^b	Ruminants ^b
KCl	100	100	100
K ₂ CO ₃	---	100	100
KHCO ₃	---	---	100
K ₂ HPO ₄	100	100	---
K ₃ C ₆ H ₅ O ₇ ·H ₂ O	100	---	---
KC ₂ H ₃ O ₂	100	100	---
K ₂ SO ₄	100	---	---
Concentrates	---	---	---
Forages	---	---	---

^aRBV = Relative biological value using KCl as the standard (100).

^bFrom requirement and plasma K level studies of the International Minerals and Chemical Corporation (1983) and Peeler (1972).

Palatability

Rations consisting primarily of concentrates do not contain sufficient K to meet the requirements of cattle; thus, supplemental K salts need to be added to the ration. The choice of the K salt is important. It should be palatable and, when added to the diet, meet the K requirements for optimum health and performance of calves but not cause toxicity or adversely affect performance (Ward, 1966).

In a study conducted by Neathery et al. (1980) the palatability of four sources and four levels of K were determined by offering K to Holstein bull calves in a cafeteria arrangement. Sources of K included KCl, potassium acetate, K₂CO₃, and KHCO₃. The basal concentrate

diet contained .77% K (7700 ppm) and other diets contained 2 (20,000 ppm), 4 (40,000 ppm), and 6% (60,000 ppm) K from the various sources. Groups of 5 to 6 calves were offered a choice of only two test diets for 3 to 7 days.

Potassium chloride-supplemented feeds proved more palatable than the K_2CO_3 -supplemented diets, but less palatable than diets containing $KHCO_3$ or potassium acetate. Palatability decreased with increased dietary amounts of K and feeds supplemented with 2 (20,000 ppm), 4 (40,000 ppm), and 6% (60,000 ppm) K as KCl and 2 (20,000 ppm) or 4% (40,000 ppm) K as potassium acetate, K_2CO_3 , and $KHCO_3$ were consistently less palatable than the control diet. Ferrell et al. (1983) noted sorting of fine particles with higher KCl levels in a 95% concentrate feedlot diet. They suggested this might be due to the bitterness of KCl.

Animal Performance

Potassium source may contribute to animal performance if it influences feed intake, efficiency of feed conversion, and/or average daily gain. Some researchers have claimed that K source may alter digestive physiology through alteration of rumen buffering or rate of passage (Beede et al., 1983). Differences in carcass characteristics and milk yield also have been observed with various K sources, and research supports the concept that the K source may affect the health of the animal.

The K source may influence feed intake, average daily gain, and the efficiency of feed conversion through the action of the anion with which it is associated. Bicarbonate or carbonate should increase ruminal pH

and counteract the acidic conditions created by feeding high energy diets, thereby increasing the efficiency of ruminal fermentation and feed utilization. Bicarbonates also could possibly increase osmotic pressure, increasing both rate of passage of digesta through the forestomach and feed intake. These mechanisms collectively should increase production (Beede et al., 1983).

Including 5% (50,000 ppm) KHCO_3 in the finishing ration of lambs decreased feed intake and average daily gain (Kunkel et al., 1953). This level of K may have been excessive. Roberts and Driedger (1966) found that a dietary level of .6% (6000 ppm) K was required in a finishing lamb ration. Lambs fed supplemental K in the form K_2CO_3 had greater weight gains and feed consumptions than lambs fed supplemental KCl. Including both KCl and K_2CO_3 in the diet increased daily feed consumption of lambs by 30 g above that with the K_2CO_3 in the ration, but weight gain was reduced by 71.4 g/day below the K_2CO_3 ration. Calhoun and Shelton (1982) tested K sources for high concentrate lamb rations. Their basal K level was .46% (4600 ppm). Final K levels with added KCl were .82 (8200 ppm) and 1.22% K (12,200 ppm); final K levels with KHCO_3 were 1.04 (10,400 ppm) and 1.52% K (15,200 ppm). KHCO_3 increased live weight gains, feed intake, and feed efficiency the first 28 days, but not during the remainder of the trial. No significant response was observed to KCl supplementation. The researchers suggested that the KHCO_3 response noted during the first 28 days was due to the buffering effect of HCO_3^- . Rumen pH was 5.1 at slaughter but did not differ with K source.

Nicholson et al. (1960) added a special mineral supplement composed of KHCO_3 plus bicarbonate and carbonate salts of calcium, magnesium,

and Na to a diet of corn cobs, corn starch, soybean oil, and urea. Calves receiving this ration had improved feed consumption and average daily gain. The mineral-supplemented calves had alkaline urine (8.07 pH) while calves fed the basal ration had acidic urine (5.88 pH). In other beef cattle trials conducted by Oltjen et al. (1965), Wise et al. (1965), Algeo et al. (1965), and Kelley and Preston (1984), the source of supplemental K did not significantly affect feed consumption, average daily gain, or feed efficiency.

In a dairy trial testing buffers and heat stress, Beede et al. (1983) noted that KHCO_3 added at 1.0% (10,000 ppm) of the diet [1.2% K (12,000 ppm) final ration] reduced feed intake and milk yield. Reduced feed intake could have been due to a palatability problem, since the KHCO_3 was mixed with a complete blended dry diet containing cottonseed hulls, corn gluten meal, and ground corn grain. Blood pH, pCO_2 , and HCO_3^- values, all indices of acid-base status, were not significantly altered. Hence, feeding a buffer to an animal showing signs of alkalosis did not compromise the animal's acid-base balance.

The bicarbonate ion or KHCO_3 per se may possibly affect fat deposition or fat transport so that animals fed KHCO_3 would have fatter carcasses than animals fed KCl. If true, then carcass dressing percentage and numerical yield grade should be greater for animals fed KHCO_3 supplements (Algeo et al., 1965).

Calhoun and Shelton (1982) noted no significant differences in carcass dressing percent or yield grade of lambs fed either a KCl or KHCO_3 -supplemented ration. Algeo et al. (1965) had similar results with steers fed high concentrate diets containing KCl or KHCO_3 [final K level = .75% (7500 ppm)]. However, there was a trend toward fatter,

higher grading carcasses from the animals fed KHCO_3 than animals fed either KCl or the basal .42% K (4200 ppm) diet. In a trial by Kelley and Preston (1984), fat thickness was greater but yield grade was less for steers fed KHCO_3 than steers fed KCl. No explanation was given as to how fat thickness can increase while yield grade decreases. Dressing percent, hot carcass weight, kidney-pelvic-heart fat, rib eye area, and quality grade were unaffected by dietary treatment. On the other hand, Oltjen et al. (1965) found 2.1% K_2CO_3 addition to steer diets [.76% K (7600 ppm) basal level; 1.78% K (17,800 ppm) final diet level] significantly reduced marbling score, and depressed carcass grade by one-third of a grade. Fat cover over the rib eye was depressed by 3.1 mm, contributing to a lower dressing percentage. Buffers also significantly increased the percentage bone in the 9th-10th-11th rib cut by 1.5%. Slaughter weights among treatments were similar.

The source of K may have an effect on the health of the animal as well. Potassium tends to decrease the incidence of urinary calculi in lambs. At a dietary level of 1% (10,000 ppm) K, the degree of protection from urinary calculi was dependent on the anion or chemical radical associated with the K cation. The incidence of urinary calculi was increased with the monohydrogen phosphate anion, while the chloride, bicarbonate, and carbonate anions reduced the incidence of calculi with the bicarbonate anion being the most effective (Crookshank, 1966).

Development of Potassium Recommendations

Only recently have the K requirements of ruminants been extensively studied. Roughages were considered to provide adequate levels of K, and nutritionists regarded K as an essential but not a critical nutrient

(International Minerals and Chemical Corporation, Technical Services Department, 1981). Crampton and Lloyd (1959) stated "the abundance of K in both plant and animal foods precludes the danger of a deficiency of this element in a mixed diet." Ward (1966) wrote that "dietary deficiencies of this element are very uncommon, if not unknown, and any natural diet consumed by ruminants would probably never be deficient in K." Even the NRC (1976) states that "K requirements would be amply met with high forage diets."

In recent years, interest in high concentrate rations for beef, dairy cattle, and sheep has increased. One of the greatest differences between high-roughage and high-concentrate rations is that K intake may be four-fold greater in the roughage diets. Rations containing some roughage are probably adequate in K, but the possibility of a K deficiency exists for animals fed high-concentrate rations (Ward, 1966).

Dairy Cattle

With the increased use of high-concentrate, low-roughage rations and nonprotein nitrogen sources in the feed, the modern dairy cow may become K deficient. The most prevalent symptom of K deficiency is reduced feed intake with a corresponding decrease in milk production (International Minerals and Chemical Corporation, Technical Services Department, 1981). This symptom is difficult to detect since feed intake also will change with many animal and environmental conditions.

Du Toit et al. (1934) first investigated the K requirement for dairy cattle. They reported a ration containing .32% (3200 ppm) K was adequate to maintain milk production at two gallons per day over a period of two lactations. Although this work was based on two cows, it

was commonly cited as evidence that no supplemental K was needed by the dairy cow. Ward (1966) stated that the dietary K requirement of ruminants was probably no more than .5% (5000 ppm) of the ration.

A change-over feeding experiment using eight lactating dairy cows was conducted by Pradham and Hemken (1968) to study K deficiency symptoms. Potassium deficient [.06% (600 ppm) and .15% (1500 ppm) K] and K adequate (.8% or 8000 ppm K) rations were prepared using a mineral mix of KCl and K_2CO_3 . Deficiency signs appeared in about 3-4 weeks in cows on the .06% (600 ppm) K rations. The deficient cows decreased feed intake by 34% and milk production by 32%. Other deficiency symptoms included pica, loss of hair glossiness, decreased pliability of the hide, lower K concentrations in blood plasma and milk, and higher hematocrit. The hematocrit reading reflects body dehydration with the low K diet.

The mineral element present in highest concentration in cow's milk is K, and a high producing dairy cow excretes large quantities of K in the milk daily. Sasser et al. (1966) reported that even under conditions of widely varying K intakes, the K concentration of the milk is constant. Concentrations of the K in milk differed among cows but not among rations.

Dennis et al. (1976) fed three dietary levels of K [.45 (4500 ppm), .55 (5500 ppm), and .66% (6600 ppm)] to Holstein cows past the peak of lactation. Dietary K level had no impact on milk fat, solids-not-fat content, milk production, or blood serum cations. However, feed intake was affected, with the cows fed the .66% (6600 ppm) K level ingesting 4.1 kg more feed than cows fed the .45% (4500 ppm) level. Body weight gain of the cows on the highest K level was sixfold that of the cows on

the lowest level of K. In an early lactation study by Dennis and Hemken (1978), three levels of K [.51% (5100 ppm), .75% (7500 ppm), and .99% (9900 ppm)] were investigated. Milk production and change in body weight differed. Feed intake changes when cows were switched from test diets [.51 (5100 ppm), .75 (7500 ppm), and .99% (9900 ppm) K] to the .99% K (9900 ppm) ration were +3.4, +1.0, and +.5 kg per day, respectively. Milk production and body weight changes did not differ. Blood serum changes in percent K were -.6, -.4, and +1.1 for the three treatments.

In a mid-lactation study, Dennis and Hemken (1978) noted feed intake changed +1.3, -.3, and +.1 kg for cows fed .46 (4600 ppm), .69 (6900 ppm) and .97% (9700 ppm) K rations after a control period during which all cows were fed the high K diet. Body weight and milk production were affected by treatment. The feed intake and serum K levels indicated that .69% (6900 ppm) K might not be high enough for high producing cows in early lactation.

Erdman et al. (1980) reported that with first lactation cows in midlactation, increasing K concentration from .52 (5200 ppm) to .77 (7700 ppm) or 1.04% (10,400 ppm) of the ration increased feed intake. In a second trial with midlactation cows, increasing dietary K from .42 (4200 ppm) to .84% (8400 ppm) increased dry matter intake 1.2 kg per day but had no effect on fat corrected milk. In both experiments, increasing dietary K to .77 (7700 ppm) and .84% (8400 ppm) increased serum K.

The current recommendation for K in dairy cow diets is .8% (8000 ppm) (NRC, 1978). This level may be adequate except in cases of lactational and/or heat stress. Lactational stress is the result of

extremely high milk production with high K output in the milk. Since 25 to 40% of the dietary K is excreted in milk, high producing cows need K continuously to maintain high milk production. At peak lactation the K requirement may exceed 1% (10,000 ppm) of ration dry matter.

Heat stress also increases K losses through sweating, and in addition reduces feed intake and lowers milk production. Beede et al. (1983) reported that increasing K from .66 (6600 ppm) to 1.08% (10,800 ppm) increased feed intake and milk yields in heat stressed cows, but increasing total dietary K further (to 1.64% or 16,400 ppm) decreased milk yield and feed consumption. Sodium level in this trial was not mentioned. In a subsequent trial by Beede et al. (1983), importance of the relative proportions of dietary K and Na was suggested. Heat stressed dairy cows responded to higher [1.5% (15,000 ppm)] dietary K by increasing milk yields when an adequate level of Na [.67% (6700 ppm)] was provided in the diet.

The fertility of cows also can be affected by the intake of Na and K with the diet, and particularly by the ratio of these two elements (Georgievskii et al., 1982a). A Na:K ratio of 1:5 appears optimal; if the ratio exceeds 10:1, pregnancy rate is reduced. The author gave no reason for this.

Sheep

The current estimate of the K requirement for sheep is .5 to .8% (5000 to 8000 ppm) of the diet (NRC, 1985). However, relatively little research has examined K requirements by sheep for optimum performance. The earliest study to ascertain the K recommendations for sheep was conducted by Eaton and Avampato (1952). Comparing hay diets containing

1.6% (16,000 ppm) K or 3.2% (32,000 ppm) K, they noted that body weight gains did not differ.

Telle et al. (1964) reported the minimum K level for growing and finishing lambs was near .34% (3400 ppm) of the ration dry matter, but the optimum level was .55% (5500 ppm) K. Growth depression occurred at levels less than .3% (3000 ppm) K, and higher levels [.81% (8100 ppm)] were not as effective as .55% (5500 ppm) K. Rumen fluid analysis showed that K concentration increased as dietary K increased while Na concentration decreased. Since in vitro cellulose digestion can be depressed with a high Na:K ratio, Telle and his associates proposed the higher Na:K ratio at the lower K dietary level decreased activity or reduced numbers of rumen organisms and, thereby, reduced growth rate of lambs. Ruminal papillae length also increased with higher dietary K levels which could increase the absorptive surface and indirectly increase animal performance.

Optimal feedlot performance by lambs was observed with a ration containing .3 (3000 ppm) to .5% (5000 ppm) K (Campbell and Roberts, 1965). With a low K diet [.1% (1000 ppm)], lambs exhibited decreased appetite, low serum levels of K, and a decreased nitrogen retention. However, K intake had no effect on apparent digestibilities of nitrogen, dry matter, or energy. Although supplemental K was provided in the K_2CO_3 form, rumen fluid pH was not affected by the dietary level of K. These workers concluded that the K requirement for growing lambs was between .3% (3000 ppm) and .5% (5000 ppm) of the ration.

Reffett and Boling (1982) fed diets as follows: (1) basal, (2) basal plus 4% K from KCl, (3) basal plus 4% Na from NaCl, or (4) basal plus 2% K from KCl and 2% Na from NaCl. Water intake and urinary output

were highest with the diet containing 4% Na and were lowest with the basal diet. Dry matter digestibility was similar across treatments; ADF digestibility and apparent crude protein digestibility were greatest with the 4% Na diet and lowest with the 2% Na and K diet. When expressed as a percentage of nitrogen intake, nitrogen retention in lambs fed 4% K and 2% K/2% Na was slightly greater than the control lambs.

Energy retention and growth of lambs may be depressed at high K intakes. Levels of dietary K from .7 (7000 ppm) to 3.0% (30,000 ppm) linearly decreased energy retention and weight gain of lambs (Jackson et al., 1971). However, feed consumption and net energy for maintenance and growth were not altered by the various Na and K treatments. Giduck et al. (1981) also reported that high dietary K levels [4% (40,000 ppm)] may alter the metabolism of lambs fed high or low soluble carbohydrate diets. High K intakes decreased magnesium absorption, decreased ruminal pH, and depressed serum magnesium and calcium levels. High K intake is a contributing factor to grass tetany.

Greene et al. (1983a) observed that magnesium absorption from the rumen was depressed with dietary K levels of 2.4 (24,000 ppm) and 4.8% (48,000 ppm), but dietary K altered neither calcium absorption at any site in the digestive tract nor total phosphorus absorption. Magnesium retention was not altered, but calcium retention increased when K was added above .6% (6000 ppm) of the ration.

Beef Cattle: Range Conditions

Little work has been done on the K requirement for beef cows and steer calves grazing range. It had been generally accepted that forages

contained enough K for optimal production, though recent work has suggested supplemental K might enhance animal performance under winter range conditions.

Karn and Clanton (1977) conducted a series of experiments to determine the effect of K supplementation on weight changes in weanling steer calves and bred cows grazing native winter forage. Weight gains were increased when K was added to a supplement containing urea. While a K requirement was not stated, it was suggested that supplements fed at a rate of .68 kg/day to weanling calves on winter range should contain at least 2.0% (20,000 ppm) K.

Waggoner et al. (1979) reported a benefit from K supplementation of bred cows on native winter range. A 37% crude protein block, containing either 2.25 (22,500 ppm) or 4.15% (41,500 ppm) K, was fed free choice. Compared to the cows on the low K supplement, cows receiving the higher level of K supplementation lost less weight during calving and early lactation, had a higher rate of conception, and nursed calves were 7.27 kg heavier at branding at about 3 months of age.

In the following winter, Waggoner et al. (1980) compared the performance of beef cows fed either .91 kg of a .6% (6000 ppm) K, high energy cube (intake equal to 5.5 g K/day) with free choice access to a high energy molasses block [4.0% (40,000 ppm) K], for which intake averaged .62 kg per day (24.8 g K/day). Cows fed the high K blocks gained more weight from December to April and calves gained more while nursing. Cows fed the lower K cubes lost less weight between calving and weaning, but this may have been due to greater energy intake with the high energy cube.

Potassium was supplemented at rates of 0 or 57 g daily from

December to March for cows grazing Texas native range averaging .23% (2300 ppm) K (Hutcheson, 1980). Potassium supplementation gave 5.8% higher calf survival, weaned calves were 37 lb heavier, cows lost less weight during the winter, and conception rate was higher. These changes reflect a need for K supplementation with low K forages as K affects body weight, milk production, and reproductive performance.

In summary, the K requirement for calves growing at a slow rate, such as on range, may be .3 (3000 ppm) to .4% (4000 ppm). The requirement of the gestating cow is probably higher [.5 to .7% (5000-7000 ppm) K], though this suggestion is based on limited data. The minimum maintenance requirement of K for yearling steers appears to be near 75 meq (3 g) per 100 kg body weight per day (Clanton, 1980).

Beef Cattle: Receiving Diets

Cattle subjected to marketing and shipping stresses undergo several metabolic changes. Weight is lost both from water from the digestive tract and from body cells. The cellular loss of water can create deficiencies of both K and Na. Most feedyard diets contain .5% salt (NaCl) and .6-.8% (6000-8000 ppm) K. Since the suggested requirement for Na is .08% (800 ppm), the Na intake is more than twice the requirement. However, with K fed at the requirement of .6-.8% (6000-8000 ppm), K may be insufficient for rapid repletion of body fluids.

Receiving diets containing 1% (10,000 ppm) K (basal) and 1.5% (15,000 ppm) K were compared by Hutcheson and McLaren (1978). Addition of K improved gains by .05 kg/head over the first 28 days after arrival. Phillips and McLaren (1981) noted a similar post-transit response in

Angus calves receiving even a higher level of K supplementation. Increasing dietary K from .8% (8000 ppm) to 1.5% (15,000 ppm) K increased gains by .35 kg over the first 21 days of feeding though dry matter intake was not altered. Therefore, feed efficiency was improved.

Cole and Hutcheson (1982) examined two levels of K [.8 (8000 ppm) and 1.3% (13,000 ppm)] in receiving rations. They observed that K level had no affect on daily gains or gain:feed ratios though the higher K level reduced mortality by 5% suggesting that it improved calf health.

In a later study of post-transit K levels for feeder calves (Hutcheson et al., 1984), K addition to the diet after arrival improved the performance of transported calves. Blood packed cell volume (PCV) increased as the receiving diet K level increased from .7 (7000 ppm) to 2.2% (22,000 ppm). This increase in PCV could reflect dehydration, increased red blood cell count, or, the authors stated, it could be a result of more rapid influx of K and water into the red blood cells and increased mean corpuscular volume. Blood hemoglobin was not measured. Increases in serum K, whole blood K, and decreased serum osmolality suggested that rehydration was more rapid among calves fed the higher K levels. The K level recommended for transported calves was calculated to be 24.7 g/100 kg of body weight [or 1.3-1.4% (13,000-14,000 ppm) dry matter basis of the receiving diet] for the first 2 weeks after arrival. This was 20% more than the requirement for non-transported calves.

In contrast, Zinn et al. (1983) concluded from two 56 day receiving trials of crossbred calves that an increase in dietary K from 1.0% (10,000 ppm) to 1.5% (15,000 ppm) did not enhance animal health, rate of gain, or feed efficiency. However, calves consuming the 1.5% (15,000 ppm) K diet tended to make faster gains during the first two weeks of

the trial. In a metabolism trial involving four cannulated calves weighing 447 lb, increasing K concentration of the diet from 1 to 1.5% (10,000 ppm to 15,000 ppm) did not alter total tract digestion of organic matter, fiber, or protein, but ruminal organic matter digestion tended to increase (5.4% increase). Most of this increase was attributed to an increase in ruminal fiber digestion. Neither ruminal protein bypass nor microbial protein synthesis was influenced by K level.

Beef Cattle: Finishing Rations

The history of K nutrition for beef finishing rations parallels that of dairy cattle. Few studies were conducted with K until the middle 1960's. It was then recognized that diets based largely on milo, corn, or corn and cob meal together with low K hays and silages, are deficient in K for fast growing or high producing animals.

Neathery et al. (1980) offered Holstein calves a concentrate diet [basal K = .77% (7700 ppm)] containing 2% (20,000 ppm), 4% (40,000 ppm), or 6% K (60,000 ppm). Feed intake and growth were unaffected at 2% (20,000 ppm) K though 6% (60,000 ppm) K decreased voluntary feed intake and weight gains. They concluded that the maximum dietary K which produced no clinical toxicity was between 2.8 and 7.0%.

The first research aimed at establishing a K requirement for fattening steers was conducted by Roberts and St. Omer (1965). In their first trial, K levels ranged from .27% (2700 ppm) to .85% (8500 ppm) K with supplemental K provided as K_2CO_3 . Ruminal pH, Na, and K levels showed slight increases as dietary K was increased although daily feed consumption and weight gains increased with K levels up to .72% (7200

ppm). In their second and third experiments, ration K levels were from .56 (5600 ppm) to 1.05% (10,500 ppm). In these experiments, dressing percent, carcass grades, and weight gains were not significantly affected by treatment. They suggested a dietary requirement for fattening steers between .5 (5000 ppm) and .6% (6000 ppm) of the ration dry matter.

In contrast, Algeo et al. (1965) suggested that most practical diets containing .43% (4300 ppm) or more K do not require K supplementation for optimal growth. Adding K to a .42% (4200 ppm) K diet to increase K to .75% (7500 ppm) with both barley and corn diets did not increase daily gain, carcass corrected daily gain, or carcass measurements. While a trend toward improved feed conversion by the K addition to the corn basal diet was apparent, the difference was attributed to chance.

Results of K balance studies with heifers by St. Omer and Roberts (1967) indicated that Na and nitrogen balances were not affected by K intake, but there was a negative K balance at the low K level (6122 ppm K/day). No difference in the apparent digestibilities of energy, dry matter, nitrogen, crude fiber, or ether extract were detected ($P > .05$). Serum K level was lower and serum phosphorus levels were higher in heifers fed a low K level. Water consumption and urine volume were higher on the high K diet, but water balance was not affected. The daily maintenance K requirement was estimated to be 133 meq (5.3 g) per 100 kg body weight and the daily K requirement for maximum feed intake and growth was 278 meq (11.1 g) per 100 kg body weight.

Devlin et al. (1969) suggested that the weight gain and feed efficiency values from two trials indicated that the K requirement for

finishing beef steers was between .62% (6200 ppm) and .72% (7200 ppm) of the ration dry matter. K_2CO_3 was added to a low K basal diet in two experiments to create dietary K levels (DM) ranging from .27 (2700 ppm) to .85% (8500 ppm) K in the first trial and .36 (3600 ppm) to .77% (7700 ppm) K in the second trial. Potassium deficiency, as indicated by poor appetite, loss of body weight, pica, and low serum K levels, was observed in steers fed diets containing .27 (2700 ppm) and .36% (3600 ppm) K. Rumen fluid levels of Na and K, in vitro microbial activity, and rumen fluid pH increased as dietary K was increased from .27 (2700 ppm) to .85% (8500 ppm). Relatively high rumen pH levels (7.0-7.4) were probably due to the supplementation of K as K_2CO_3 .

In the first of two experiments, Standish et al. (1974) fed a 90% concentrate, 10% roughage ration to crossbred steers with or without addition of .35% (3500 ppm) K. In the second experiment, a 92.5% barley and 7.5% supplement was fed with or without .38% (3800 ppm) supplemental K added. Basal K levels were not mentioned. Gains were increased with K supplementation by 11.3% in the first experiment and by 5.8% in the second experiment. Addition of K also increased efficiency of feed use but did not affect carcass characteristics.

Several trials were conducted at Oklahoma State University to examine the effect of supplemental K. In one of the earlier trials, Zinn and Owens (1980) fed 45 heifers a high concentrate corn-based ration [.66% (6600 ppm) K] with and without 2.5% supplemental $CaCO_3$ or 1% (10,000 ppm) supplemental KCl [.5% (5000 ppm) supplemental K]. Daily gain and feed efficiency tended to favor the supplemented diet. Dry matter digestibility was significantly greater for the KCl-supplemented ration than for the $CaCO_3$ -supplemented ration, but the control and

KCl-supplemented rations did not differ in digestibility. Level of K in the control ration was .66% (6600 ppm), within the NRC recommendations for K. In a later trial, Zinn et al. (1982a) supplemented a high moisture corn diet [.48% K (4800 ppm)] with KCl to increase dietary K to 1% (10,000 ppm) of the ration for feedlot steers. Potassium supplementation increased feed intake, live weight gain and dry matter digestibility slightly, but when corrected for the small difference in dressing percentage, gains and efficiencies were influenced little by added K. Dressing percentage tended to be lower for the steers fed the 1% (10,000 ppm) K ration. These results contrast with those of another trial by Zinn et al. (1982b) in which supplementing K to 1% (10,000 ppm) of the diet slightly reduced feed intake but improved live weight gain and feed efficiency by 21%. These workers postulated that these results may have been due to water retention in the tissues and had dressing percentage been determined and weights adjusted for dressing percent, the gains would not have been improved.

Zinn and Axe (1983) fed Holstein steers three ration conditioner combinations at two levels of K [.65 (6500 ppm) and 1% (10,000 ppm) dietary K]. Steers receiving the 1% (10,000 ppm) dietary K gained 5% more rapidly than steers receiving the .65% (6500 ppm) level. Feed intake, feed efficiency differences, and dressing percentages were not changed. In a smaller crossover trial, they evaluated the impact of added K [.65 (6500 ppm) versus 1% (10,000 ppm)] on four cannulated calves. Differences in total tract digestion were non-significant ($P > .10$) but ruminal digestion tended to be increased at the 1% (10,000 ppm) K level, with a 28% increase in ruminal acid detergent fiber digestion and an 8% increase in ruminal nitrogen digestion.

Currently the NRC (1984) recommends K be fed between .5% (5000 ppm) and .7% (7000 ppm) with a suggested value of .65% (6500 ppm) and a maximum tolerable level of 3.0% (30,000 ppm). The recommendation of a range in the requirement rather than a specific value recognizes that the requirement is not precisely known and, as for most minerals, needs are affected by a variety of dietary and animal (body weight, sex, rate of gain) factors.

Effects of Ionophores

In the beef animal, nutrition can be divided into two distinct but interrelated entities. The first entity is the animal and encompasses the animal's physiology; the second entity is ruminal fermentation and focuses on the microorganisms in the rumen. The goal is to maximize total animal performance by manipulating and improving the efficiency of ruminal fermentation. This can be accomplished through increasing ruminal propionic acid yield, depressing methanogenesis, and depressing ruminal proteolysis and deamination of dietary proteins (Bergen and Bates, 1984).

In the last decade, a class of compounds has been detected which can accomplish some of these objectives. These compounds are the carboxylic polyether ionophore antibiotics which are produced by various strains of *Streptomyces*. Called ionophores, these substances interact with metal ions and carry these ions across lipid membranes (Bergen and Bates, 1984). The three major ionophores currently marketed are monensin, lasalocid, and salinomycin. Including ionophores in the diet changes the animal's mineral metabolism and physiology of tissues dependent on ion movement (Elsasser, 1984).

In the following pages, modes of action of ionophores are described. Most of the research to date has focused on monensin, though salinomycin and lasalocid will be mentioned in cases where sufficient research has substantiated their action. A summary table of the effects of three ionophores on metabolism of sheep or cattle is presented.

Mineral Metabolism

Classification of a compound as an ionophore is based upon its ability to facilitate or increase metal ion movement across hydrophobic lipid membranes. Ionophores form soluble complexes with the hydrated metal ions. Lipid membranes are more permeable to these complexes than to non-complexed ions. While the movement of the complex through the membrane is usually passive, movement of an ion against its concentration gradient may also be facilitated through the counter-transport of another ion in the opposite direction. Thus, an ionophore can influence the transport of ions in addition to those with which it binds (Elsasser, 1984). This phenomenon is called the "anti-porter system".

Monensin, lasalocid, and salinomycin transport monovalent alkali metals such as Na^+ and K^+ . However, they vary in their affinities for these metal ions (Elsasser, 1984). The relative affinities of monensin are $\text{Na}^+ > \text{K}^+ > \text{Li}^+ > \text{Rb}^+ > \text{Cs}^+$ while lasalocid's affinity pattern is $\text{K}^+ > \text{Rb}^+ > \text{Na}^+ > \text{Cs}^+ > \text{Li}^+$. Monensin mediates primarily $\text{Na}^+ - \text{H}^+$ exchange because the affinity of monensin for Na^+ is ten times that for K^+ , the nearest competitor. Lasalocid displays a higher affinity for K^+ and equal affinities for Ca^+ and Na^+ . These ionophores also vary in their binding ratio of the ionophore to

the metal. Monensin binds monovalent ions in a 1:1 ratio whereas two molecules of lasalocid bind one monovalent ion. Transport rates of the complexes depend upon the affinity constant of the drug for the ion and in part upon the environment and physical factors.

Ionophores can alter mineral balance and ion transport in several ways (Elsasser, 1984). These include:

- (1) increased bioavailability of ions from feed and water,
- (2) increased ion uptake and transport across biological membranes and tissues,
- (3) altered distribution and storage in cells, tissues, and bones,
- (4) modified element-element interactions, and
- (5) altered homeostatic and regulatory mechanisms controlling uptake, concentration, and excretion.

Modification of Digestibility

Monensin may initially reduce and later increase digestibility. This has been observed for dry matter, cellulose, and acid-detergent fiber. Initially, inhibition of certain bacterial strains in the rumen reduces rate of digestion. Later, due to altered bacterial activity or for other unknown reasons, digestibility rebounds and may overcompensate. Dry matter digestibility of forages by cattle is often increased with adaptation to monensin. Likewise, dry matter and starch digestibilities by cattle have increased with adaptation to monensin. Once the animals are adapted to monensin, NDF and ADF digestibilities are not depressed (Schelling, 1984). Ricke et al. (1984) noted no significant differences in dry matter and fiber (NDF, ADF, or hemicellulose) digestibilities in lambs fed either lasalocid or monensin.

Roughage and protein level in the diet can alter monensin effects. Monensin increases nitrogen digestibility by cattle fed low protein

diets. Higher nitrogen digestibilities have been noted with cattle fed low, medium, or high roughage diets (Schelling, 1984).

Overall, it appears that monensin increases digestibility slightly. However, other conditions such as level of feed intake, rumen fill, and ruminal fluid and particulate passage rates may be contributing factors (Schelling, 1984).

Protein Utilization

Monensin may reduce the requirement for dietary protein by reducing ruminal degradation of dietary protein. Monensin also decreases the rate of free amino acid degradation in the rumen fluid. Because proteolysis and deamination are reduced, rumen ammonia concentrations decrease (Chalupa, 1980).

While monensin reduces microbial yield in unadapted microbial cultures, microbial growth appears unaffected in adapted cultures. In adapted animals, decreased bacterial nitrogen reaches the abomasum but there is an increase in the escape of dietary protein. Monensin does exhibit a protein-sparing effect by making more effective use of amino nitrogen (Goodrich et al., 1984). Ricke et al. (1984) noted that monensin decreased rumen ammonia levels in sheep while lasalocid increased rumen ammonia pools.

VFA Concentrations and Properties

A consistent observation with ionophore feeding is a decrease in the acetate to propionate ratio in the rumen (Chalupa, 1980). Ruminal butyrate concentrations are usually but not always decreased with ionophores. Both lasalocid and monensin decreased the acetate:

propionate ratios at 6 and 12 hours post feeding in sheep (Ricke et al., 1984). With lasalocid, acetate, propionate, and total VFA pool size increased at 12 hours. From this study it appeared that lasalocid may not depress acetate and butyrate production to the same degree as monensin.

This shift in the VFA ratio increases energetic efficiency in several ways. First, production of propionate conserves more of the feed energy than does acetate production. Second, propionate in the past was suggested to be utilized by tissues more efficiently than acetate. This theory has been discounted by more recent research. Another possible advantage is that propionate is more flexible as an energy source than acetate (Schelling, 1984).

Monensin and lasalocid slightly increase lactate concentrations, but do not change rumen fluid pH when cattle are not stressed with a carbohydrate diet. Under carbohydrate stress studies, both in vivo and in vitro, these ionophores reduce lactate concentration and prevent pH drops. Both monensin and lasalocid aid in the prevention of lactic acidosis (Nagaraja et al., 1981), with lasalocid being more potent than monensin.

These changes are partly a result of selective alteration of rumen microflora (Elsasser, 1984). Protozoal populations are decreased in some but not all studies (Chalupa, 1980). Monensin decreased protozoal numbers by 4 to 63% in cattle and sheep under pasture and several different feedlot conditions (Schelling, 1984). Monensin metabolically inhibits hydrogen-producing and formate-producing bacteria. This increases the competitiveness of succinate-producing and propionate-producing bacteria. Monensin inhibited growth of four

strains of lactic acid-producing rumen bacteria but did not inhibit three strains of bacteria which metabolize lactate to propionate.

Reduced Feed Intake

Lasalocid and monensin improve feed efficiency in ruminants by decreasing feed intake and either maintaining or improving rate of gain (Bergen and Bates, 1984). Feed intake depressions range from 16% when monensin is first fed to 5% after 112 days on feed. Realistic feed intake depressions are about 5 to 6% with cattle fed high concentrate diets. Feed intake depressions with high grain diets are much greater than with high roughage diets. With roughage diets, intake depressions average about 3% with monensin (Schelling, 1984). Performance data on 16,000 head (Goodrich et al., 1984) revealed that cattle fed monensin-containing diets gained 1.6% faster, consumed 6.4% less feed, and required 7.5% less feed per 100 kg gain than cattle fed control diets.

Change in Gas Production

Monensin depressed methane production by rumen microbes, but inhibition is only partial (Schelling, 1984). In vitro and in vivo reductions of 4 to 31% have been recorded. While other chemical agents (i.e. amicloral) also decrease methane production, they may increase hydrogen gas production. Monensin does not increase hydrogen gas production (Chalupa, 1980). At low levels, monensin does not affect CO₂ production, but at high levels monensin depresses production of CO₂ (Schelling, 1984).

Modification of Rumen Fill and Rate of Passage

It is impossible to separate the effects of rumen fill and rate of passage. Therefore, both will be considered together as both of these parameters alter extent and site of digestion.

As liquid turnover rate increases, microbial fermentation favors increased acetate and methane production and increased growth efficiency of the rumen microbes (Bergen and Bates, 1984). Harrison et al. (1975) contends these changes resulted in decreased fermentation efficiency. Ricke et al. (1984) noted a reduction in both the liquid and solids dilution rates of sheep fed either a monensin or lasalocid-containing diet. Ruminant fluid volume was not affected by either additive. The overall responses to monensin and lasalocid might possibly be ascribed to effects of ionophores on liquid and particulate turnover.

Other Modes of Action

Bovine pulmonary edema and emphysema are produced by intraruminal degradation of tryptophan to 3-methylindole. Monensin inhibits this reaction and minimizes lung lesions (Schelling, 1984).

Monensin increases the production of propionate and thereby influences the precursors used for gluconeogenesis and glucose turnover. Monensin may increase body glucose turnover by 14%. Since propionate serves as the primary substrate for gluconeogenesis, the use of amino acids for this process is minimized (Schelling, 1984). Small increases in blood glucose (2-5 mg/100 ml), plasma insulin (1-2 micro units/ml), and blood urea (1-5 mg/100 ml) have been reported in response to monensin. Similar concentrations of plasma non-essential amino acids in conjunction with a decrease in levels of essential amino acids may

reflect improved utilization of essential amino acids for protein synthesis (Chalupa, 1980).

Other variable responses to monensin exist. Monensin has been reported to stimulate earlier puberty in heifers via the increased release of luteinizing hormone. Feeding monensin reduces face fly and horn fly numbers. Face fly and horn fly pupae in cattle feces were reduced by 20 and 23% respectively. Surviving pupae were smaller and exhibited decreased egg production (Schelling, 1984).

Potassium and Ionophores

In Vitro Ruminal Fermentation and Patterns

Ionophores selectively inhibit certain rumen bacteria or metabolic reactions of bacteria. This ameliorates growth of organisms which produce succinate and propionate while inhibiting organisms producing formate, hydrogen, lactate, acetate, and methane. These changes could account for the alterations in ruminal fermentations observed when ionophores are fed and result in more efficient rumen fermentation and growth of the animal (Dawson and Boling, 1984a).

The concept of altered microbial strains was prevalent until Dawson and Boling (1983a) discovered that the metabolic changes in the rumen were not associated with increased proportions of monensin-resistant bacteria. Selection of monensin-resistant microbial groups within the rumen could not account for changes in ruminal fermentations or the growth-promoting activity of these antibiotics.

In later research, Dawson and Boling (1984a) suggested that the ability of these organisms to concentrate K cations within the cell was an important factor in determining their susceptibility to ionophores.

All strains of *B. ruminicola* tested were sensitive to monensin and lasalocid in media containing low concentrations of K (50.8 ppm) but tended to be resistant to higher concentrations of ionophores when K levels were higher (481 ppm). Sodium concentrations ranged from 276 ppm in the high K medium to 3082 ppm in the low K medium. Dawson and Boling (1983b) observed similar effects of minerals on the activities of monensin and lasalocid against strains of *Ruminococcus albus*, *Ruminococcus flavefacians*, *Butyrivibrio fibrisolvens*, and *Bacteroides succinogenes*. As much as 32 times more ionophore was required to inhibit bacterial growth in media containing high K concentrations. Increases in the K concentration in media increased cell yield and decreased lag times in cultures grown in the presence of monensin and lasalocid. Growth responses suggested that bacteria had higher K requirements in the presence of the ionophores.

This K effect is consistent with the model for ionophore activity. Ionophores deplete intracellular K concentrations of microorganisms. Increased K concentrations decrease the antimicrobial and selective activities of monensin and lasalocid against rumen bacteria and indicate that K plays a key role in the antimicrobial activities of the ionophores (Dawson and Boling, 1984b). However, depletion of cellular K by itself cannot account for antimicrobial activities, because resistant strains continued to grow even when monensin depleted the K levels (Dawson and Boling, 1983b).

Dawson et al. (1983) revealed that all gram-positive organisms were susceptible to lasalocid at concentrations of 2.5 ppm or greater, and Na and K concentrations did not alter the antimicrobial activity of lasalocid against gram positive bacteria. However, the antimicrobial

activity of lasalocid against the gram-negative bacteria was related to the cation concentrations of the medium. Increased K concentrations tended to decrease the antimicrobial activity of lasalocid while increased Na concentrations amplified antimicrobial activity. The antimicrobial activity of lasalocid may be influenced by both Na and K concentrations in the rumen.

Salsbury and Romatowski (1978) noted that at 11,691 ppm, NaCl enhanced the change in the molar proportion of propionate shown by 15 ppm monensin. KCl (1864 or 4100 ppm) also increased the amount of propionate formed in the presence of monensin, but this effect appeared to be related to an increase in total VFA's, not to a change in the molar proportion of these acids. In the absence of monensin, NaCl (11,691 ppm) increased the amount and proportion of propionate, but KCl (1864 or 4100 ppm) had no effect. This may indicate VFA production is dependent on the Na and monensin concentrations. Potassium appears to alter the action of monensin.

In Vivo Trials

In vitro studies have suggested ionophores affect ruminal K levels. Lemenager et al. (1978) wintered cows on native range with either a urea-grain, 30% soybean meal, or 15% soybean meal based supplement with or without 200 mg monensin/head/day. When averaged over protein source, monensin increased ruminal K levels by 21.6%, but did not alter ruminal nitrogen, ammonia, non-ammonia nitrogen, Na, or total VFA concentrations. Propionate levels were increased with corresponding decreases in acetate and butyrate.

In contrast, Starnes et al. (1983) found that steers fed monensin

(33 ppm) or lasalocid (33 ppm) had lower concentrations of soluble K and calcium in the rumen fluid but had slightly higher soluble concentrations of copper. Both ionophores increased the apparent absorption of Na. Serum concentrations of the macro-minerals were not affected by treatment. Edlin et al. (1984), however, noted lower serum K levels with diets containing monensin or lasalocid.

Research of Spears and Harvey (1985) revealed that ionophores increased blood K levels. Steers on a control diet had lower K concentrations in the red blood cells than steers receiving 33 ppm lasalocid with similar level of K [.5% (5000 ppm)] and NaCl [.6% (6000 ppm)]. Increasing NaCl from .1 (1000 ppm) to .6% (6000 ppm) in the presence of lasalocid increased the molar proportion of ruminal acetate and reduced propionate at 90 days. Increasing dietary K from .5 (5000 ppm) to 1.5% (15,000 ppm) in the presence of lasalocid reduced Na and increased the soluble K concentration in the rumen fluid.

Although in vitro research suggests that ionophores may alter mineral metabolism, current in vivo research indicates that the K maintenance requirement of growing steers is not altered by feeding monensin at 200 mg/day (Kelley and Preston, 1985). However, monensin increased metabolic fecal K, decreased endogenous urinary K, and markedly increased the daily K balance relative to the K intake.

While in vitro studies have indicated that diets containing both ionophores and supplemental K alter the total volatile fatty acid levels and proportions, not all in vivo studies support this theory. Ferrell et al. (1983) conducted a large scale feedlot trial to determine the optimum level of K supplementation [.43 (4300 ppm) to 1.0% (10,000 ppm)] in diets containing either monensin or lasalocid at 30 g/ton. Potassium

supplementation increased gains the first 21 days, but feed intake and rate of gain were reduced the remainder of the trial. Ruminal, fecal, and carcass characteristics, except for carcass weight, were unaffected by treatment. Potassium levels of .7 (7000 ppm) to .85% (8500 ppm) tended to reduce carcass weights via reduced feed intake. With higher K levels, rumination was less frequent.

In a replicated 3 x 3 latin square, Starnes et al. (1984) noted that the ad lib feed intake (and hence, mineral intake) of growing steers was not affected by including either 33 ppm lasalocid or monensin. Both ionophores increased the apparent absorption of Na, magnesium, and phosphorus, but K and calcium absorption were unaffected. Serum concentrations of the macro-minerals were similar for all treatments. Steers fed either ionophore had lower K, magnesium, and calcium concentrations in the rumen fluid. Both ionophores decreased rumen osmolality as might be expected with the decrease in soluble mineral concentrations.

While it can be concluded that ionophores alter the metabolism of certain minerals in growing steers fed high energy diets, further research is warranted to disclose the mechanism of the ionophore-mineral interactions on site and extent of digestion, rate of passage, and rumen volume.

SUMMARY OF IONOPHORE EFFECTS ON METABOLISM

Measurement	Ionophore		
	Monensin	Lasalocid	Salinomycin
Minerals bound	Na>K	K>Na	Ca>Na,K
Digestibility:			
ADF	o, +		
Starch	+	+	+
N	+		
Ruminal:			
Proteolysis	-		
Deamination	-		
Microbial efficiency	-		
VFA ratios:			
Acetate	-	-	
Propionate	+	+	
Butyrate	-	o	
Total		+	
Lactate	-	-	
Ruminal pH	+		
Intake	-	o	+?
Methane	-		
Rate of passage:			
Solids	-	-	
Liquids	-	-	
Pulmonary edema	-		
Potassium:			
Ruminal	+,-,-	-, -	
Blood	o,-,+o	+,o	
Absorption	+,o	+,o	
Sodium:			
Ruminal	o		
Blood	o,-,+o	+,o	
Absorption	+	+	
Calcium:			
Ruminal	o,-	-, -	
Absorption	o	o	

CHAPTER III

INFLUENCE OF POTASSIUM LEVEL AND SOURCE ON THE PERFORMANCE AND BEHAVIOR OF FEEDLOT STEERS

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Summary

Three levels of K [.5% (basal), .65%, and .8% of dietary dry matter] with supplemental K from two sources (KCl and K_2CO_3) were fed to 120 steer calves for 113 days. Steers averaged 772 pounds at the start of the trial and were fed a 96% concentrate diet with 4% cottonseed hulls as a roughage. For the total trial, K supplementation increased live weight gain, average daily gain, feed consumption, and metabolizable energy intake. During the early part of the trial, K supplementation improved feed efficiency. Benefits of added K were only apparent during the first 57 days of the study.

Steers fed .8% K had heavier live weights at 113 days, gained more weight during the latter half of the trial, and had a greater total gain than steers fed .65% K. Feed efficiency was improved with .8% K, and metabolizable energy intake was increased.

Source of K had no consistent effect on performance. No treatment differences in the percent time eating, laying, ruminating, or in pH, dry matter, or starch content of the feces were noted. Frequencies of feeding, ruminating, and laying were correlated with animal performance,

but the mechanisms of the relationship and their control appears complex.

(Key Words: Feedlot, Potassium Carbonate, Potassium Chloride, Steers, Behavior)

Introduction

Adequacy of K in feedlot cattle diets was not considered a problem prior to the 1960's. The NRC (1970) stated the K requirements of beef cattle have not been critically measured. Recent recommendations indicate that the optimum level for growing and finishing steers is between .6 and .8% of ration dry matter. Most high forage rations provide this level of K. Grains usually contain less than .5% K, so K may be deficient in high-concentrate rations. Certain feedstuffs such as molasses and alfalfa are rich sources of K.

With the advent of higher concentrate levels in feedlot diets to increase feed efficiency, forages compose a smaller fraction of the diet. Because of cost and physical problems, less molasses is being used in diets than in the past. The introduction of least cost formulation rations and "synthetic" feedstuffs, such as nonprotein nitrogen, requires the critical re-evaluation of mineral supplementation (International Minerals and Chemical Corporation, Technical Services Department, 1981).

Dietary K requirements for feedlot cattle have been investigated intensively during the last twenty years, and dietary K recommendations have ranged from .43% to 1.5% diet dry matter (Algeo et al., 1965; Phillips and McLaren, 1981). Currently, NRC (1984) recommends a range in the K requirement of .5 to .7% of diet dry matter, with a suggested value of .65%. However, the NRC requirements do not consider other factors such as variability of ingredient nutrients, animal performance

potential, energy level of the feed, ambient temperatures, or stress. Diets might be specially formulated to account for such variables (International Minerals and Chemical Corporation, Technical Services Department, 1981).

Not only is the K level critical, but the K source may be important, influencing feed intake, efficiency of feed conversion, or average daily gain. Potassium source may alter digestive physiology through changes in rumen buffering or rate of passage (Beede et al., 1983), carcass characteristics (Algeo et al., 1965), and animal health (Crookshank, 1966). Currently, the cost of potassium from potassium carbonate (K_2CO_3) is 7.6 times the cost of potassium chloride (KCl). The prices quoted August 1985 in Chemical Marketing Reporter were \$.10/lb K from KCl and \$.76/lb K from K_2CO_3 (Anonymous, 1985). Hence, most supplemental K today is provided as KCl. But the total amount of supplemental K added per metric ton of feedlot diet is about 3 lb so a switch to K_2CO_3 would increase cost by \$1.98/ton.

Objectives of this trial were to determine the effect of several levels and two sources (KCl and K_2CO_3) of K on the performance of finishing steers.

Materials and Methods

One-hundred twenty crossbred steer calves with an initial weight of 772 pounds were trucked 100 miles from Purcell, Oklahoma on July 17, 1983 for feeding at Stillwater. The calves were tagged, weighed, and processed at the Beef Cattle Range Center near Stillwater with vaccinations of IBR and four-way blackleg. Ralgro implants were administered. Steers were blocked by weight into 4 weight groups which

in turn were allotted within weight group to 20 pens with 6 steers per pen. The final 4% roughage diet (Table 1) was diluted with cottonseed hulls to a level of 40% roughage for 3 days, 30% roughage for 4 days, and 20% roughage for 4 days.

Five finishing diets were fed (Table 1). The control diet contained no supplemental K but by calculation contained .5% K. The other four diets contained .65% or .8% K from supplemental KCl or K_2CO_3 . All supplements were fed in a pelleted form.

The steers were weighed initially and subsequently on days 28, 57, 85 and 113. Weight gains were calculated using a 5% pencil shrink. On day 135, the steers were trucked to Dodge City, Kansas where mean live weights and carcass weights for each treatment were obtained. Dressing percentages were based on a 2% pencil shrink of the loaded weights at Dodge City.

On day 68 the steers were observed every 30 minutes for 24 hours (1500 hr to 1450 hr) to monitor the time spent feeding, standing, laying, standing and ruminating, or laying and ruminating. At the end of the observation period on day 69, fecal grab samples were obtained from 2 to 6 steers/pen, and pH of the sample was recorded immediately. Fecal samples were analyzed for dry matter (DM; 105 C for 24 hours), ash (600 C for 12 hours), and starch (MacRae and Armstrong, 1968).

Treatment mean square errors for each of the variables measured were analyzed using a general linear model program. The model was a randomized block with blocking by initial weight. Orthogonal comparisons evaluated effects of K supplementation, K source, K level, and the interaction of K source and level. When the probability of the treatment mean square error was under 5% for a particular variable,

treatment means were compared by a Duncan's multiple range test (Steel and Torrie, 1980). Weight group means were similarly analyzed using a general linear model program with blocking by diet with one degree of freedom each to test for linear, quadratic, and cubic effects of weight. When weight group effects were significant ($P < .05$), weight group means were compared by a Duncan's multiple range test.

Results and Discussion

Steers receiving either level of supplemental K (Table 2) had heavier live weights at the conclusion of each feeding period ($P < .05$) than steers fed the basal (.5% K) ration. This agrees with results of previous studies (Roberts and St. Omer, 1965; Devlin et al., 1969; Standish et al., 1974; and Zinn et al., 1982a) in which K levels above .5% increased live weight gain. With basal diets at 1.0 and .43% K, however, Zinn et al. (1983) and Ferrell et al. (1983) found little or no animal responses to K supplementation.

Potassium supplementation had its greatest impact on live weight gain during the first 57 days of the trial (Table 2). Supplementation increased mean shrunk weight by 35 pounds during the first 28 days. This is similar to the consistent benefits from K supplementation for newly received cattle reported by Hutcheson (1980). Benefits may be due to increased water retention. Dehydration during shipment is an important contributor to "stress", and severe dehydration can limit the efficiency of the immune system (Hutcheson et al., 1984). Short term responses to K were theorized to be associated with increased fluid retention in the gut or tissues (Zinn et al., 1982a). However, Brethour and Duitsman (1972) found no difference in dressing percentages between

steers on a basal diet and steers receiving 100 g K_2CO_3 per day the final 3 days before slaughter. Thus, short term responses to K may not be associated with increased fluid retention alone.

The advantage in live weight at 28 days was at least partially retained from days 58 to 113, especially for steers fed .8% K. Steers fed the .8% K diet had heavier live weights at 113 days than steers fed the .65% K diet ($P < .05$). Devlin et al. (1969) reported daily gain of cattle fed .52% K was greater than for cattle fed .77% K diets. However, Zinn et al. (1980, 1982a) found benefits from K up to 1% of DM, and Zinn and Axe (1983) noted steers receiving 1% dietary K tended to outgain those receiving .65% K by about 5%.

Average daily gain for the total feeding period (113 days) was increased by 17.3% with added K ($P < .05$), with most of the difference occurring during the first 28 days. Hutcheson (1980) and Zinn et al. (1982b) also found that added K was most beneficial during the early portion of the feeding trials. Maintaining this early advantage in gain does not match with the results of Ferrell et al. (1983), in which KCl supplementation tended to increase rate of gain early in the feeding trial, but later reduced both feed intake and gain.

During the second half of the trial, .8% K supplementation produced significantly greater gains than the .65% K level. For the total trial, gain of steers fed .8% K surpassed the gain of steers fed .65% K ($P < .05$) due to performance between days 58 and 113. Devlin et al. (1969) noted increased gains for steers fed greater than .62% K, but less than .72% K, while Zinn and Owens (1980) found daily gain and feed efficiencies were favored with dietary K levels of 1.18% over a basal of .66% K.

Daily feed consumption (Table 3) was increased ($P < .05$) with K

supplementation by 14.1%. Intake was increased both during the first (15.1%) and second half (13.1%) of the trial. Dennis et al. (1976), Erdman et al. (1980), and Beede et al. (1983) have reported that K supplementation at levels greater than .52% of dry matter stimulated feed intake by dairy cows. This conflicts with earlier findings of Zinn et al. (1982b) and Ferrell et al. (1983) in which 1% K (1.22% KCl) and .7 to .85% K (.48 to .76% KCl) reduced feed intake.

Steers fed K_2CO_3 consumed 1.4% more feed than the steers fed KCl over the total trial, due mainly to greater intake (3.3%) during the first half of the trial. Between days 29 and 57, steers fed K_2CO_3 consumed 6.6% more ($P < .05$) feed than steers fed KCl. This increase in feed intake from day 29 to 57 could be a transient effect of the carbonate cation acting as a buffer or by increased osmotic pressure which elevated the rate of passage of digesta through the rumen. Calhoun and Shelton (1982) found $KHCO_3$ increased feed intake during the first 28 days of a lamb feeding trial, but not during the remainder of the trial. They suggested that this might be due to the buffering effect of HCO_3^- .

Potassium supplementation improved efficiency of feed use by 16% during the first 57 days ($P < .05$), but tended to have a negative effect (-10.6%) during the second half of the trial. The overall advantage of K supplementation was 2.8%. Potassium supplementation at the .8% level significantly ($P < .05$) improved feed efficiency during the latter half of the trial and over the total trial. Algeo et al. (1965) observed that feed efficiency was improved with K supplementation of a corn ration. Zinn and Owens (1980) reported 1% supplemental KCl improved daily gain and feed efficiency, and Zinn et al. (1982b) found 1% K increased feed

efficiency 21% in a short (28-day) study. Later Zinn et al. (1982a) and Zinn and Axe (1983) discovered gains and efficiencies were increased by elevating dietary K from .48% and .65%K to 1% of diet DM.

Calculated from performance and intake data, the metabolizable energy content of the diet was not increased with K supplementation. This indicates that the gain and feed efficiency advantages from K supplementation over the control diet were due to increased feed intake. Yet .8% K diets tended to have greater calculated metabolizable energy contents than the .65% K diets. No explanation for this quadratic effect is apparent. Metabolizable energy intake was significantly greater with added K, and the metabolizable energy intake was greater ($P<.05$) with .8% K than .65% K. This conflicts with results of Ferrell et al. (1983) who reported that metabolizable energy intake was decreased at levels of .7% to .8% K, and that metabolizable energy intake was greatest with a .43% K diet.

The dressing percentage (Table 4) calculated from hot carcass weights and a 2% pencil shrink of the live weights averaged 63.8%. No change in dressing percentage with elevated dietary K was observed. This agrees with reports of Roberts and St. Omer (1965), Algeo et al. (1965), and Zinn and Axe (1983). Zinn et al. (1982a) found that the dressing percentage tended to be lower for steers fed a 1% K ration.

The bicarbonate cation has been suggested to produce a fatter carcass than the chloride ion (Algeo et al., 1965). If true, carcass dressing percentage should increase with KHCO_3 or carbonate supplementation. In this study, carcass dressing percentage was unaffected by K source. Studies of Calhoun and Shelton (1982), Algeo et al. (1965), and Kelly and Preston (1984) noted no significant

differences in the dressing percentage of lambs or steers fed KHCO_3 versus those fed KCl .

No significant differences in the percent of total time spent eating, laying, or ruminating (Table 5) were detected. However, cattle receiving supplemental K spent 34% more time eating and 5.8% more time ruminating than cattle fed the basal K diet. Zinn et al. (1982) postulated that if steers eat more slowly and frequently when K is added to the diet, they may chew their food more thoroughly when eating which would reduce the need for later rumination of corn particles. Ferrell et al. (1983) noticed animal behavior to be altered slightly by K level. They found eating and laying times were not altered, but rumination was reduced by almost half with a dietary K level of 1%.

There were no significant differences in fecal pH or percentage fecal dry matter, ash, or starch (Table 5). This agrees with Ferrell et al. (1983) who reported differences in the composition of feces were nonsignificant at K levels of .43 to 1.00% and Zinn et al. (1982a) who observed no differences in fecal pH between cattle fed .65% or those fed 1.00% dietary K diets.

Results of this trial indicate that for steers under 950 pounds or fed for less than two months, gain and efficiency can be increased by feeding more than .5% K. This response appeared to be associated with increased feed intake. Advantages in live weight gains beyond two months were due mainly to greater feed intake but the early advantage in gain and feed intake was maintained. Feed efficiency over the latter half and total trial was not increased with K supplementation. Whether withdrawal of supplemental K at 56 days would reduce feed intake or feed efficiency remains to be determined.

Starting weight may influence the performance of feedlot steers (Gill et al., 1981; Owens and Gill, 1982). In this trial, the steers were blocked by weight group and performance was analyzed by weight group. The subsequent discussion will be based on change in performance per 45 kg increase in initial weight.

Rate of gain (Table 6) decreased linearly, quadratically, and cubically as initial weight increased. Average daily gain was reduced by 16.1% ($P < .05$) the first two months of the trial and by 12.7% over the total trial ($P < .05$). Heavier steers tend to have lower rates of gain as they may be reaching maturity earlier in the trial and depositing more fat. Deposition of live weight as fat requires more energy than as lean tissue plus water.

Gill et al. (1981) noted that average daily gain increased 7.7% as initial weight increased for steers of relatively small weight (230 to 270 kg). Such animals may gain more rapidly than the 340 to 360 kg steers in our trial. In a trial utilizing steers weighing 290 to 325 kg by Owens and Gill (1982), average daily gain was reduced .4% with heavier steers, but in a later trial (240 to 375 lb steers), gain was increased 1.6%. No explanation for this difference was mentioned though differences in final weights may be involved.

Feed intake did not increase ($P > .05$) with increasing weight. In two trials by Owens and Gill (1982), feed intake was greater for the heavier set of steers in both trials (4.3 and 5.6%), and Gill et al. (1981) observed that feed intake increased by 12.6% for every increase in steer weight of 45 kg. In the present trial, daily feed intake (Table 7) also tended to increase with increasing animal weight though the difference was small (1.4% the first half, 4.0% the last half, and

2.7% over the total trial). The lower rate of gain per 45 kg increase in initial weight of the heavier animals reflects the fact that heavier animals were consuming less feed above maintenance or the fat content of their gain was increased.

Efficiency of feed conversion was 22.4% poorer early in the trial and 18.6% poorer over the total feeding period for steers weighing 380 than 323 kg ($P < .05$). Gill et al. (1981) also reported poorer feed efficiencies (3.5%) with heavier animals. In two other trials, Owens and Gill (1982) observed 4.6% poorer and .6% better efficiency for heavier cattle. Efficiency of conversion of feed to live weight gain is depressed when feed intake above maintenance is low or when tissue gain is higher in fat and lower in protein and water. Metabolizable energy content of the feed (estimated from the net energy equations) suggests the heavier animals are depositing more fat. Also, digestibility may be declining as steers increase in feed intake or weight.

The amounts of time spent ruminating and eating were similar ($P > .05$) among weight groups. The heaviest steers spent 11% more time laying than the other three weight groups ($P < .05$). Compared with the lightest group, the heaviest steers spent slightly more time eating which matches their slightly greater feed intake. The time spent ruminating was similar across weight groups. With increased intake and no change in rumination, heaviest steers may have had a faster rate of passage, or less mastication of their feed which could reduce digestibility.

Fecal dry matter, starch, ash, and pH (Table 8) did not vary ($P > .05$) among the weight groups though the percentage dry matter and starch in the feces tended to be greater with heavier steers. This

trend in digestibility could partially explain why the metabolizable energy of the diet was reduced with the heaviest weight group.

Animal behavior of this trial was also summarized across all steers to examine the diurnal patterns of eating, ruminating and laying. Time spent ruminating included ruminating in either the standing or laying position.

Between 2200 and 700 h, less than 2.5% of the steers were eating (Figure 1). Eating oscillated during the rest of the day, with between .8 and 9.3% of steers eating until evening. Eating peaked from 1850 to 2100 h with 23.7% eating at 2000 h. Since all pens had self-feeders, feed freshness and external cues (feed trucks) did not initiate eating. Eating in the evening may be more prevalent when daytime temperatures are highest. On the measurement days, temperature peaked at 74 and 75 F, respectively. The peak at 800 h may reflect sunrise which occurred at 7:19 a.m.

Ruminating incidence (Figure 2) tended to be the inverse of feeding. Between 2200 to 600 h, 8.5 to 24.6% of the steers were ruminating. Lowest rumination incidence times were from 0650 to 0900 and from 1900 to 2250.

Laying time (Figure 3) also varied inversely to the feeding time. From 2200 to 700 h, most steers were laying down. However, during the day (950 to 1500 h) 50 to 80% of the steers were laying down which may reflect the warm temperature. From 1500 to 2150 h, as temperature decreased, the steers spent less time laying and were more active.

Eating and ruminating behavior may alter performance and digestion (Owens and Ferrell, 1983). In this trial, performance and fecal measurements were regressed against the frequency of eating, ruminating,

and laying to examine these relationships.

Eating behavior of the steers is presented in Table 9. During the 24-hour observation, 80% of the steers spent between 2 and 17% of their time eating. During the 48 observations at half hour intervals, 48 steers (20%) of the steers were never observed to be eating. This may be due to chance with steers eating short meals between observation times, timidity of animals so that presence of an observer inhibited eating behavior, or very infrequent meals. In performance trials, certain bulls consumed large meals on alternate days (Rich, T.D., personal communication) so certain steers may not have consumed feed during the observation period. As eating time increased up to 13% of total time, live weight and average daily gain for the total trial tended to increase. Average daily gain of the steers spending over 10% of their time eating, however, tended to be reduced as compared to steers eating from 2 to 8% of their time. Very frequent meals may reflect sorting their feed and consumption of more forage and less grain. Fecal pH and fecal starch tended to decline with more frequent eating whereas fecal ash content increased ($P < .01$). This could reflect higher digestibility or more selection of forage in the diet. As the steers spent more time eating, the time spent laying also increased ($P < .03$).

During the 48 observations, all 118 steers were observed to be ruminating one or more times (Table 10). Even though the ration contained 87% rolled corn plus only 4% cottonseed hulls, this provided sufficient abrasiveness to stimulate rumination. Animals with higher live weights had an increased frequency of rumination ($P < .02$). Rate of weight gain also increased ($P < .02$) with rumination frequency. Owens and Ferrell (1983) reported that steers ruminating more frequently had

greater daily gains ($P < .05$). In our trial, average daily gain appeared to plateau once rumination exceeded 8% of the time. This disagrees with Owens and Ferrell (1983) who noted that gain plateaued and fecal dry matter and starch tended to be increased at higher frequencies of rumination. In this trial, fecal dry matter decreased ($P < .04$), and fecal starch tended to be reduced with increasing rumination ($P < .06$). More mastication should increase starch digestibility if particle size limits starch digestion. Steers spending more than 15% of the time ruminating tended to spend more time laying ($P < .15$).

Frequency of laying (Tables 11-A and 11-B) was positively correlated with live weight at 28, 57, 85 and 113 days ($P < .01$). Daily gain tended to increase as laying time increased up to 71% of the time ($P < .1$). Fecal pH tended to increase as the steers spent more time laying ($P < .07$). Time spent eating was maximum for steers spending 50% of the time laying ($P < .03$), but rumination tended to be maximum for steers spending 70% of the time laying ($P < .15$).

Results from this trial suggest that the frequencies of feeding, ruminating, and laying are correlated with animal performance, but the mechanisms of the relationship and their control appears complex. If one can coordinate eating, ruminating, and laying time, one might optimize total animal performance.

TABLE 1. COMPOSITION OF FINISHING DIETS
FED TO FEEDLOT STEERS

	Diet				
	.5% K ^g	.65% K (KCl) ^a	.65% K (K ₂ CO ₃) ^a	.80% K (KCl) ^a	.80% K (K ₂ CO ₃) ^a
	Percentage of Dry Matter				
Dry rolled corn (IFN 4-02-931)	87.87	87.57	87.60	87.27	87.33
Cottonseed hulls (IFN 1-01-599)	4.00	4.00	4.00	4.00	4.00
Supplement :					
Soybean meal (IFN 5-20-637)	4.15	4.15	4.15	4.15	4.15
Cottonseed meal (IFN 5-01-621)	2.00	2.00	2.00	2.00	2.00
Calcium carbonate (IFN 6-01-069)	1.00	1.00	1.00	1.00	1.00
Sodium chloride (IFN 6-04-152)	.30	.30	.30	.30	.30
Urea (IFN 5-05-070)	.40	.40	.40	.40	.40
Cane molasses (IFN 4-04-696)	.25	.25	.25	.25	.25
Trace mineral	.01	.01	.01	.01	.01
Vitamin A ^c	.01	.01	.01	.01	.01
Rumensin ^d	.03	.03	.03	.03	.03
Tylan ^e	.05	.05	.05	.05	.05
Potassium carbonate ^f	0	0	.27	0	.54
Potassium chloride (IFN 6-03-755) ^f	0	.30	0	.60	0

^aSource of supplemental K added to achieve the final percentage K in the total ration.

^bSupplement ingredients were mixed and pelleted before adding to the rest of the ration.

^cVitamin A = 30,000 USP/g.

^dRumensin = 60 g/lb.

^eTylan = 10 g/lb.

^fSupplemental K sources were added into the ration in lieu of dry rolled corn.

^gBasal composition calculated to provide:

96.85% NEm

62.85% NEg

11.68% Crude Protein

.42% Ca

.33% P

84.98% TDN

TABLE 2. WEIGHT GAINS OF FEEDLOT STEERS FED DIFFERENT LEVELS OF POTASSIUM FROM VARYING SOURCES

K Level, Percentage of DM Potassium Source	.5 -----	.65 K ₂ CO ₃	.80 K ₂ CO ₃	.65 KCl	.80 KCl	SE ^d
Live weights, lb:						
Initial	773 _b	772	773 _a	773 _a	770	1.17
28 days	845 _b	886 _a	876 _a	878 _a	880 _a	8.35
57 days	932 _b	978 _a	974 _a	964 _a	963 _a	7.95
85 days	1025 _c	1050 _{ab}	1057 _a	1033 _{bc}	1052 _{ab}	7.08
113 days	1074 _c	1114 _{ab}	1131 _a	1093 _{bc}	1130 _a	8.75
Daily gain, lb/day:						
0-28 days	1.07 _b	2.48 _a	2.13 _a	2.19 _a	2.34 _a	.29
29-57 days	2.90 _b	3.02	3.22	2.81	2.73	.21
0-57 days	1.96 _b	2.75 _a	2.68 _a	2.51 _a	2.54 _a	.13
58-85 days	3.14 _a	2.46 _{bc}	2.80 _{ab}	2.32 _c	3.01 _a	.14
86-113 days	1.69 _b	2.16 _{ab}	2.52 _a	2.06 _{ab}	2.64 _a	.23
58-113 days	2.41 _{bc}	2.31 _{bc}	2.66 _{ab}	2.19 _c	2.83 _a	.13
0-113 days	2.18 _c	2.53 _{ab}	2.67 _a	2.35 _{bc}	2.68 _a	.07

^{abc} Means in a row with different superscripts differ statistically (P<.05).

^d Standard error of the means.

TABLE 3. FEED CONSUMPTION OF FEEDLOT STEERS FED DIFFERENT LEVELS OF POTASSIUM FROM VARYING SOURCES

K Level, Percentage of DM Potassium Source	.5 -----	.65 K ₂ CO ₃	.80 K ₂ CO ₃	.65 KCl	.80 KCl	SE ^d
Daily feed, lb/day:						
0-28 days	18.52	20.87	20.90	20.97	20.85	.79
29-57 days	18.93 ^c	23.18 ^a	22.60 ^{ab}	21.55 ^b	21.41 ^b	.50
0-57 days	18.73 ^b	22.04 ^a	21.77 ^a	21.27 ^a	21.14 ^a	.58
58-85 days	20.22 ^b	23.19 ^a	22.90 ^a	22.10 ^{ab}	22.48 ^a	.66
86-113 days	18.54 ^b	20.28 ^{ab}	21.11 ^a	20.63 ^{ab}	22.64 ^a	.76
58-113 days	19.38 ^b	21.74 ^a	22.00 ^a	21.37 ^a	22.56 ^a	.64
0-113 days	19.05 ^b	21.89 ^a	21.88 ^a	21.32 ^a	21.84 ^a	.51
Feed conversion efficiency, feed/lb gain:						
0-57 days	9.90 ^a	8.23 ^b	8.18 ^b	8.50 ^b	8.35 ^b	.38
58-113 days	8.08 ^b	9.47 ^{ab}	8.34 ^{ab}	9.93 ^a	8.01 ^b	.50
0-113 days	8.80 ^{ab}	8.69 ^{abc}	8.26 ^{bc}	9.13 ^a	8.15 ^c	.19
Metabolizable energy content of feed, mcal/kg	2.63 ^a	2.58 ^{ab}	2.65 ^a	2.53 ^b	2.65 ^a	.03
Metabolizable energy intake, mcal/day	22.71 ^c	25.65 ^{ab}	26.27 ^a	24.53 ^b	26.29 ^a	.52

^{abc} Means in a row with different superscripts differ statistically (P<.05).

^d Standard error of the means.

TABLE 4. CARCASS DATA OF FEEDLOT STEERS FED DIFFERENT LEVELS OF POTASSIUM FROM VARYING SOURCES

K Level, Percentage of DM	.5	.65	.80	.65	.80	
Potassium Source	-----	K ₂ CO ₃	K ₂ CO ₃	KCl	KCl	SE ^b
Live weights, no shrink	1114	1140	1152	1116	1144	
Carcass weight	698	713	722	694	717	11.87
Dressing percentage ^a	63.9	63.8	63.9	63.4	64.0	

^aDressing percent is based on 2% pencil shrink of live weights.

^bStandard error of the means.

TABLE 5. ANIMAL BEHAVIOR AND FECAL MEASUREMENTS OF FEEDLOT STEERS
FED VARIOUS DIETARY POTASSIUM SOURCES AND LEVELS

K Level, Percentage of DM	.5	.65	.80	.65	.80	
Potassium Source	-----	K ₂ CO ₃	K ₂ CO ₃	KCl	KCl	SE ^a
Time spent, %:						
Eating	3.37	4.43	5.03	4.77	3.82	.91
Laying	51.39	53.47	52.60	55.64	55.99	1.44
Ruminating	12.27	12.93	12.59	12.24	14.15	1.16
Fecal measures:						
pH	6.04	6.09	6.07	6.21	6.11	.11
Dry matter, %	23.26	25.01	26.84	25.51	23.02	1.21
Ash, %	6.46	6.75	6.96	5.61	7.22	.72
Starch, %	19.96	22.32	21.47	29.18	23.90	3.62

^aStandard error of the means.

TABLE 6. WEIGHT GAINS BY INITIAL STEER WEIGHT GROUP

	Weight Group				SE	Linear Slope	Effect ^f
	1	2	3	4			
Live weight, lb.							
Initial	711 ^d	758 ^c	784 ^b	835 ^a	1.05	163	L,C
28 days	821 ^d	854 ^c	889 ^b	928 ^a	7.47	30.9	L
57 days	923 ^c	931 ^c	976 ^b	1019 ^a	7.11	-117	L,Q
85 days	1002 ^c	1012 ^c	1058 ^b	1101 ^a	6.33	-118	L,Q
113 days	1081 ^c	1075 ^c	1120 ^b	1159 ^a	7.82	-183	L,Q
Daily gain, lb/day							
0-28 days	2.46	1.89	2.15	1.66	.26	-4.8	
29-57 days	3.34	2.57	2.85	2.97	.19	-4.5	Q
0-57 days	2.91 ^a	2.21 ^b	2.51 ^b	2.33 ^b	.11	-4.9	L,Q,C
58-85 days	2.69	2.74	2.78	2.77	.12	-.04	
86-113 days	2.66	2.14	2.09	1.97	.21	-2.2	L
58-113 days	2.67	2.44 ^b	2.44 ^b	2.37 ^b	.11	-1.1	
0-113 days	2.79 ^a	2.32 ^b	2.47 ^b	2.35 ^b	.06	-3.1	L,Q,C

^{abcd} Means in a row with different superscripts differ statistically (P<.05).

^e Standard error of the means.

^f Effect notes linear (L), quadratic (Q), or cubic (C) effects of weight groups (P<.05).

TABLE 7. FEED CONSUMPTION BY STEERS OF DIFFERENT INITIAL WEIGHTS

	Weight Group				SE ^d	Linear Slope	Effect ^e
	1	2	3	4			
Daily feed, lb/day							
0-28 days	20.40	19.92	20.48	20.89	.71	-4.2	
29-57 days	21.70	20.65	21.85	21.95	.44	-10.6	
0-57 days	21.06	20.29	21.18	21.43	.52	-7.5	
58-85 days	22.14	20.76	22.83	22.97	.59	-16.4	
86-113 days	20.41	19.22	21.24	21.69	.68	-14.8	
58-113 days	21.28	19.99	22.04	22.33	.57	-15.6	
0-113 days	21.17	20.14	21.60	21.88	.46	-11.5	
Feed conversion efficiency, feed/lb gain							
0-57 days	7.31 ^b	9.41 ^a	8.46 ^a	9.34 ^a	.34	15.7	L,C
58-113 days	8.10	8.32 ^b	9.13 ^b	9.51	.45	-2.5	L
0-133 days	7.58 ^c	8.74 ^b	8.77 ^b	9.33 ^a	.17	5.9	L,C
Metabolizable energy content of feed, mcal/kg							
	2.68	2.59	2.59	2.58	.03	-.4	L
Metabolizable energy intake, mcal/day							
	25.72 ^a	23.70 ^b	25.39 ^a	25.55 ^a	.47	-17.2	Q,C

^{abc} Means in a row different superscripts differ statistically ($P < .05$).

^d Standard error of the means.

^e Effect notes linear (L), quadratic (Q), and cubic (C) effects of weight groups ($P < .05$).

TABLE 8. ANIMAL BEHAVIOR AND FECAL MEASUREMENTS OF STEERS BY INITIAL WEIGHTS

	Weight Group				SE ^d	Linear Slope	Effect ^e
	1	2	3	4			
Time spent, %:							
Eating	3.0	5.1	4.7	4.4	.8	10.8	
Ruminating	12.4 _b	12.7 _b	13.8 _b	12.4 _a	1.0	-7.2	
Laying	52.6 _b	51.8 _b	52.7 _b	58.2 _a	1.3	1.2	L,Q
Fecal measures:							
pH	5.89	6.24	6.13	6.16	.1	2.1	
Dry matter, %	24.9	24.4	23.6	26.0	1.1	6.7	
Ash, %	6.3	7.4	6.4	6.4	.6	10.4	
Starch, %	24.6	19.9	21.7	27.3	3.2	-19.6	

^{ab}Means in a row with different superscripts differ statistically (P<.05).

^cStandard error of the means.

^dEffect notes linear (L), quadratic (Q), or cubic (C) effect of weight groups (P<.05).

TABLE 9. PERFORMANCE OF STEERS VERSUS PERCENTAGE TIME SPENT EATING

Item	Percentage Time Spent Eating								Linear	
	0	2	4	6	8	10	13	17	Slope	Prob.
No. of steers	24	16	39	22	6	9	1	1		
Live weight, lb:										
Initial	753	765	778	787	781	770	785	752	2.2	.11
57 days	937	963	974	975	998	948	1010	950	2.7	.14
113 days	1078	1101	1127	1124	1152	1080	1160	1090	2.9	.20 ^a
Daily gain, lb/day:										
0-28 days	1.92	2.40	2.32	1.70	2.53	1.53	1.82	2.67	-.021	.59
0-57 days	2.40	2.63	2.58	2.44	2.94	2.29	3.06	2.64	.007	.78
58-113 days	2.39	2.34	2.61	2.53	2.60	2.24	2.54	2.38	.004	.87
0-113 days	2.39	2.49	2.60	2.49	2.77	2.27	2.81	2.51	.005	.77
Time spent, %:										
Ruminating	12.9	12.4	12.5	14.6	12.2	12.5	10.4	8.3	-.022	.88
Laying,	49.3	51.6	56.1	56.1	50.7	56.0	54.2	56.3	.562	.03
No. of Steers	15	4	25	16	3	4	0	1		
Fecal:										
pH	5.83	6.47	6.20	6.05	6.05	6.12	---	6.48	.023	.11
DM, %	24.9	21.8	25.3	24.7	24.2	22.3	---	23.8	-.091	.63
Ash, %	5.8	7.6	6.3	6.7	6.6	9.2	---	12.1	.254	.01
Starch, %	25.7	21.9	24.2	24.8	21.7	13.5	---	15.0	-.653	.17

^aDenotes quadratic effect (P<.05).

TABLE 10. PERFORMANCE OF STEERS VERSUS PERCENTAGE TIME SPENT RUMINATING

Item	Percentage Time Spent Ruminating													Linear	
	2	4	6	8	10	13	15	17	19	21	23	25	29	Slope	Prob.
No. of steers	3	5	9	10	19	22	19	10	11	4	3	2	1		
Live weight, lb:															
Initial	800	741	759	748	779	785	787	753	762	782	785	751	765	.4	.69
57 days	990	936	923	950	967	980	985	928	950	1020	1003	910	1030	1.4	.25
113 days	1087	1036	1061	1100	1121	1126	1133	1068	1117	1138	1173	1100	1230	3.4	.02
Daily gain, lb/day:															
0-28 days	2.40	2.24	2.07	2.88	1.65	1.78	2.48	1.64	1.85	2.68	2.94	1.34	3.21	-.001	.97
0-57 days	2.46	2.60	2.07	2.72	2.46	2.56	2.60	2.25	2.47	3.28	2.94	1.99	3.75	.017	.28
58-113 days	1.64	1.70	2.34	2.54	2.60	2.48	2.51	2.38	2.84	1.99	2.88	3.22	3.39	.035	.01
0-113 days	2.05	2.15	2.20	2.63	2.53	2.52	2.55	2.31	2.65	2.64	2.91	2.60	3.57	.026	.02
Time spent, %:															
Eating	6.3	3.8	3.5	4.6	4.7	4.5	4.2	3.1	4.0	4.7	3.5	6.3	6.3	-.009	.88
Laying	54.9	50.4	49.1	51.7	54.4	52.4	58.1	51.3	58.0	54.7	50.0	56.3	54.2	.244	.15
No. of steers	2	4	4	8	11	11	14	8	2	0	2	2	0		
Fecal:															
pH	5.94	6.02	6.36	6.32	6.08	6.04	6.07	5.95	6.50	----	6.19	5.6	----	-.011	.24
DM, %	30.6	27.2	26.4	24.1	25.1	24.8	23.6	23.4	23.5	----	22.3	23.9	----	-.241	.04
Ash, %	4.8	5.8	5.8	7.2	6.3	6.1	7.3	6.0	10.5	----	6.8	8.2	----	.101	.10
Starch, %	34.7	30.0	27.4	24.9	22.9	24.6	21.1	23.0	13.9	----	24.6	16.2	----	-.575	.06

TABLE 11-A. PERFORMANCE OF STEERS VERSUS PERCENTAGE TIME SPENT LAYING

Item	Percentage Time Spent Laying												
	25	33	35	38	40	42	44	46	48	50	52	54	56
No. of steers	1	3	1	4	2	3	8	6	4	2	14	17	13
Live wt, lb:													
Initial	784	782	724	763	729	779	753	758	775	806	766	766	771
57 d	900	937	930	963	1000	970	933	935	963	970	925	978	963
113 d	990	1033	1060	1128	1105	1123	1096	1110	1095	1050	1076	1133	1094
Daily gain, lb/day:													
0-28 d	.16	1.46	2.30	2.86	2.14	3.52	1.66	2.29	1.75	.41	1.61	2.36	1.81
0-57 d	1.25	1.89	2.80	2.66	3.89	2.51	2.34	2.29	2.44	2.04	1.97	2.86	2.53
58-113 d	1.53	1.64	2.21	2.80	1.78	2.60	2.78	2.97	2.25	1.36	2.56	2.62	2.22
0-113 d	1.38	1.76	2.50	2.73	2.84	2.55	2.55	2.63	2.35	1.70	2.26	2.75	2.38
Time spent, %:													
Eating	0	1.4	0	2.1	2.1	2.8	3.9	5.6	5.2	8.3	3.6	4.5	6.3
Ruminating	4.2	9.0	12.5	9.9	16.7	12.5	13.5	11.1	9.9	9.4	13.7	14.6	12.2
No. of steers	1	2	1	2	0	2	5	5	3	1	7	8	6
Fecal:													
pH	6.02	5.83	5.73	5.80	----	5.82	6.26	6.26	6.03	6.32	5.95	6.04	6.01
DM, %	28.0	25.4	27.7	27.3	----	24.4	23.4	27.1	26.9	13.7	21.8	27.5	25.3
Ash, %	4.9	5.8	5.1	7.2	----	7.4	5.9	6.2	4.5	9.9	7.8	4.8	6.7
Starch, %	39.3	19.3	27.6	30.2	----	16.5	24.3	24.2	24.4	8.0	18.6	32.0	21.8

TABLE 11-B. PERFORMANCE OF STEERS VERSUS PERCENTAGE TIME SPENT LAYING

Item	Percentage Time Spent Laying										Linear Slope	Prob.
	58	60	63	65	67	69	71	73	75	77		
No. of steers	12	5	4	8	4	3	1	1	1	1		
Live wt, lb:												
Initial	774	762	768	804	795	755	833	819	805	838	1.00	.04
57 d	967	958	963	1009	1000	1003	1080	980	1000	1030	1.89	.003
113 d	1111	1128	1093	1163	1133	1137	1290	1090	1180	1210	2.23	.004
Daily gain, lb/day:												
0-28 d	1.72	2.63	2.68	2.34	2.56	3.35	2.48	2.64	1.79	.95	.013	.32
0-57 d	2.52	2.59	2.57	2.71	2.71	3.48	3.39	1.96	2.54	2.46	.014	.09
58-113 d	2.45	2.88	2.21	2.61	2.25	2.26	3.56	1.87	3.05	3.05	.006	.46
0-113 d	2.49	2.74	2.39	2.66	2.48	2.87	3.47	1.92	2.80	2.76	.010	.10
Time spent, %:												
Eating	3.3	5.4	5.7	3.4	5.7	3.5	4.2	6.3	6.3	4.2	.068	.03
Ruminating	14.2	13.3	14.1	13.3	13.5	7.6	18.8	14.6	14.6	10.4	.073	.15
No. of steers	8	1	3	5	3	2	1	1	1	0		
Fecal:												
pH	6.03	5.79	6.26	6.34	6.18	6.53	6.14	6.23	6.24	----	.008	.07
DM, %	23.2	21.6	21.1	23.5	27.4	20.70	29.3	23.1	30.1	----	-.046	.44
Ash, %	6.6	7.0	7.7	8.9	5.2	8.6	8.5	9.4	3.4	----	.044	.14
Starch, %	21.7	19.0	14.6	19.9	39.4	18.8	24.4	9.3	44.9	----	-.032	.83

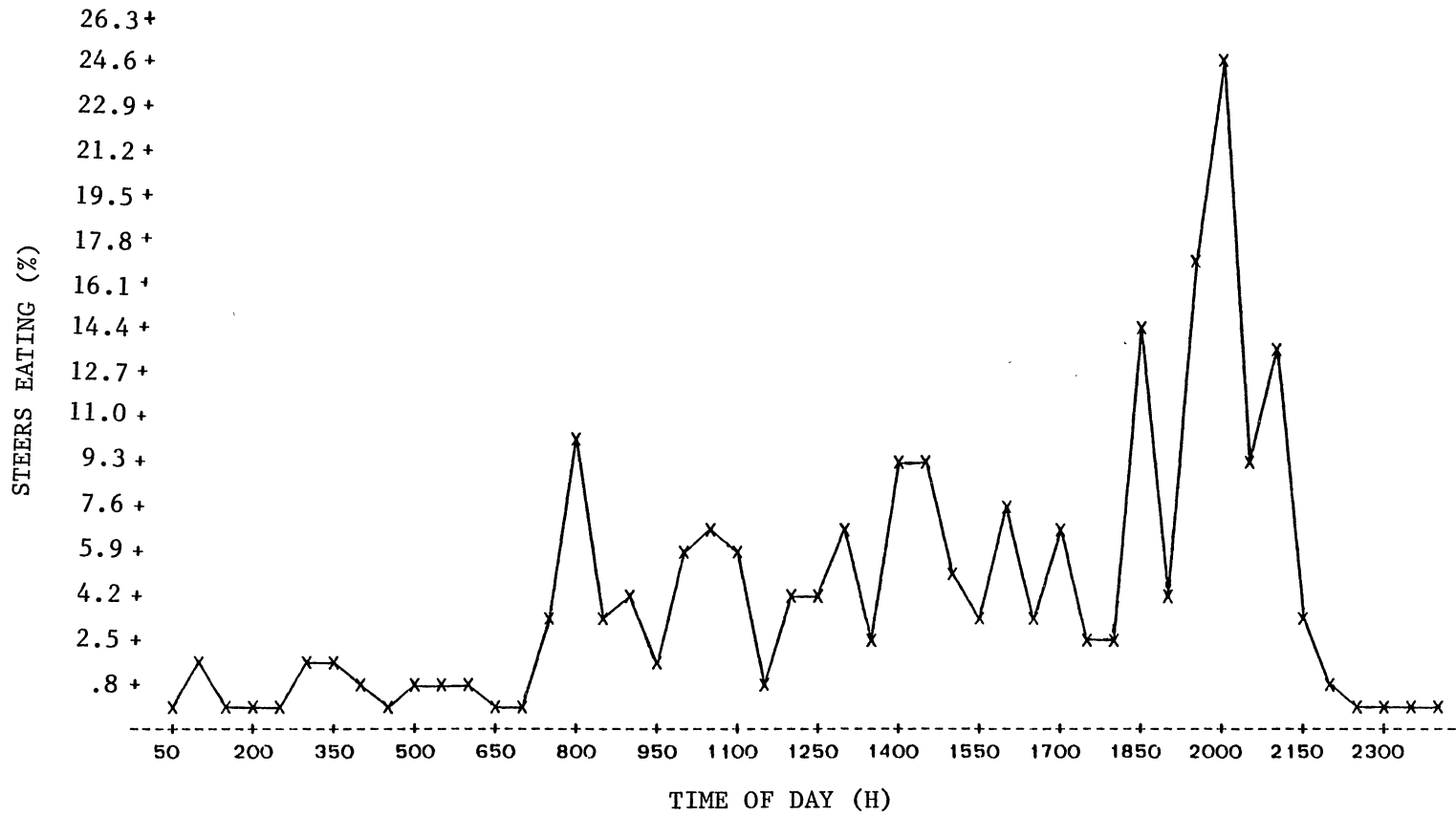


Figure 1. Eating Pattern

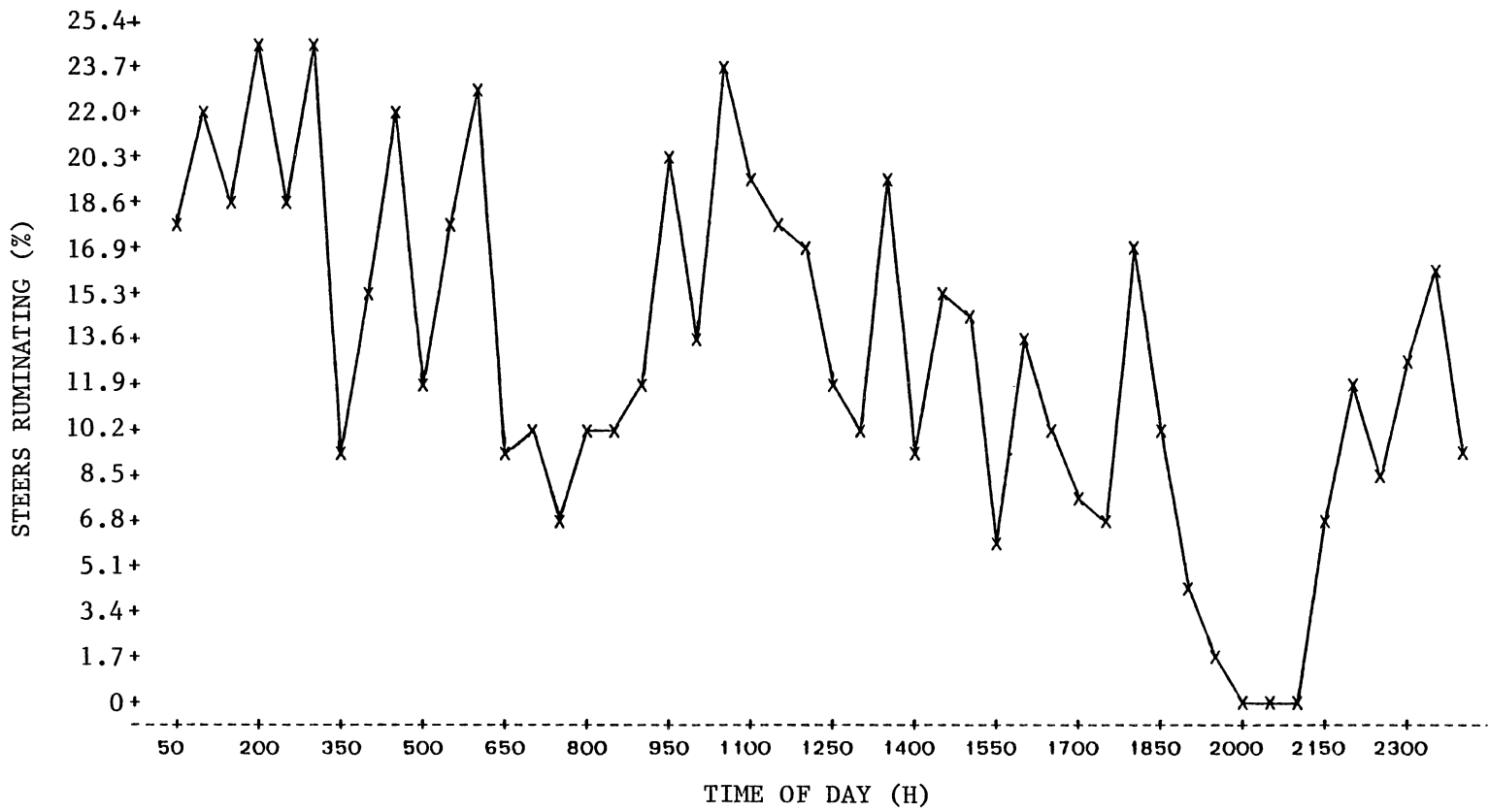


Figure 2. Ruminating Pattern

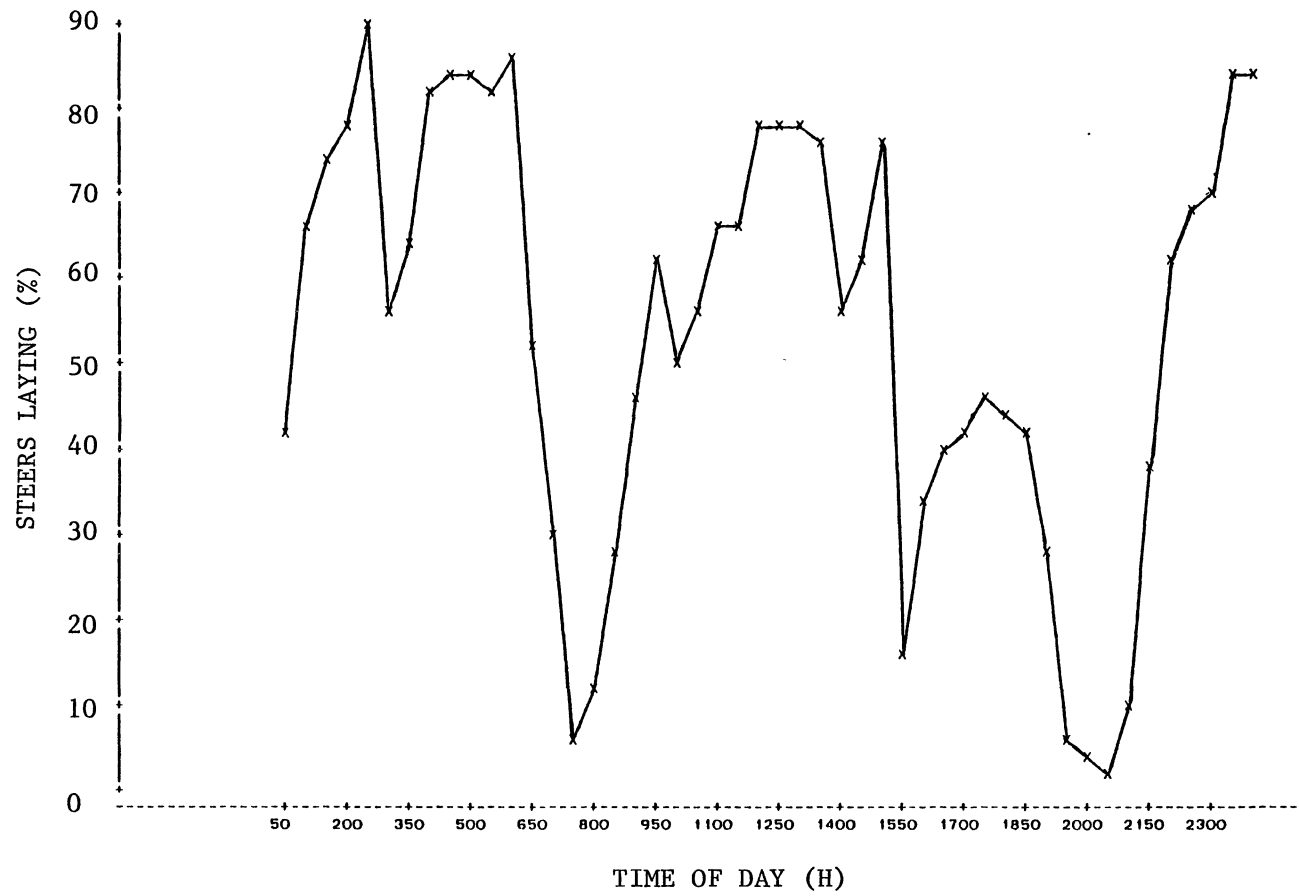


Figure 3. Laying Pattern

CHAPTER IV

INFLUENCE OF POTASSIUM LEVEL ON DIGESTIBILITY OF CONCENTRATE DIETS BY FEEDLOT STEERS

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Summary

Effects of potassium (K) level on site and extent of digestion and on ruminal passage rates were investigated with four cannulated steers fed a 90% rolled corn, 10% cottonseed hull diet. Four levels of K [.54 (control), .63, .75, and .85% of diet dry matter with supplemental K from KCl] were fed in a 4 X 4 latin square experiment. Ruminal ammonia-N was greatest with the .85% K diet. Ruminal organic matter, starch, and nitrogen digestibilities did not differ significantly, but tended to be greater for the .85% K diet (70.3, 83.1, and 54.82%) and lower for the .75% K diet (62.0, 76.6, and 42.2%). Post-ruminal digestibilities of organic matter and starch tended to be the greatest with the .75% K diet. Digestibility of ADF increased linearly with addition of K to the diet with the primary response being post-ruminal. Although fluid and particulate passage rates were not significantly affected by K level, the overall correlation of ruminal liquid (CoEDTA) with solids (Yb-labeled corn) passage rates was positive ($r=.46$; $P<.07$). Extent of ruminal starch digestion was inversely related to fluid dilution rate ($r=-.36$; $P<.18$) and to particulate passage rate ($r=-.65$;

P<.01). Microbial efficiency decreased as ruminal OM digestibility increased ($r=-.95$; $P<.0001$). Ruminal acid detergent fiber digestion was lowest and post-ruminal acid detergent fiber digestion greatest with the .75% K diet. From 3 to 10 g of K and 19 to 26 g of Na were recycled to the rumen daily. Post-ruminal absorption of K and Na were not affected by treatment and exceeded 93 and 97%, respectively. At lower dietary K levels, recycling of Na and K tended to be greater. Though the ratios of K to Na in the rumen, duodenum, and feces were not consistently altered by K intake, higher ratios of K to Na were associated with higher liquid and particulate passage rates. These ions may change rate of passage or rate of ADF digestion.

(Key Words: Feedlot, Potassium Chloride, Steers, Site of Digestion, Passage Rate)

Introduction

Early research (Roberts and St. Omer, 1965) indicated that K at .5 to .6% of the ration dry matter was optimum for rapid weight gains in finishing steers. Devlin et al. (1969) in two trials found weight gain and feed efficiency optimum with K at more than .62 but less than .72% of the ration dry matter. Previous trials at Oklahoma State University (Zinn and Owens, 1980; Zinn et al., 1982a; Zinn et al., 1982b) suggest that higher K supplementation levels may increase feedlot performance. Currently, the K recommendation for growing and finishing steers ranges from .5 to .7% of ration dry matter, with a suggested value of .65% K (NRC, 1984). Because most concentrate feeds fall below this percentage, K supplementation of high-concentrate rations is commonly practiced.

Limited research suggests K supplementation might benefit the animal in several ways. First, added K may increase the moisture

content of rumen fluid for bacterial fermentation (Parthasarathy and Phillipson, 1953; Ward, 1966). Potassium increases water intake and water turnover rate (Ward, 1966). Enhanced ruminal digestion, particularly of fiber, has been reported with elevated K levels (Hubert et al., 1958; Zinn et al., 1983). Since K in the diet may be converted to potassium bicarbonate in the rumen, K may buffer rumen contents and reduce the incidence of sub-acute and acute digestive disturbances (Devlin et al., 1969; Giduck et al., 1981; Zinn and Owens, 1980). Also, feed intake may be stabilized with K (Zinn et al., 1982b; Zinn et al., 1983).

The ratio of Na to K may be even more critical than K level alone (Bryant et al., 1959; Scott, 1966; Caldwell and Hudson, 1974). Durand and Kawashima (1980) concluded 500 to 1500 mg/l (500 to 1500 ppm) of available K and Na are required for optimal rumen fermentation. This is presumably for osmotic and ionic balance rather than meeting a nutrient requirement of microorganisms per se.

The objectives of this study were to examine the influence of various levels of KCl supplementation and differing ratios of K to Na on the site and extent of digestion and on ruminal passage rates in growing steers.

Materials and Methods

Four steers (115 kg) each fitted with a ruminal cannula and a t-cannula in the duodenum proximal to the bile duct were used in a 4 X 4 latin square design. Diets consisted of a control (C) diet with no supplemental K added which contained .54% K (Table 1). Potassium chloride replaced rolled corn in the test diets to produce test diets

which contained .63%, .75%, and .85% K on a dry matter basis. All diets contained added chromic oxide as an indigestible marker. Steers were fed twice daily at 0800 and 2000 h so that daily dry matter intake was equal to 2.25% of each steer's initial shrunk weight.

Periods lasted 14 days with sampling from the rumen, duodenum and rectum on days 13 and 14. At 2000 h on day 11, a rectal sample was obtained prior to feeding 100 g of ytterbium-labeled corn to estimate passage rate of particles through the rumen. A fluid marker (50 ml of CoEDTA containing 132 mg Co plus 75 ml distilled water) was intraruminally dosed prior to the morning feeding on day 13. Rumen samples were withdrawn via cannula on day 13 before dosing and 1, 3, 6, 9, and 12 h post-feeding. Ruminal pH was measured, the fluid was strained through four layers of cheesecloth, 1 ml 20% H₂SO₄ was added per 100 ml strained fluid, and the sample was frozen for later analysis.

Samples from the duodenum (250 ml) and rectum were obtained 39, 45, 51, 57, 66, 72, 78, and 84 h after feeding the ytterbium-labeled corn, and pH was measured immediately. Duodenal and a portion of the fecal samples from each animal within each period were composited on an equal wet volume basis and dried for 48 h in a 60 C oven. The remaining portion of each individual fecal sample was dried individually for Yb analysis. Feed samples were collected on days 11-14 and composited within each diet and period. All samples were ground in a Wiley mill fitted with a 2mm screen and stored at room temperature for future analysis.

Rumen samples were thawed, and 45 g were centrifuged at 10,000 g for 10 minutes. The supernatant fluid was reserved for analysis of

CoEDTA concentration by atomic absorption spectroscopy and for ammonia-N (Broderick and Kang, 1980). Feed, duodenal, and fecal samples were analyzed for dry matter (DM; 105 C for 24 h), ash (600 C for 12 h), Kjeldahl nitrogen (N; AOAC, 1975), starch (MacRae and Armstrong, 1968), acid detergent fiber (Goering and Van Soest, 1970), and chromium (Fenton and Fenton, 1979). Individual fecal samples were analyzed for ytterbium concentration (Hart and Polan, 1984) to determine particulate passage rate. Passage rate was estimated as the slope of the regression of the natural logarithm of Yb concentration in dry matter against sampling time. Duodenal samples were analyzed for ammonia-N by distillation over magnesium oxide (AOAC, 1975) and for nucleic acid-N (Zinn and Owens, 1982a).

Composited feed, duodenal, and fecal samples were analyzed for the concentrations of Na and K. All glassware, equipment and test tubes were washed and rinsed successively with distilled water, a 1:1 solution of nitric acid and double-deionized water, and with double-deionized water. One gram of dry sample or one gram centrifuged ruminal liquid was weighed into 100 ml glass beakers, and 20 ml of a 1:1 nitric acid solution or 10 g for ruminal liquid was added. Eight samples plus a blank beaker containing only the 1:1 nitric acid solution were then digested on a hot plate for five hours with nitric acid solution added as necessary to maintain volume. Digestion time was shortened to 2 h for ruminal liquid. Beakers were allowed to evaporate to 3-5 ml after 5 h and were then cooled.

Whatman filter paper (#1), 100 ml volumetric flasks, and the stoppers were pre-rinsed with 1:1 nitric acid solution to remove residual Na and K prior to filtering the samples. The contents of the

beakers were poured onto the filter paper, and the beakers were rinsed twice with double-deionized water to remove any residue. The filter paper was then rinsed twice with the double-deionized water. The volumetric flask was brought to volume and allowed to stand overnight. Two aliquots of each flask were removed and retained in 15 ml plastic test tubes which were stored at room temperature for later analysis.

Samples were diluted to be read by atomic absorption. To determine the amount of sodium chloride which needed to be added to eliminate atomic absorption interference, two feed, duodenal, fecal, and rumen samples were randomly selected and diluted with water containing 1000, 2000, or 3000 ppm Na (from NaCl) to achieve final Na concentrations of 500, 667 and 750 ppm. Readings were highest and had plateaued at 667 ppm (with the 2000 ppm sodium dilution solution). Hence, all samples to be analyzed for K were prepared by diluting with a 2000 ppm Na solution. The same solution was used to prepare the K standards (KCl) and blanks. The samples were analyzed for atomic absorbance, and the sample readings were regressed against the standard curve developed using KCl at 0, .5, 1.0, 1.5, 2.0, and 2.5 ppm K.

In a similar fashion, the amount of K to minimize Na interference was determined. Na standards (0, .2, .4, .6, .8, and 1.0 ppm) were prepared with a 1000, 2000, and 3000 ppm K solution present to achieve final K concentrations of 500, 667, and 750 ppm. One feed and one rumen sample was tested similarly to determine what level of K should be used to dilute the samples for reading Na. At a concentration of 667 ppm (with the 2000 ppm K solution) the sodium reading had plateaued. Hence, samples to be analyzed for Na were diluted in plastic test tubes with a solution containing 2000 ppm KCl. This solution also was used to

prepare the Na standards and the blank. All samples were analyzed by atomic absorbance, and the readings were regressed against the Na standard curve developed using NaCl at 0, .2, .4, .6, .8, and 1.0 ppm Na.

Treatment means were analyzed using a general linear model program for a 4 by 4 latin square. Classes included steer, period, and treatment which was divided to test linear, quadratic, and cubic effects of K. When the treatment effect was significant ($P < .05$) treatment means were compared by Duncan's new multiple range test (Steel and Torrie, 1980). Simple correlations among various factors were calculated by linear regression of the two factors.

Results and Discussion

Mean ruminal, duodenal, and fecal pH (Table 2) did not change significantly ($P > .05$) with increasing K supplementation levels which agrees with the results of Campbell et al. (1965). Devlin et al. (1969) and Giduck et al. (1981) found ruminal pH increased with increasing intake of K. Devlin et al. (1969) fed the carbonate ion of K and Giduck et al. (1981) used an unusually high K level. In this trial, ruminal and fecal pH in all treatments were higher than expected for a 90 percent ration. This may be related to the modest but limited feed intake which should reduce the incidence of subacute and acute digestive disturbances. Duodenal pH was about 2.3, similar to other reports in which similar concentrate levels were fed. Since neither ruminal nor duodenal pH was increased with added KCl, increased buffering capacity of ruminal contents from added K, as suggested by Zinn and Owens (1980), seems unlikely.

Ruminal ammonia-nitrogen (Table 2) was greater ($P < .05$) for steers receiving .85% K than those fed .54% K. This may be due to increased ruminal nitrogen degradation (Table 5) or reduced use of ammonia-nitrogen for synthesis of microbial protein. Microbial nitrogen flow to the duodenum tended to be greater for the .54% and .75% K diets (23.2 and 23.4 g/d) than with the .63% and .85% K rations (20.7 and 21.0 g/d).

Ruminal fluid dilution rates and rumen volumes (Table 2) did not differ ($P > .05$). However, ruminal fluid dilution rates tended to be lower with the .54% and .75% K diets, and ruminal fluid volumes were greater with these same diets. By regression across animals and treatments, both ruminal fluid and particle passage rate (Figure 1) were inversely related ($r = -.88$; $P < .0001$; $r = -.45$; $P < .08$) to ruminal volume. Outflow rate (ml/h) was not significantly altered by dietary K level but tended to increase with increasing levels of K in the diet. Urinary excretion of K may have increased water excretion and intake, as water intake and urine excretion volume usually are proportional to K intake. Ruminal liquid and solids passage rates were correlated positively ($r = .46$; $P < .07$).

There was a significant cubic effect ($P < .03$) of increasing dietary K on K flow to the duodenum, with the flow decreasing at the .63% K level, increasing at the .75% K level, and decreasing again at the .85% K level. Flow of K to the duodenum was weakly correlated with ruminal fluid passage rate ($r = .25$; $P < .34$).

If K flow to the duodenum alters ruminal fluid passage rate, this may have an effect on dry matter flow into the duodenum (Figure 2). A similar cubic relationship ($P < .1$) between duodenal dry matter flow and dietary K level was noted. Regression analysis across all treatments

revealed a positive correlation between K flow and dry matter flow to the duodenum ($r=.74$; $P<.0009$).

Ruminal fluid outflow and percentage dry matter flow to the duodenum discussed above were calculated based on the rumen fluid passage marker, CoEDTA. These measures rely on accuracy of marker techniques and require frequent sampling to obtain an estimate of ruminal passage. An alternative method by which liquid passage from the rumen can be calculated is from the quantity of wet matter entering the duodenum based on the passage rate marker, chromic oxide.

Liquid flow to the duodenum, calculated in this manner, displayed a similar cubic response ($P<.09$) to dietary K level. The amount of K entering the duodenum and liquid flow to the duodenum calculated in this manner were correlated ($P<.009$) as shown in Figure 3. Although patterns were similar, flow rates calculated from chromic oxide passage were 1.4 to 2.4 times greater than the flow rates predicted from ruminal sampling following dosing with CoEDTA (Figure 4). These two flow rates were not closely correlated ($P<.58$). Failure of the two methods to check is surprising and disappointing.

Several reasons for this discrepancy can be proposed. First, migration of the fluid marker in and out of particles could alter pool sizes and flow rates. Rate of passage from the rumen depends on location of the marker. Marker in the free form would leave the rumen more rapidly than marker bound to particulate matter. A second, more plausible explanation for the discrepancy is addition of secretions from the abomasum. Hill (1965) noted that the abomasal secretion in sheep totals 5-6 liters and in the cow totals 30-35 liters per 24 hours. In this study, duodenal flow exceeded ruminal liquid output by a mean of

475 ml/h or 11.4 l/day. Regulation of the secretory flow is quite complex. Secretory flow appears to be enhanced by distention of the abomasum, increased pH, and humoral and neural factors. With roughage diets, dry matter of duodenal and ruminal digesta is reasonably similar. But in this study with concentrate diets, duodenal digesta was much higher in moisture than ruminal digesta.

Figure 5 shows the relationship between the liquid flow to the duodenum (expressed as a fraction of the average of the rumen volumes) and the ruminal fluid and particulate passage rates. The liquid flow rate to the duodenum and ruminal fluid passage rate were not correlated, as shown by the horizontal line ($r=-.002$; $P<.99$). Calculating ruminal fluid passage by means of the amount and percentage of duodenal dry matter may not be a reliable estimate of true ruminal passage. However, duodenal flow (calculated from dry matter flow and dry matter content) and ruminal particulate passage appear to be related (Figure 5). As particulate passage increased, the duodenal flow tended to increase ($r=.61$; $P<.01$). Hence, it may be possible to predict ruminal particulate passage by duodenal sampling techniques if ruminal pool size is known. These results suggest that absorption or secretion of liquid between the rumen and duodenum vary with factors other than dry matter content. Previously, Zinn et al. (1981) suggested that duodenal chyme may be biologically adjusted to maintain a concentration of crude protein of 100 g liquid per g of protein compared to 78.5 in this study. In 10 studies by Zinn and Owens (1982a) values ranged from 68 to 89 g liquid per g of protein. In our study, the regression of duodenal flow of chyme against flow of N (Figure 6) was positively correlated ($r=.94$) which was significant ($P<.0001$). This may be due to the N flow

stimulating HCl input from the abomasum or to differences in abomasal absorption due to an increase in osmotic pressure in the abomasum.

Ward (1966) suggested that K level of the diet might alter the rate of passage of fluid contents. During hyperthermic conditions, Schneider et al. (1984) noted that rate of passage of liquid digesta through the rumen of dairy cows was greater with a 1.7% K, .6% Na diet than a .8% K, .18% Na diet. Water intake with the high K and Na level was 15% greater than with the control diet (.8% K and .18% Na), but rumen volume was not reported. Thus, differences in passage rates may have been related to differences in rumen volume as well as water intake.

The means for K and Na concentrations of the rumen fluid (Table 2) are averaged from six sampling times. The ruminal ratios of K to Na (ppm) were .44:1, .21:1, .54:1, and .19:1 for the .54%, .63%, .75% and .85% K diets, respectively. Rumen fluid concentration of both cations and their ratios did not differ significantly ($P > .05$) with dietary K level. Though K concentrations tended to increase with increases in dietary K, differences were smaller than expected and variable. This may be due to the interchange of these ions between the rumen and blood. Parthasarathy (1952) noted the passage of ions from blood into the rumen occurred if ruminal Na and K concentrations were lower than in the blood. This would be rare, but might occur if an animal was sick and the electrolyte balance was disrupted. Blood concentrations of Na and K were not measured in this trial.

Two methods of reporting K and Na concentrations exist. The first method is to report the concentrations as ppm. If this is chosen, then the units become mg/l and are basically units of weight. This is acceptable if we assume that sampling error is small or that metabolic

control is on a weight, not a molar, basis. The second method is to report K and Na concentrations as meq/l. Reporting mineral contents as ratios to one another, such as Na:K, takes into account variables such as weight, pH, and temperature and compensates for sampling error. Ratios may reduce the standard error. If metabolic control is based on a molar ratio or molar basis, an equivalent basis would be preferable.

The question is which method to use in reporting these data. If the sums of the K and Na concentrations (total meq/l) are more constant than the sums reported as ppm, it would be preferable to report the data as meq/l. With the rumen fluid Na and K concentrations, the variability of the sums of the ion concentrations in meq/l units is less than the variability of the sums reported as ppm. In this case, it appears preferable to report these data as meq/l. In this report, both expressions will be presented.

Although dietary level of K had no significant impact ($P > .05$) on unadjusted or adjusted ruminal organic matter digestibilities, adjusted ruminal organic matter coefficients (Table 3) tended to be higher for the .85% K ration than the .75% K diet (70.3 versus 62.0%). In previous studies of Zinn and Axe (1983) and Zinn et al. (1983), K supplementation slightly increased ruminal organic matter digestion (6.6% and 5.4%) with most of the advantage being attributed to increased ruminal fiber digestion. Adjusted ruminal organic matter digestibility was negatively correlated with particle passage rate ($r = -.65$; $P < .01$) and ruminal fluid dilution rate ($r = -.31$; $P < .24$). With faster passage out of the rumen, one would expect extent of digestion to decrease.

Total tract organic matter digestion (Table 3) tended to increase with increasing dietary K, in agreement with the report by Zinn and Axe

(1983). Ruminal organic matter digestion, expressed as a percentage of total tract organic matter digestion (Table 3), was greater ($P < .05$) with the .85% than the .75% K diet. Adjusted ruminal organic matter digestion and ruminal organic matter digestion, as a percentage of total, were positively correlated ($r = .99$, $P < .01$), so the increase in ruminal digestion with added K was maintained when ruminal digestion was expressed as a percentage of total. Particulate passage rate and rumen fluid dilution rate were negatively correlated with ruminal organic matter digestion as a percentage of total ($r = -.58$, $P < .02$; $r = -.34$, $P < .02$). Faster passage decreased extent of fermentation in the rumen.

Post-ruminal organic matter digestion was significantly lower with the .54% K diet (Table 3) than the two intermediate levels of K supplementation ($P < .05$). Though differences in rate of particulate passage through the post-ruminal tract might be involved, this was not measured. The correlation between particulate passage rate from the rumen and extent of post-ruminal organic matter digestion was low ($r = .001$, $P > 1.00$).

Ruminal starch digestion (Table 4) did not differ ($P > .05$) with K level but was 7 percent greater for the .85% K diet than the .75% K diet. Ruminal starch digestion was negatively correlated with the ruminal ratio of K to Na ($r = -.62$; $P < .01$). Ruminal starch digestion also was negatively correlated with fluid dilution rate ($r = -.36$, $P < .18$) and with particulate passage rate ($r = -.65$, $P < .01$) (Figure 7).

Post-ruminal starch digestion was significantly greater at .63% than at .45% K. Whether the increase in post-ruminal starch digestion was due to increased digestion in the large intestine or increased fermentation of starch in the small intestine plus colon was not

determined in this study. As in the rumen, the large intestine or colon may have an optimal K to Na ratio for fermentation.

Total tract starch digestion paralleled total tract organic matter digestion and did not differ with K level ($P > .05$). This agrees with reports by Zinn and Owens (1980) and Zinn et al. (1982a). Particulate passage rate from the rumen was inversely related to total tract starch digestion ($r = -.63$; $P < .01$).

Ruminal, post-ruminal, and total tract nitrogen digestibilities (Table 5) were not altered by treatment ($P > .05$). This agrees with results of earlier reports (St. Omer and Roberts, 1967; Zinn et al., 1982b; Zinn and Axe, 1983; Zinn et al., 1983). Ruminal nitrogen and total tract nitrogen digestibilities tended to be greatest with the .85% K supplementation. Particulate passage rate and adjusted ruminal nitrogen digestibility were negatively correlated ($r = -.43$; $P < .1$) (Figure 7).

A cubic effect ($P < .02$) of K on microbial efficiency was detected with efficiency decreasing with the first added K level, increasing at .75% K and again dropping with .85% K. An elevated liquid and particulate passage rate may reduce the efficiency of microbial growth and decrease flow of microbial nitrogen from the rumen if time of fermentation is inadequate for maximum microbial colonization. However, adjusted ruminal organic matter digestibility and microbial efficiency were negatively correlated ($r = -.95$; $P < .0001$). Such a relationship has been noted previously and could reflect that particles act to transport bacteria from the rumen. Relative rates of (1) microbial dilution, (2) feed removal, (3) fermentation rate and capacity, and (4) lag time for fermentation are needed to assess the total effect of altered dilution

rates on microbial output from the rumen (Goetsch and Owens, 1984). Lower liquid and particulate passage rates may enhance the proportion of duodenal chyme which is microbial protein but decrease microbial efficiency. Alternatively, this relationship may be due to marker problems or reflect ruminal flow conditions more optimal for efficient microbial growth. In this trial, microbial efficiency (Figure 8) was correlated positively with particulate passage ($r=.64$, $P<.008$) and liquid passage rate ($r=.36$, $P<.17$).

Ward (1966) proposed that K increases the osmotic pressure of the rumen fluid and functions to maintain a higher moisture content in the rumen fluid which could increase bacterial fermentation. Bryant et al. (1959), Chow and Walker (1964), and Scott (1966) have suggested that the ratio of K to Na in the rumen is important for growth of rumen microorganisms. Caldwell and Hudson (1974) found the optical density doubling times of microorganisms (used as an index of growth rate) increased with an increasing concentration of K in the presence of excess Na ions, and Devlin et al. (1969) noted that in vitro microbial activity increased as dietary K was increased to .85% K in the diet. In this trial, the ratio of K to Na in the rumen (Figure 9) was correlated positively with microbial efficiency ($r=.73$, $P<.001$) indicating that growth may be more efficient at higher K:Na ratios.

Total tract digestion of acid detergent fiber (Table 6) was greater ($P<.05$) for the .85% than the .54% K diet, and total tract digestion increased linearly with increasing dietary K. Zinn and Axe (1983) also noted total tract acid detergent fiber digestion tended to increase as K increased from .65 to 1.00% of ration dry matter; but Zinn et al. (1983) reported no change in total acid detergent digestibilities between 1.0%

and 1.5% dietary K. Differences in total tract acid detergent fiber digestion appeared to be due primarily to the differences in post-ruminal digestion ($P < .05$). Whether this is due to altered conditions for postruminal fermentation or other factors is uncertain.

Apparent ruminal digestion of acid detergent fiber was significantly lower ($P < .05$) with the .75% K diet than with other K levels. Zinn and Axe (1983) noted ruminal acid detergent fiber digestion was 28% greater with 1.0 than .65% K, and Zinn et al. (1983) reported 66% greater digestion at 1.5% K than at 1.0% K though in both these studies the increase in acid detergent digestion in the rumen was insignificant ($P > .05$). Ruminal acid detergent fiber digestion in our study was inversely related to the ruminal ratio of K to Na ($r = -.52$; $P < .04$). The ruminal ratios of K to Na (ppm) were .44:1, .21:1, .54:1, and .19:1 for the .54%, .63%, .75% and .85% K diets, respectively. Potassium and Na concentrations and ratios appear important for cellulose fermentation. Chow and Walker (1964) reported that the normal ratio of K to Na in sheep rumen fluid was .33 to 1. Possibly the .75% K diet was too low in Na for cellulolytic bacteria. Bryant et al. (1959) found that at lower levels of Na, higher levels of K were necessary for rapid growth of rumen bacteria so the total as well as the ratio is important.

Ruminal starch and acid detergent fiber digestion were positively correlated ($r = .5$; $P < .05$). Depressed ruminal fiber digestion could alter starch digestion by increasing the lag time for digestion of cell structures which shield starch or by increasing the amount of particulate residues, rumen motility, and rate of exit of particles containing starch from the rumen (Goetsch and Owens, 1984). Ruminal

acid detergent fiber digestion as a percent of total tract digestion paralleled the trends of apparent ruminal fiber digestion. Greater ruminal than total tract ADF digestion with the .54 K diet must reflect errors in sampling.

Post-ruminal acid detergent fiber digestion was inversely related to ruminal acid detergent fiber digestion ($r=-.75$; $P<.001$). This inverse relationship reflects compensatory fiber digestion of ADF escaping fermentation in the rumen. Post-ruminal starch digestion was positively correlated with post-ruminal fiber digestion ($r=.51$; $P<.04$). Digestions would be expected considering the fact that the cecum and large intestine possess considerable fermentative capability and can digest soluble carbohydrates and cellulose, yet in the rumen, starch presence can inhibit fiber digestion if pH drops below 6. In this study, fecal pH always exceeded 6, so such an effect is unlikely. When the microbes of the cecum and large intestine digest acid detergent fiber, this may reduce the size of particles containing starch which then are exposed to be fermented to volatile fatty acids. Hence, post-ruminal acid detergent fiber digestion should increase starch fermentation. Intestinal disappearance of K was negatively correlated with post-ruminal acid detergent fiber digestion ($r=-.41$; $P<.11$). Post-ruminal fiber digestion also may depend on certain levels or ratios of K to Na in the cecum and large intestine though neither of these parameters were measured in this study.

Although dietary K levels were calculated to be .48%, .64%, .79%, and .95% K, which should have provided a range in K intakes, total K intakes were not greatly different (Table 7).

The amount of K leaving the abomasum and appearing in feces was

greatest for the .75% K diets. The amount of K entering the duodenum paralleled microbial efficiency ($r=.80$; $P<.0002$). Both microbial efficiency and duodenal K may be related to particulate passage rate. Particulate passage rate (Figure 10) was correlated positively with the ratio of K to Na in the rumen ($r=.76$; $P<.0006$). Increasing dietary K levels alter not only the total concentrations of K and Na ions but also their ratio. Such alterations could disrupt the osmotic balance and alter passage rates of both fluid and particulate matter.

Apparent ruminal uptake of K was negative reflecting recycling of 3 to 10 g of K daily from the blood or from the saliva back into the rumen. At typical blood levels, recycling of 3.5 to 4.2 g daily would be expected if daily salivary input was 11.6 to 13.9 l. Potassium recycling tended to be reduced at higher levels of dietary K. Recycling of K was correlated positively with the ratio of K to Na in the rumen ($r=.91$; $P<.0001$) and particulate passage rate ($r=.71$; $P<.002$). In previous trials with steers (Greene et al., 1983c) and with lambs (Greene et al., 1983a), a low level (.6%) of K resulted in a recycling of K to the rumen but pre-intestinal absorption was positive with higher levels of dietary K.

Fecal K was greatest ($P<.05$) with the .75% K ration and was correlated positively with the ratio of K to Na in the rumen ($r=.65$; $P<.006$) and in the duodenum ($r=.62$; $P<.01$). Postruminal K uptake exceeded 94%. Previous results concerning effects of dietary K level on fecal excretion of K are conflicting (Greene et al., 1983a; Greene et al., 1983b; Greene et al., 1983c). In two trials with sheep (Greene et al., 1983a; 1983b) fecal K of lambs increased ($P<.05$) with increasing levels of dietary K; whereas, in a trial with cattle (Greene et al.,

1983c) fecal K was not changed ($P > .05$) but tended to be greater with elevated K levels in the diet.

No significant differences ($P < .05$) existed in post-ruminal or total tract uptake of K although total tract uptake tended to be greater at higher K intakes. This agrees with results of Greene et al. (1983a, 1983b, 1983c).

Sodium also was primarily absorbed post-ruminally (Table 8). A large amount (19 to 26 g/day) of Na from the blood and from the saliva was recycled into the rumen of steers with all treatments but tended to be less with higher intakes of K. From 48.8 to 58.6 g could be expected from salivary flow alone if salivary input was 11.6 to 13.9 l. Sodium flux from extracellular water to the gut helps maintain the ionic concentration of the fluid and should be greater with lower K intakes (Ward, 1966). With increasing K intake, fecal Na excretion tended to decrease slightly. Post-ruminal and total tract Na uptake tended to increase with increasing level of dietary K. Post-ruminal uptake of Na exceeded 97%. These results agree with those of Greene et al. (1983c).

Differences in the ruminal, duodenal, or fecal ratios of K to Na were not consistent ($P > .05$); Table 9). As mentioned previously, the ratio of K to Na was highly correlated with many of the digestibility changes though the ratio per se may not be directly responsible for the differences. Instead, differences may be due to changes in the rates of passage of fluid and particulate matter. Levels or ratios of these cations are certainly involved with osmotic pressures and can alter influx and efflux of water from the gut. Rumen fluid passage rate and ratios of K to Na were correlated positively in the rumen ($r = .25$; $P < .36$).

The relationship of concentrations or ratios of Na and K to particulate passage rate has received less attention. In this trial, particulate passage rate was correlated positively with the ratio of K to Na in the rumen ($r=.76$; $P<.0006$).

Further research should investigate the effects of K and Na concentrations and ratios on digestibility, rates of passage, and rumen volumes in feedlot steers. The next challenge will be to select an optimal ratio and develop rations to optimize Na and K levels.

TABLE 1. COMPOSITION OF FINISHING DIETS WITH VARYING
DIETARY LEVELS OF POTASSIUM (K)

Ingredient	Diets			
	.54% K	.63% K	.75% K	.85% K
Dry rolled corn (IFN 4-02-931)	81.41	81.13	80.84	80.57
Cottonseed hulls (IFN 1-01-599)	10.36	10.35	10.35	10.35
Soybean meal (IFN 5-20-637)	4.03	4.03	4.03	4.03
Cottonseed meal (IFN 5-01-621)	2.01	2.01	2.01	2.00
Calcium carbonate (IFN 6-01-069)	1.00	1.00	1.00	1.00
Sodium chloride (IFN 6-04-152)	.30	.29	.29	.30
Urea (IFN 5-05-070)	.56	.56	.56	.56
Cane molasses (IFN 4-04-696)	.25	.25	.24	.24
Trace mineral	.01	.01	.01	.01
Vitamin A ^a	.01	.01	.01	.01
Rumensin ^b	.02	.02	.02	.02
Tylan ^c	.04	.04	.04	.04
Potassium chloride (IFN 6-03-755)	.00	.29	.58	.87
Chromic oxide ^d	.30	.30	.30	.30
Analysis:				
Crude protein, %	11.0	10.7	10.9	11.1
Starch, %	53.5	51.4	53.2	53.3
K, ppm	5403	6346	7502	8488
Na, ppm	1211	1065	1091	1214
Ash, %	3.4	3.4	3.2	3.8

^aVitamin A = 30,000 USP/g.

^bRumensin = 60 g/lb.

^cTylan = 10 g/lb.

^dAll diets contained chromic oxide as an indigestible marker at .3% above the total ration.

TABLE 2. MEAN DIGESTIVE TRACT MEASUREMENTS IN STEERS
FED A HIGH CONCENTRATE DIET WITH VARYING
DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^d	Effect ^e
	.54% K	.63% K	.75% K	.85% K		
Ruminal pH	6.22	6.17	6.24	6.21	.11	
Duodenal pH	2.33	2.39	2.33	2.34	.05	
Fecal pH	6.30	6.47	6.54	6.45	.07	
Ruminal ammonia -N, mg/dl ^c	3.51 ^b	3.84 ^{ab}	3.72 ^{ab}	5.57 ^a	.56	L
Ruminal fluid						
Passage rate, %/h	4.14	5.15	4.61	6.00	.72	
Volume, liter	12.7	10.8	12.0	10.5	.9	
Outflow, ml/h	484	522	544	581	57	
Particulate passage rate, %/h	4.32	3.37	3.80	4.03	.57	
Flow to duodenum ^f :						
K, g/h	1.01	.85	1.24	.93	.10	C
Dry matter, g/h	51.3	50.6	56.9	45.9	3.0	
Dry matter, %	10.9	9.9	11.0	7.9	1.6	
Flow to duodenum ^g :						
Liquid flow, ml/h	1090	906	1071	909	56	
Fluid passage rate, %/h	9.47	8.34	9.31	7.90	.49	
Rumen fluid K, ppm ^c	1514	1518	1819	1644	116	
meq/l	38.7	38.8	46.5	42.1		
Rumen fluid Na, ppm ^c	2011	1815	1884	1788	134	
meq/l	87.5	79.0	82.0	77.8		

^{ab}Means in a row with different superscripts differ statistically (P<.05).

^cThe mean is averaged over six sampling times.

^dStandard error of the means.

^eMeans in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

^fThese values are based on the use of markers.

^gThese values are based on actual dry matter content of duodenal chyme.

TABLE 3. ORGANIC MATTER DIGESTION IN STEERS FED A HIGH CONCENTRATE DIET WITH VARYING LEVELS OF POTASSIUM (K)

Item	Diets				SE ^d	Effect ^e
	.54% K	.63% K	.75% K	.85% K		
Intake, g/d	2545	2546	2544	2514		
Leaving abomasum, g/d						
Total	1041	1044	1177	943	62.4	
Microbial	227	203	229	206	9.1	
Ruminal digestion						
% unadjusted	58.9	58.9	52.9	62.1	2.4	
% adjusted ^c	67.9	66.9	62.0	70.3	2.4	
Ruminal digestion, % of total	71.3 ^a	68.7 ^{ab}	61.7 ^b	72.1 ^a	2.5	Q
Feces, g/d	446	367	389	350	27.5	
Post-ruminal digestion, % of entering	45.2 ^b	56.5 ^a	57.2 ^a	52.2 ^{ab}	2.7	Q
Total tract digestion, %	82.4	85.6	84.5	86.0	1.1	

^{ab} Means in a row with different superscripts differ statistically (P<.05).

^c Adjusted for microbial organic matter.

^d Standard error of the means.

^e Means in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

TABLE 4. STARCH DIGESTION IN STEERS FED A HIGH CONCENTRATE DIET WITH VARYING DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^c	Effect ^d
	.54% K	.63% K	.75% K	.85% K		
Intake, g/d	1410	1356	1398	1393		
Leaving abomasum, g/d	248	261	315	232	45.4	
Apparent ruminal digestion, %	82.4	80.7	76.6	83.1	3.4	
Ruminal digestion, % of total	86.2	82.7	79.1	85.6	3.4	
Feces, g/d	62.6	37.2	48.4	41.4	8.2	
Post-ruminal digestion, % of entering	76.1 ^b	88.2 ^a	85.0 ^{ab}	81.3 ^{ab}	3.2	Q
Total tract digestion, %	95.5	97.4	96.4	97.1	.6	

^{ab} Means in a row with different superscripts differ statistically (P<.05).

^c Standard error of the means.

^d Means in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

TABLE 5. NITROGEN (N) DIGESTION IN STEERS FED A HIGH CONCENTRATE DIET WITH VARYING DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^d	Effect ^e
	.54% K	.63% K	.75% K	.85% K		
Intake, g/d	46.2	45.3	46.1	46.2		
Leaving abomasum, g/d						
Total N	50.6	48.9	53.0	45.2	2.8	
Microbial N	23.2	20.7	23.4	21.0	.9	
Ammonia N	3.5	3.3	4.0	3.4	.4	
Rumen by-pass feed N	24.0	24.9	25.7	20.8	2.2	
Rumen digestion, %						
% unadjusted	-9.7	-8.5	-19.1	1.7	8.2	
% adjusted ^c	48.3	45.0	42.2	54.8	5.3	
By-pass feed N, %	51.7	55.1	57.8	45.2	5.3	
Microbial efficiency, g microbial N/kg OM truly digested in rumen	13.8 ^{ab}	12.4 ^b	16.4 ^a	12.2 ^b	1.0	C
Ruminal digestion, % of total	-14.5	-12.0	-31.8	1.6	11.8	
Feces, g/d	13.4	11.7	12.3	11.4	.7	
Post-ruminal digestion, % of entering	73.4	76.0	76.8	74.9	1.0	
Total tract digestion, %	71.0	74.0	72.3	75.1	1.7	

^{ab} Means in a row with different superscripts differ statistically (P<.05).

^c Adjusted for microbial and ammonia nitrogen.

^d Standard error of the means.

^e Means in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

TABLE 6. ACID DETERGENT FIBER DIGESTION IN STEERS FED A HIGH CONCENTRATE DIET WITH VARYING DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^c	Effect ^d
	.54% K	.63% K	.75% K	.85% K		
Intake, g/d	292 ^b	292 ^b	292 ^a	290 ^b		
Leaving abomasum, g/d	159 ^b	175 ^b	197 ^a	156 ^b	6.3	Q,C
Apparent ruminal digestion, %	44.9 ^a	39.8 ^a	31.0 ^b	45.7 ^a	2.1	Q,C
Ruminal digestion, % of total	104.6 ^a	80.7 ^b	58.2 ^c	84.8 ^{ab}	5.8	L,Q,
Feces, g/d	165 ^a	150 ^{ab}	143 ^{ab}	131 ^b	7.2	L
Post-ruminal digestion, % of entering	-5.8 ^c	11.0 ^b	25.2 ^a	12.9 ^b	3.5	L,Q
Total tract digestion, %	43.4 ^b	48.6 ^{ab}	50.3 ^{ab}	54.5 ^a	2.3	L

^{ab} Means in a row with different superscripts differ statistically (P<.05).

^c Standard error of the means.

^d Means in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

TABLE 7. POTASSIUM (K) UPTAKE IN STEERS FED A HIGH CONCENTRATE DIET WITH VARYING DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^d	Effect ^e
	.54% K	.63% K	.75% K	.85% K		
Intake, g/d	14.2 ^{ab}	16.8 ^b	19.7 ^a	19.6 ^{ab}		
Leaving abomasum, g/d	24.3 ^{ab}	20.5 ^b	29.7 ^a	22.3 ^{ab}	2.4	C
Apparent ruminal uptake, %	-72.8 ^c	-24.0 ^{ab}	-51.1 ^{bc}	-16.6 ^a	9.4	L,C
Ruminal uptake, % of total	-80.1 ^c	-26.5 ^{ab}	-58.7 ^{bc}	-18.9 ^a	10.7	L,C
Feces, g/d	1.2 ^b	1.2 ^b	1.8 ^a	1.2 ^b	.2	C
Post-ruminal uptake, % of entering	95.0	94.1	93.7	95.0	.5	
Total tract uptake, %	91.8	92.5	90.6	93.9	.9	

^{abc} Means in a row with different superscripts differ statistically (P<.05).

^d Standard error of the means.

^e Means in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

TABLE 8. SODIUM (NA) UPTAKE IN STEERS FED A HIGH CONCENTRATE DIET WITH VARYING DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^c	Effect ^d
	.54% K	.63% K	.75% K	.85% K		
Intake, g/d	3.1	2.8	2.9	3.2		
Leaving abomasum, g/d	29.6	26.3	25.7	22.1	2.8	
Apparent ruminal uptake, %	-1021. ^b	-847. ^{ab}	-844. ^{ab}	-600. ^a	112	L
Ruminal uptake, % of total	-1342	-1152	-943	-671	266	
Feces, g/d	.70	.55	.25	.35	.19	
Post-ruminal uptake, % of entering	97.5	97.9	99.0	98.3	.7	
Total tract uptake, %	80.0	79.5	90.8	89.0	6.7	

^{ab} Means in a row with different superscripts differ statistically (P<.05).

^c Standard error of the means.

^d Means in a row differ linearly (L), quadratically (Q), or cubically (C) (P<.05).

TABLE 9. POTASSIUM (K)/SODIUM (NA) RATIOS OF STEERS FED A HIGH CONCENTRATE DIET WITH VARYING DIETARY LEVELS OF POTASSIUM (K)

Item	Diets				SE ^C
	.54% K	.63% K	.75% K	.85% K	
Feed	5.26	6.15	7.22	7.42	.96
Rumen	.44	.21	.54	.19	.14
Duodenum	.89	.85	1.29	1.12	.19
Fecal	7.91	5.41	10.63	5.90	2.93

^aStandard error of the means.

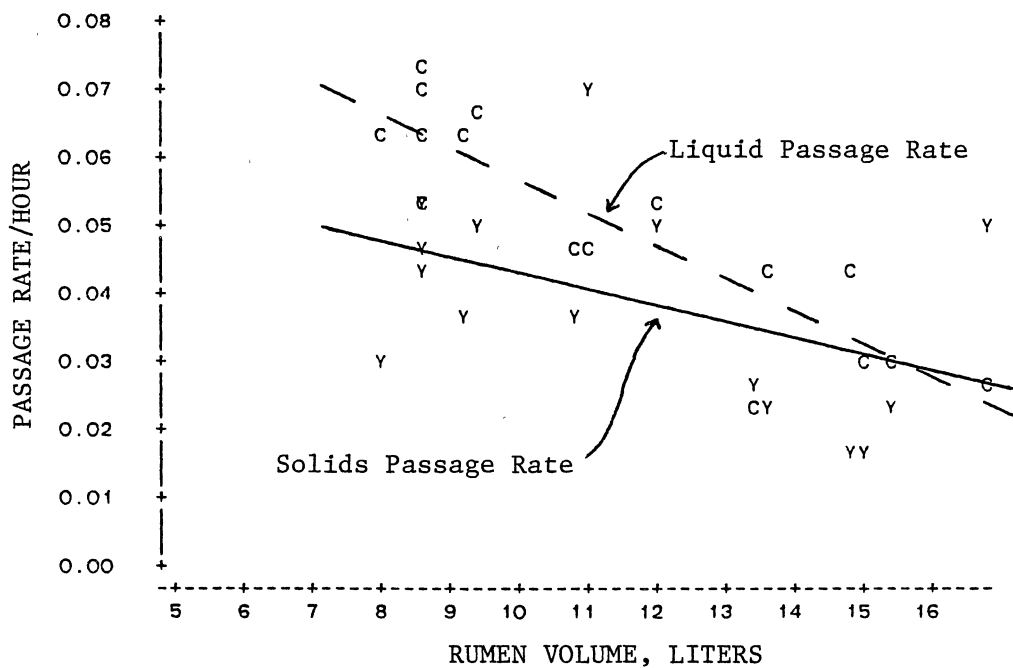


Figure 1. Solids (Y) and Liquid (C) Passage Rates vs Rumen Volume
 $Y = .065 - .0023 (\text{Rumen Volume, liters})$;
 $r = -.45$; $P < .081$.
 $C = .106 - .0049 (\text{Rumen Volume, liters})$;
 $r = -.88$; $P < .0001$.

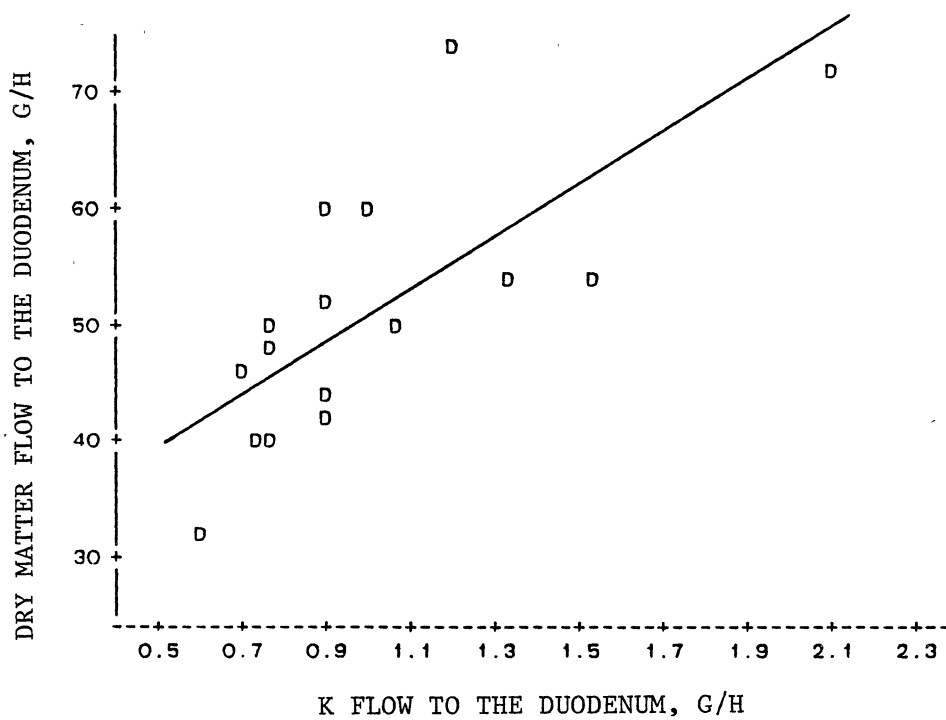


Figure 2. Dry Matter Flow to the Duodenum (D) vs Potassium Flow
 $D = 28.504 + 22.488 (K \text{ Flow to the Duodenum, g/h})$; $r = .74$; $P < .0009$.

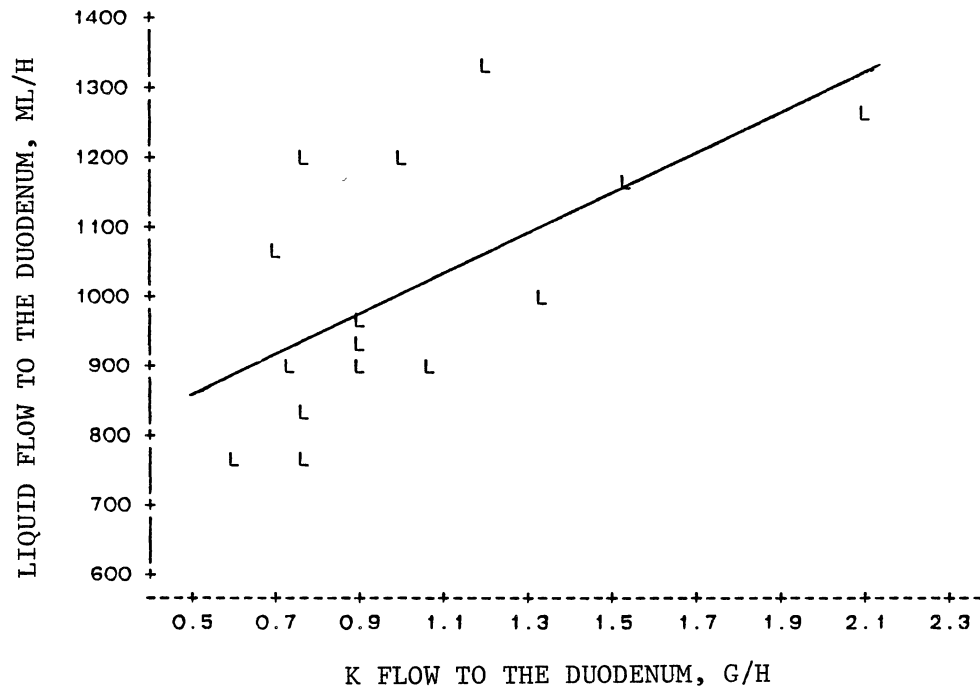


Figure 3. Liquid Flow (Based on DM Content) (L) vs K Flow to the Duodenum
 $L = 713.898 + 291.657 (K \text{ Flow to the Duodenum, g/h})$; $r = .63$; $P < .0088$.

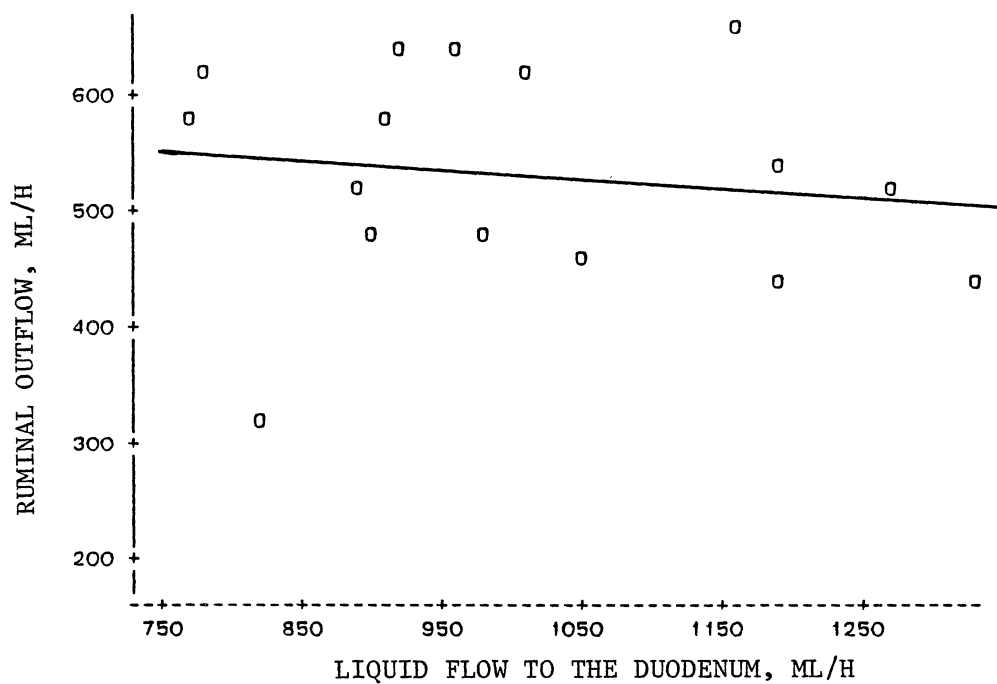


Figure 4. Ruminal Outflow (O) vs Liquid Flow to the Duodenum (Based on DM Content)
 $O = 612.768 - .079$ (Liquid Flow to the Duodenum, ml/h); $r = -.15$; $P < .58$.

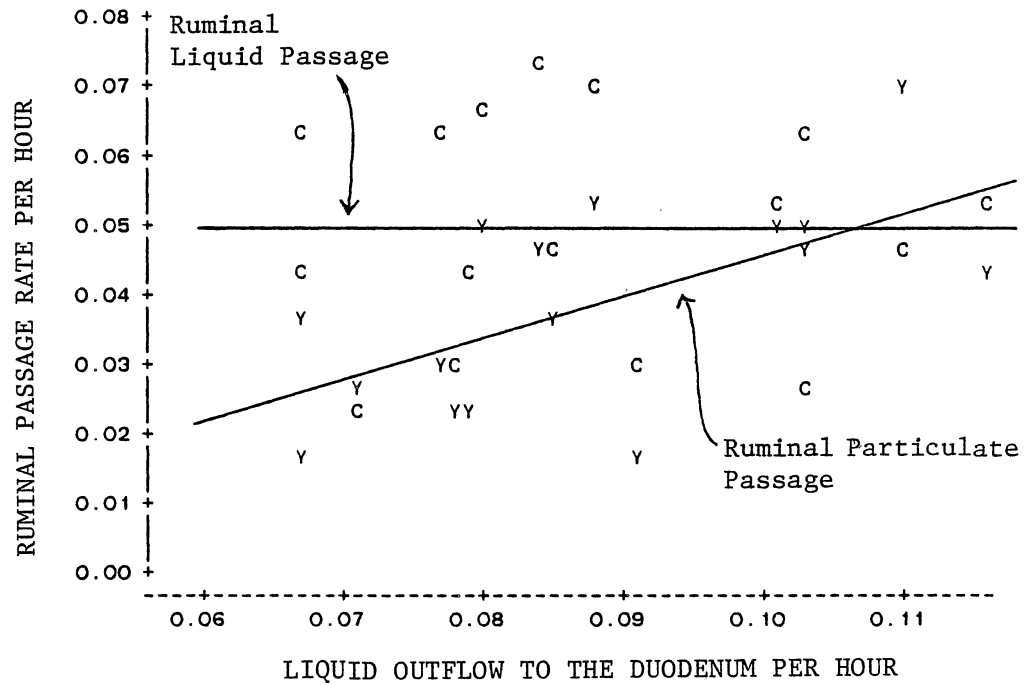


Figure 5. Ruminal Liquid (C) and Particulate Passage Rates (Y) vs Liquid Outflow to the Duodenum (Based on DM Content)
 $C = .050 - .0026$ (Liquid Outflow to the Duodenum per Hour); $r = -.002$; $P < .99$.
 $Y = -.014 + .601$ (Liquid Outflow to the Duodenum per Hour); $r = .61$; $P < .01$.

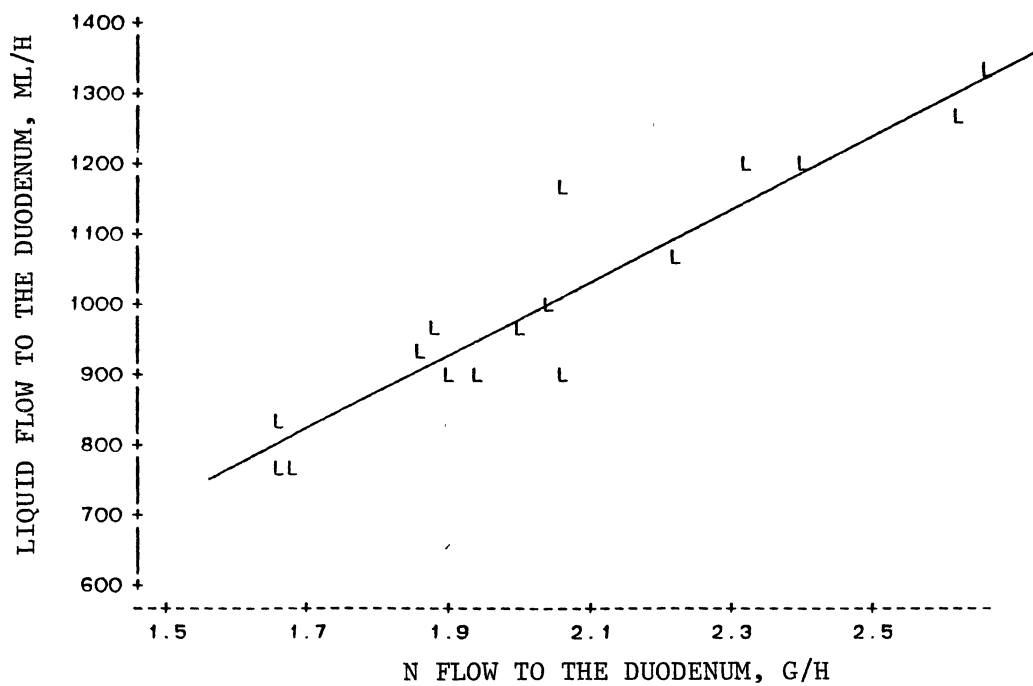


Figure 6. Liquid Flow to the Duodenum (L) vs N Flow
 $L = -74.485 + 525.603 (N \text{ Flow to the Duodenum, g/h})$; $r = .94$; $P < .0001$.

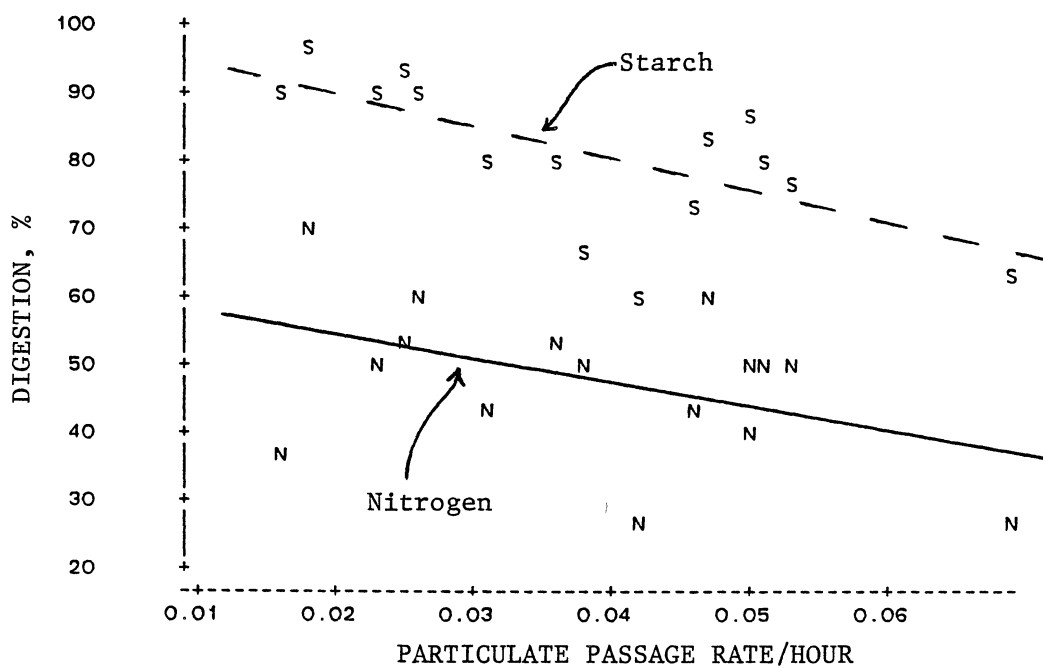


Figure 7. Ruminal Nitrogen (N) and Starch (S) Digestion vs Particulate Passage Rate
 $N=60.885-342.92$ (Particulate Passage Rate/Hour);
 $r=-.43$; $P<.1$.
 $S=98.277-452.305$ (Particulate Passage Rate/Hour); $r=-.65$; $P<.01$.

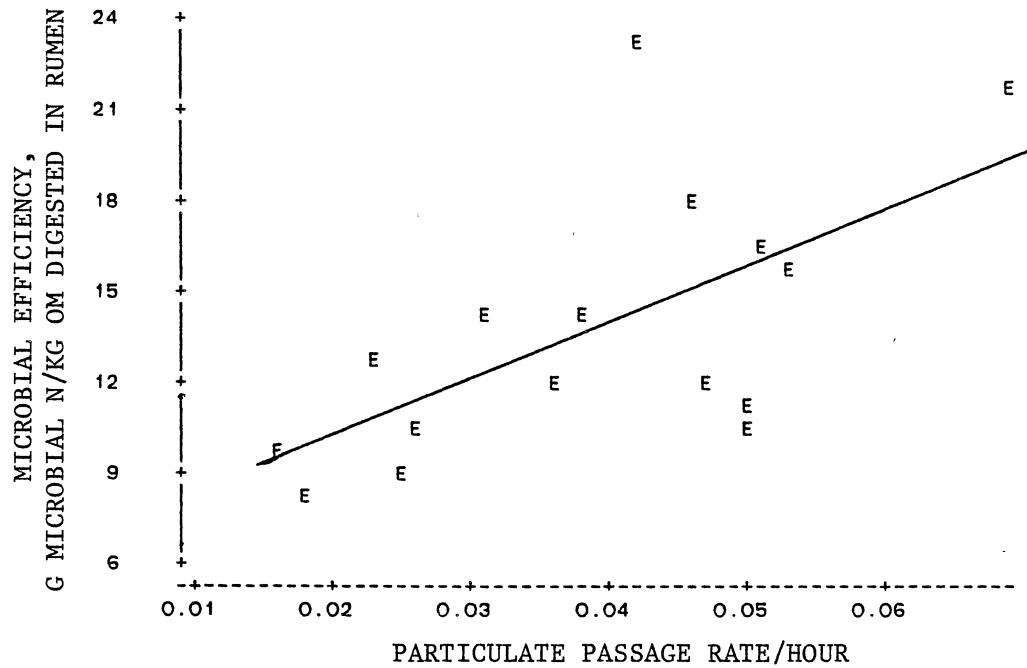


Figure 8. Microbial Efficiency (E) vs Particulate Passage Rate
 $E = 6.353 + 188.510 (\text{Particulate Passage Rate/ Hour})$; $r = .64$; $P < .008$.

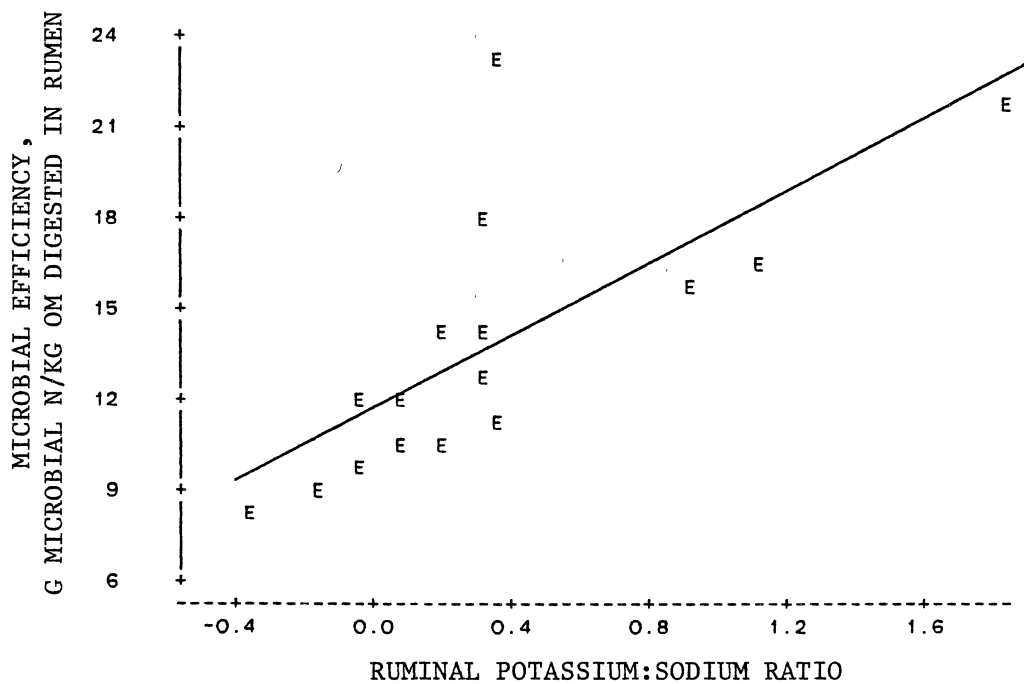


Figure 9. Microbial Efficiency (E) vs Ruminal Potassium:Sodium Ratio
 $E = 11.622 + 5.930 (\text{Ruminal Potassium:Sodium Ratio})$; $r = .73$; $P < .001$.

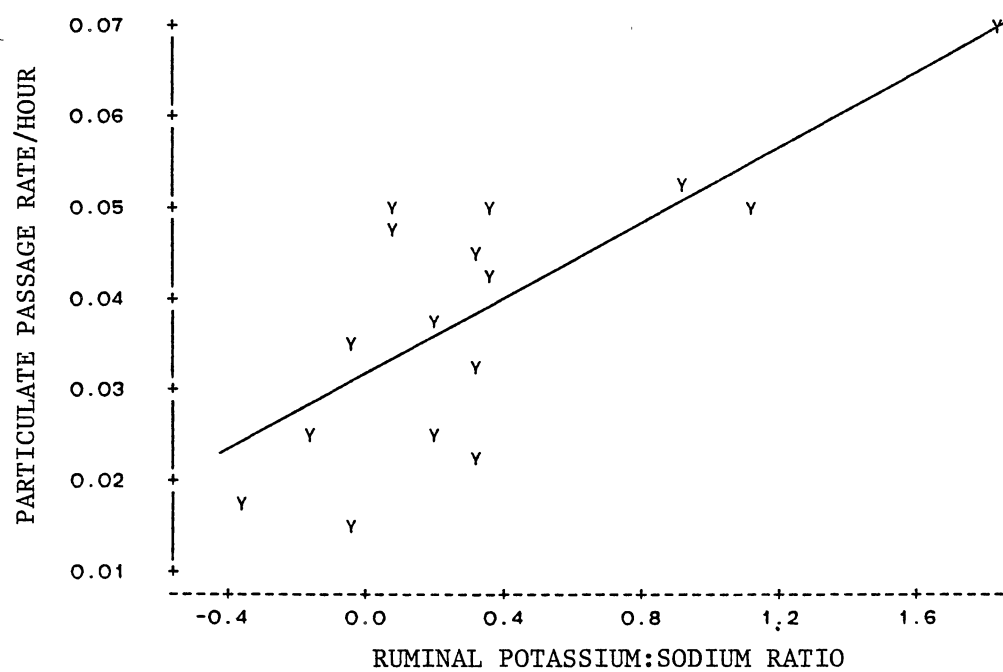


Figure 10. Particulate Passage Rate (Y) vs Ruminal Potassium:Sodium Ratio
 $Y = .032 + .021(\text{Ruminal Potassium:Sodium Ratio})$; $r = .76$; $P < .0006$.

CHAPTER V

EFFECTS OF SUPPLEMENTAL POTASSIUM AND IONOPHORES ON FERMENTATION IN A SEMI-CONTINUOUS RUMEN CULTURE SYSTEM

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Summary

A semi-continuous rumen culture system was used to determine the effect of supplemental KCl and ionophores on in vitro fermentation of a 60% rolled milo, 30% prairie hay ration. Potassium levels selected for investigation were .56% (basal) and 4.97% of total ration dry matter. Monensin, salinomycin, and lasalocid were provided at 27, 11 and 33 ppm, respectively. Fermentors were maintained at a dilution rate of 2.1%/h for 22 days. Addition of each ionophore tended to increase organic matter digestion, total volatile fatty acid production, and the molar percentage of propionic acid. Concentrations of ammonia, numbers of protozoa, and the percent K solubilized also were increased with ionophores. Bacterial numbers, ribonucleic acid-nitrogen, percent of Na solubilized, and pH of the fermentors tended to be reduced by addition of an ionophore. Significant K x ionophore interactions resulted in the combination reducing organic matter digestion and total volatile fatty acids. Ribonucleic acid-nitrogen, pH, and bacterial numbers were

increased by the K-ionophore combination. High dietary concentrations of K appear to inhibit the selective antimicrobial activities of the ionophores. High K levels had greater impact on action of monensin than on other ionophores. This trial suggests that high dietary levels of K counteract certain ionophore actions, presumably by altering the ion transport by the ionophore.

(Key Words: In Vitro, Potassium Chloride, Monensin, Salinomycin, Lasalocid)

Introduction

Dietary ionophores such as monensin, lasalocid, and salinomycin change many aspects of mineral metabolism and physiology of tissues dependent on ion movement (Elsasser, 1984). Such changes are partly a result of selective alteration of rumen microflora. Ionophore-resistant bacteria can develop, yet altered ruminal fermentation remains. Recent research of Dawson and Boling (1984a) suggests that the ability of rumen organisms to concentrate K cations within the cell is an important factor in determining susceptibility of organisms to ionophores.

Ionophores form lipid soluble complexes with certain cations and facilitate their transport across biological membranes. Thus, interactions between concentrations of cations and ionophores might be expected. Because ionophores reduce intracellular K concentrations of microorganisms, an increased K concentration in the rumen could inhibit the selective antimicrobial activities of monensin and lasalocid (Dawson and Boling, 1984b). Increasing the K concentration of an in vitro medium decreased the antimicrobial activity of lasalocid while increased Na concentrations of the medium amplified the antimicrobial effects of

lasalocid (Dawson et al., 1983). An in vitro trial of Salsbury and Romatowski (1978) revealed that adding Na to a monensin-containing culture increased the amount and molar proportion of propionic acid produced. In contrast, addition of K concentration increased the amount of propionate formed, but did not alter the molar ratio of acids produced.

Currently, monensin and lasalocid are approved for use in cattle feeds at levels of 5-30 and 10-30 g/ton of complete feed, respectively (NRC, 1984). Salinomycin has not yet been approved for use in complete cattle feeds but should soon be released. Since ionophores appear to interact with Na and K cations, the consequences of K and Na supplementation need to be determined.

The objectives of this in vitro study were to: (1) investigate the interactions between dietary K level and ionophore addition, and (2) assess potential differences in fermentation patterns among monensin, lasalocid, and salinomycin.

Materials and Methods

A modification of the semi-continuous rumen culture system (Goetsch and Galyean, 1982) was used in a 22 day trial. Rumen fluid was obtained from two 450 kg heifers. One heifer was fed a 90% concentrate, 10% cottonseed hull diet whereas the other heifer received a 90% concentrate, 10% prairie hay diet (Appendix A). At the start of the trial, rumen fluid consisting of equal portions from the two heifers was strained through four layers of cheesecloth. Rumen fluid (100 ml), 100 ml of buffer (McDougall, 1948), 1.0 g (air dry) of finely ground (2mm screen) feed, and a magnetic stir bar were introduced into 250 ml

Erlenmeyer flasks. Flasks were gassed with carbon dioxide, sealed with number eight rubber stoppers equipped with feed entry, gas release, and effluent removal ports, and placed in a 39 C water bath.

Diets for fermentor flasks consisted of 70% concentrate and 30% prairie hay. The eight treatments consisted of: (1) basal diet (calculated to contain .56% K); (2) basal diet plus K supplementation (4.97% K); (3) basal diet plus monensin (27 ppm); (4) basal diet plus salinomycin (11 ppm); (5) basal diet plus lasalocid (33 ppm); (6) basal diet plus K supplementation and monensin (4.97% K, 27 ppm monensin); (7) basal diet plus K supplementation and salinomycin (4.97% K, 11 ppm salinomycin); (8) basal diet plus K supplementation (4.97% K, 33 ppm lasalocid). Triplicate flasks received each treatment.

At 0500 and 1700 h each day, the flasks were placed on a stir plate for mixing, and 50 ml of effluent was extracted by applying suction with a plastic syringe on the piece of tygon tubing submerged in the culture media. New substrate (.5 g) was mixed into a slurry with 35 ml of buffer (pH 7) in the syringe and injected into the cultures via the entry port. The syringe was rinsed with 15 ml of .85% saline solution into the flask. Cultures were gassed for ten seconds with carbon dioxide via the effluent removal port to minimize oxygen contamination during transfers. This transfer procedure resulted in a discontinuous liquid passage rate and a solids input rate equal to 50.4%/day which equaled 2.1%/h.

On days 1 to 3, pH of the effluent removed was measured each morning and evening; thereafter, pH was measured only for the morning effluent. Measurements of treatment effects were following 7 to 10 days of stabilization on days 11, 13, 15, 17, 19, and 21. After pH

measurement of the effluent, one ml of 20% H_2SO_4 was added per 50 ml, and the sample was frozen for later analysis. Thawed effluent (45 g) from the days above was centrifuged for 10 minutes at 10,000 g and analyzed by Eli Lilly and Company for volatile fatty acid concentrations using standard gas chromatography procedures. The remaining supernatant fluid was analyzed for ammonia-nitrogen (Broderick and Kang, 1980) and Na and K concentrations by atomic absorption. A small quantity of the supernatant fluid from day 17 was diluted with either 2000 ppm K solution or 2000 ppm Na solution to determine the Na and K concentrations, respectively.

Evening effluent from days 8 through 21 was composited within treatment and preserved with 20 ml 2N H_2SO_4 per 650 ml effluent. At the end of the trial, this composite sample was agitated and 20 ml were removed, dried at 100 C, and ashed to determine organic matter output. The remaining composited material was dried at 60 C for analysis.

Analysis of the dried composite effluent and feed included dry matter (DM; 105 C for 24 h), ash (600 C for 12 h); and Na and K concentration (as described in Chapter IV). Feed was also analyzed for Kjeldahl nitrogen (AOAC, 1975). In addition, the dried composite effluent samples were analyzed for nucleic acid-N (Zinn and Owens, 1980).

At 0500 on day 11, gas production was measured via water displacement from a measured length of sealed tygon tubing. The tubing was connected to the gas release port after the flask had been gassed with carbon dioxide. Residual gas in the tygon tubing presented problems in displacement. The procedure was repeated again on day 12 with the modification that carbon dioxide was gassed into the tygon

tubing just prior to attachment to the flasks. Water displacement was recorded hourly the first six hours and at 8 and 10 h after attachment.

On day 15, acid production was monitored for each flask using the 0500 effluent. A glucose solution (5 ml of 20% glucose) was added to 40 ml of effluent in a 50 ml centrifuge tube. The tube was capped, and placed in a 39 C water bath. The pH of the tubes was measured hourly for the first 5 h. Tubes were gassed with CO₂ and recapped after each pH measurement to maintain anaerobiosis. The pH remained constant, possibly because the amount of glucose was insufficient. On day 16, the procedure was repeated using 20 ml of effluent and 10 ml of the glucose solution. The pH was monitored hourly the first four hours and at 6, 8, 10, 15, and 24 h. The drop in pH after 8 and 24 h was considered to reflect acid (lactate) production.

On the mornings of days 17, 19, and 21, one ml of effluent was withdrawn with a wide bore pipette prior to acidification and mixed with 2 ml of a .85% saline solution containing 10% (v/v) formalin and .2% (v/v) methylene blue to preserve and stain bacterial cells. Eight hours later, 7 ml of the same solution without methylene blue was added to the bacteria and protozoa samples. Duplicate protozoal counts were determined with a hemacytometer chamber at a magnification of 450x. After another ten-fold dilution with .85% saline, duplicate bacterial counts were taken with a Petroff-Hauser chamber at a magnification of 450x. Five large squares were counted.

At 1700 h on day 22, buffering capacity was measured on 40 ml of effluent from each flask. The pH was first adjusted to 7.0 by addition of .1 N NaOH. This was titrated with .1 N HCl at .5 pH increments to a final pH of 3 (Haaland et al., 1982). Buffering capacity values

presented are the amount of acid needed to decrease pH from 7.0 to 5.0.

Treatment means were evaluated using a general linear model program for a 2 x 4 factorial arrangement of treatments (Statistical Analysis System, 1982). Treatment effects with 7 degrees of freedom were divided to test effects of K level, ionophore presence, and ionophore type which included comparisons of monensin versus other ionophores, salinomycin versus lasalocid, and the potassium level interaction with these contrasts

Results and Discussion

Organic matter digestion (Table 2) was lower than anticipated for this 70% concentrate diet but was not significantly affected ($P > .05$) by K level or ionophores independently. Addition of either tended to increase organic matter digestion. Rust et al. (1978) and Ferrell et al (1982) observed and Schelling (1984) summarized that organic matter digestibility was slightly or moderately increased with monensin. When K was added to the ionophores, the increase in organic matter digestion over the basal diet was reduced ($P < .05$), particularly with the K-monensin diet. Little effect with lasalocid agrees with Funk et al. (1985) who found no evidence of a K-lasalocid interaction on organic matter digestion. The K interaction is consistent with the mode of ionophore action. Since ionophores deplete the intracellular K concentrations of microorganisms, the microbial population is shifted. Hence, increased K concentrations can decrease the antimicrobial and selective activities of ionophores against rumen bacteria. Results suggest that K plays a key role in the antimicrobial activities of the ionophores as suggested by Dawson and Boling (1984b). Interaction of K with monensin was much

with lasalocid or salinomycin, however.

Total volatile fatty acid concentrations (Table 3) were quite low but were increased by adding an ionophore ($P < .05$). Added alone, K increased total VFA concentrations. However, when K was fed in combination with an ionophore, total volatile fatty acid concentration was reduced ($P < .05$) though molar ratios of acetate, propionate, and butyrate were not affected ($P > .05$). Salsbury and Romatowski (1978) also reported that added K increased total volatile fatty acids but did not alter the molar ratios. Addition of either ionophore increased propionate and decreased acetate and butyrate concentrations. Extensive summaries (Elanco, 1975) indicate these responses to monensin also occur in vivo.

The shift in the volatile fatty acid concentrations toward propionate was greatest with the addition of lasalocid, followed by salinomycin and monensin, respectively ($P < .05$). Potassium alone increased acetate and decreased propionic acid. Potassium did not alter the effect of monensin on the acetate:propionate ratio though K increased the acetate:propionate ratio with salinomycin and lasalocid ($P < .05$), moreso with lasalocid than with salinomycin.

Ammonia levels were very low and may have reduced microbial activity. Addition of an ionophore increased ammonia-N levels ($P < .05$) as shown in Table 4. This is contrary to most reports. Because ionophores decrease proteolysis and deamination, ionophores usually decrease ammonia-N levels (Chalupa, 1980). The increased ammonia-N may have been due to the short adaptation period; microorganisms may not have had adequate time to adjust to the dietary additions of the ionophores, or the low levels may reflect ammonia use, not ammonia release. Ammonia-N levels appeared to be less with monensin than

lasalocid. Ricke et al. (1984) reported lower rumen ammonia levels with monensin-fed sheep than with lasalocid-fed lambs. Ammonia-N levels of the K-monensin treatment were greater than for the K or monensin diets implying monensin is affected more by dietary K supplementation than other ionophores.

Bacterial numbers were reduced with the addition of ionophores, but K inhibited this reduction ($P < .05$) (Table 4). Bacterial numbers were greater with the K-monensin diet than with the monensin diet. Protozoal numbers tended to increase with the addition of an ionophore. Chalupa (1980) noted that ionophores decrease protozoa populations in some, but not all studies. In this trial, K supplementation reduced protozoa ($P < .05$), especially in the presence of salinomycin. There are two possible reasons for the reduction in protozoal numbers with added K. An inverse relationship between numbers of protozoa and bacteria is expected. Dawson and Boling (1983b) found that higher K concentrations increased cell yield and decreased the lag times for bacteria grown in the presence of monensin and lasalocid. This may increase competition between bacteria and protozoa for substrates. This could explain why bacterial numbers increased and protozoa numbers decreased with the addition of K to the ionophore diets. Also, Warner and Stacy (1965) found that rumen ciliate protozoa survive best if the media is hypotonic. The media in the present trial was hypertonic. Fluctuations in RNA-N did not appear to parallel the observed changes in the counts of bacteria and protozoa.

Ionophore diets (Table 5) did not alter ($P > .05$) total gas production. This agrees with the reports of Schelling (1984) and Chalupa (1980) who respectively reported no changes in CO_2 or H_2 gas

production with monensin. Methane production is usually inhibited with monensin (Chalupa, 1980; Thornton and Owens, 1981). Methane was not measured separately in this study. Gas production was reduced by addition of K, particularly when combined with ionophores.

When glucose was added to a sample of fermenter fluid to check lactate production, change in pH, which should reflect lactate production, was not affected by the treatments at either 8 or 24 h post dosing ($P > .05$). In previous carbohydrate stress studies (Schelling, 1984), ionophores prevented the drop in pH and lowered lactate production. In this trial, the rumen fluid may have been too highly buffered to permit the pH to change markedly. Alternatively, microbes may have been starved for ammonia which would reduce their ability to use excess carbohydrates.

Buffering capacity from pH 7.0 to 5.0 was greater ($P < .05$) with the K-ionophore diets than the ionophore diets (38.1 vs 37.7 ml). The formation of KHCO_3 from KCl may have increased the buffering capacity of the flasks. Zinn and Owens (1983) stated that K may enhance buffering via conversion to KHCO_3 . The pH values were lower with addition of monensin, salinomycin, and lasalocid ($P < .05$). This may be related to the increased concentrations of total volatile fatty acids with these ionophores, especially salinomycin. Potassium addition with ionophores increased pH levels ($P < .05$) which could be due to reduced total volatile fatty acid concentrations or to formation of KHCO_3 .

From 80 to 92% of the K leaving the fermenters was present in the fluid, but the amount of K solubilized ($P < .05$) was reduced by addition of K. This is consistent with the ionophore model. Ionophores deplete intracellular K concentrations of the microorganisms. Increased dietary

K levels inhibit this transport (Dawson and Boling, 1984b). At higher K levels, less K would be exchanged by the ionophores.

With the low level of K, more of the K was soluble with monensin than salinomycin or lasalocid. This may be due to the differences in the molecular binding ratios. Monensin binds monovalent ions in a 1:1 ratio while the lasalocid binding ratio is 2:1. Lemenager et al. (1978) and Kirk et al. (1985) found that ruminal fluid K increased with monensin supplementation. The percentage K output of the K-monensin diet varied ($P < .05$) with the other two ionophores, the greatest difference being between the K-monensin and K-lasalocid diets. Results agree with those of Spears and Harvey (1985) who noted that added K in the presence of lasalocid reduced Na and increased soluble K concentrations in rumen fluid.

From 72 to 80% of the Na left the fermenters in the fluid phase, but the percentage of Na solubilized was reduced with the addition of ionophores ($P < .05$). Most of the difference was with the K-ionophore diets. High levels of K may compete with Na for the binding sites on the ionophores. Hence, Na transport could be decreased. Potassium:Na ratios were similar across treatments within each level of K. The K:Na output ratios were greater at the high levels of K ($P < .05$), as expected.

In conclusion, this in vitro trial indicates that a high level of K alters the action of ionophores, presumably by altering ion transport by the ionophore. Of the three ionophores studied, monensin action appeared to be altered most by high dietary K levels. Caution should be exercised in the application of these results to in vivo studies as ionophores may exert additional effects in vivo.

TABLE 1. COMPOSITION OF IN VITRO DIETS WITH VARYING DIETARY LEVELS OF POTASSIUM AND VARYING IONOPHORES

	IFN	Diet ^a							
		B	M	S	L	K	KM	KS	KL
		----- Percentage of Dry Matter -----							
Dry rolled sorghum	4-20-894	63.22	63.20	63.20	63.20	54.75	54.73	54.73	54.73
Sun-cured prairie hay	1-03-191	30.23	30.23	30.23	30.23	30.23	30.23	30.23	30.23
Cottonseed meal	5-01-621	5.48	5.48	5.48	5.48	5.48	5.48	5.48	5.48
Trace mineralized salt		.50	.50	.50	.50	.50	.50	.50	.50
Limestone	6-02-632	.57	.57	.57	.57	.57	.57	.57	.57
Potassium chloride	6-03-755	.00	.00	.00	.00	8.46	8.46	8.46	8.46
Monensin		.00	.02	.00	.00	.00	.02	.00	.00
Salinomycin		.00	.00	.02	.00	.00	.00	.02	.00
Lasalocid		.00	.00	.00	.02	.00	.00	.00	.02

^aBasal (B) = .56% potassium.

Monensin (M) = .56% potassium and 27 g/ton monensin.

Salinomycin (S) = .56% potassium and 11 g/ton salinomycin.

Lasalocid (L) = .56% potassium and 33 g/ton lasalocid.

Potassium (K) = 4.97% potassium.

Potassium and Monensin (KM) = 4.97% potassium and 27 g/ton monensin.

Potassium and Salinomycin (KS) = 4.97% potassium and 11 g/ton salinomycin.

Potassium and Lasalocid (KL) = 4.97% potassium and 33 g/ton lasalocid.

TABLE 2. ORGANIC MATTER (OM) DIGESTION OF IN VITRO FLASKS

Item	Diets								SE ^b	Probability of K Interaction with			Main Effects ^a
	B	M	S	L	K	KM	KS	KL		Ion	M vs. S&L	S vs. L	
OM intake, g/d	.856	.852	.856	.858	.782	.783	.783	.785					
OM output, g/d	.579	.507	.517	.549	.472	.523	.493	.496	.014	.002	.039	.302	TRX
OM digestion, %	32.4	40.5	39.6	36.0	39.5	33.2	37.0	36.9	1.73	.002	.048	.326	

^aTRX = Potassium (K) vs. Basal (P<.05).

A = Ionophore (Ion) vs. No Ionophore (P<.05).

B = Monensin (M) vs. Lasalocid (L) and Salinomycin (S) (P<.05).

^cC = Lasalocid (L) vs. Salinomycin (S) (P<.05).

^bStandard error of the means.

TABLE 3. VFA CONCENTRATIONS OF IN VITRO FLASKS

Item	Diets								SE ^b	Probability of K Interaction with			Main Effects ^a
	B	M	S	L	K	KM	KS	KL		Ion	M	S	
											vs. S&L	vs. L	
Total VFA's, mM/l	23.0	30.5	31.1	30.9	26.8	23.2	26.4	27.5	1.82	.008	.326	.704	TRX,A
Acetate, %	59.6	58.3	53.1	48.7	60.3	57.3	53.6	52.8	.54	.579	.003	.003	TRX,A,B,C
Propionate, %	27.8	29.3	37.7	40.3	26.4	31.0	35.1	37.0	.57	.949	.000	.497	TRX,A,B,C
Butyrate, %	12.4	11.9	10.7	10.9	12.6	11.5	11.1	10.2	.23	.730	.827	.463	A
Acetate:propionate	2.1	2.0	1.4	1.2	2.3	1.8	1.5	1.4					

^aTRX = Potassium (K) vs. Basal (P<.05).

A = Ionophore (Ion) vs. No Ionophore (P<.05).

B = Monensin (M) vs. Lasalocid (L) and Salinomycin (S) (P<.05).

C = Lasalocid (L) vs. Salinomycin (S) (P<.05).

^bStandard error of the means.

TABLE 4. NITROGEN (N) MEASUREMENTS AND MICROBIAL NUMBERS

Item	Diets								SE ^b	Probability of K Interaction with			Main Effects ^a
	B	M	S	L	K	KM	KS	KL		Ion	M vs. S&L	S vs. L	
N intake, g/d	.016	.016	.016	.016	.015	.015	.015	.014					
Ammonia-N, mg/dl	.086	.098	.125	.117	.093	.105	.097	.106	.01	.180	.066	.315	
Bacteria x 10 ¹⁰ /ml	2.28	.65	.78	.79	1.61	1.07	.81	.70	.12	.001	.038	.548	A
Protozoa, x10 ⁴ /ml	1.73	2.06	3.21	1.40	1.48	1.48	.33	.82	.55	.236	.241	.051	TRX
Ribonucleic Acid N, % of DM	.071	.057	.042	.047	.061	.060	.070	.063	.01	.027	.114	.416	A

^aTRX = Potassium (K) vs. Basal (P<.05).

A = Ionophore (Ion) vs. No Ionophore (P<.05).

B = Monensin (M) vs. Lasalocid (L) and Salinomycin (S) (P<.05).

C = Lasalocid (L) vs. Salinomycin (S) (P<.05).

^bStandard error of the means.

TABLE 5. VARIOUS MEAN DIGESTIVE MEASUREMENTS OF IN VITRO FLASKS

Item	Diets								SE ^b	Probability of K Interaction with			Main Effects ^a
	B	M	S	L	K	KM	KS	KL		Ion	M vs. S&L	S vs. L	
Gas production, mm	16.1	26.7	19.1	14.9	14.9	9.7	12.1	12.8	4.35	.310	.117	.585	TRX
Lactate production, pH change at													
8 h vs. 1 h	-.08	-.11	-.03	-.06	-.05	-.12	-.10	-.10	.04	.363	.604	.822	
24 h vs. 1 h	-.26	-.16	-.14	-.06	+.03	-.18	-.14	-.21	.11	.076	.748	.503	
Buffering capacity, ml to bring pH to 5.0	39.3	37.8	37.5	37.7	38.2	38.6	38.2	37.6	.28	.003	.406	.233	A
pH	6.75	6.71	6.67	6.68	6.72	6.75	6.71	6.70	.02	.029	.716	.620	A, B

^aTRX = Potassium (K) vs. Basal (P<.05).

A = Ionophore (Ion) vs. No Ionophore (P<.05).

B = Monensin (M) vs. Lasalocid (L) and Salinomycin (S) (P<.05).

C = Lasalocid (L) vs. Salinomycin (S) (P<.05).

^bStandard error of the means.

TABLE 6. POTASSIUM AND SODIUM MEASUREMENTS OF IN VITRO FLASKS

Item	Diets								SE ^b	Probability of K Interaction with			Main Effects ^a
	B	M	S	L	K	KM	KS	KL		Ion	M	S	
											vs. S&L	vs. L	
K Intake, g/d	.027	.026	.026	.027	.068	.069	.067	.059					
K output of flasks, g/d	.024	.024	.024	.023	.055	.055	.054	.052	.001	.524	.568	.658	TRX
K output of liquid, %	89.7	91.8	89.7	85.0	81.3	79.1	80.0	87.0	2.25	.671	.038	.019	TRX
Na intake, g/d	.387	.387	.387	.387	.388	.389	.388	.387					
Na output of flasks, g/d	.308	.289	.289	.285	.288	.279	.284	.281	.01	.308	.701	.965	
Na output of liquid, %	79.7	74.8	74.5	73.7	74.2	71.8	73.0	72.5	2.04	.304	.633	.944	A
K:Na intake ratio	.070	.067	.068	.069	.176	.178	.172	.153					
K:Na output ratio	.079	.083	.082	.080	.194	.196	.189	.183	.01	.500	.545	.802	TRX

^aTRX = Potassium (K) vs. Basal (P<.05).

A = Ionophore (Ion) vs. No Ionophore (P<.05).

B = Monensin (M) vs. Lasalocid (L) and Salinomycin (S) (P<.05).

C = Lasalocid (L) vs. Salinomycin (S) (P<.05).

^bStandard error of the means.

CHAPTER VI

EFFECT OF SUPPLEMENTAL POTASSIUM AND MONENSIN ON RUMINAL DIGESTION AND PASSAGE RATES

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Summary

Four ruminally cannulated cattle (457 kg) were fed 63% milo diets in a 4 x 4 latin square with a 2 x 2 factorial arrangement of treatments to determine the effects of K level and ionophore on ruminal digestion and in situ disappearance of corn or soybean meal. Treatments included two levels of K [.86% or .64% (basal)] with and without monensin (27 ppm). Buffering capacity and ruminal concentrations of Na, K, and total volatile fatty acids were not affected by treatment. Potassium supplementation depressed the molar percentage of isobutyrate ($P < .05$) and increased ruminal liquid passage rates by 15% ($P < .07$). Monensin depressed ruminal ammonia-N ($P < .03$), and the molar percentages of acetate ($P < .06$), butyrate ($P < .06$), and valerate ($P < .01$) while increasing molar ratios of propionate and isovalerate ($P < .01$). Particulate passage rates of Yb-labeled milo and Dy-labeled prairie hay tended to be faster when monensin was fed. In situ dry matter disappearance of corn was not affected ($P > .05$) by treatment. In situ disappearance of dry matter and nitrogen from soybean meal tended to increase with the monensin addition to the diet, possibly due to the higher ruminal pH with monensin

supplementation. Ruminal pH was correlated positively with linear digestion rates of soybean meal dry matter ($r=.48$; $P<.06$) and nitrogen disappearance ($r=.67$; $P<.005$). The only K x monensin interaction ($P<.05$) detected in vivo was a reduction in the percentage valeric acid produced with addition of both K and monensin.

(Key Words: In Situ, Potassium Chloride, Monensin, Feedlot, Rate of Passage)

Introduction

Monensin is an ionophore approved for use in cattle feeds at levels between 5 and 30 g per ton of complete feed (90% dry matter basis). Generally, monensin improves feed efficiency, depresses feed intake and has little effect on the daily gain of feedlot cattle. The ratio of volatile fatty acids produced in the rumen is altered with monensin, with an increased proportion being propionate. The quantity of dietary protein that escapes degradation in the rumen and retention of certain minerals both have been increased by monensin feeding suggesting that it modifies protein and mineral availability (NRC, 1984).

Ionophores form lipid soluble complexes with certain cations and facilitate cation transport across biological membranes. Thus, interactions between dietary cations and ionophores might be expected. Recent pure culture studies indicate that at increased K concentrations, the selective antimicrobial activity of monensin and lasalocid is reduced (Dawson and Boling, 1983b). Lemenager et al. (1978) and Spears and Harvey (1985) noted increased levels of K in the rumen of steers with monensin and lasalocid feeding; whereas, Starnes et al. (1984) observed that ruminal K levels decreased in steers fed monensin or lasalocid. Kirk et al. (1985) reported no significant change in ruminal

fluid K levels when feeding monensin to lambs.

Though K supplementation to a level of 1.0% of the diet generally increases performance, in one study with monensin diets, supplemental K decreased weight gain in cattle but did not significantly alter feed efficiency, ruminal acid levels, and ammonia concentrations (Ferrell et al., 1983). In a lamb trial by Greene et al. (1985), monensin decreased the acetate:propionate ratio, and additional K did not affect this ratio. Kelly and Preston (1985) concluded that the maintenance requirement for K by growing steers was not altered by monensin. However, Kirk et al. (1985) indicated that monensin altered Na and K metabolism in ruminants, presumably a result of monensin-induced effects. Schelling (1984) noted that monensin ruminal responses mimic the response observed from a decreased rumen turnover and indicated that monensin may decrease ruminal passage rate.

The objectives of this study were to determine the interactions between dietary K level and monensin addition on ruminal digestion and to investigate the effect of K and monensin on passage rates of ruminal fluid and particles.

Materials and Methods

Two Angus-Hereford heifers (474 kg) and two Brahman-Hereford cross steers (440 kg), fitted with large ruminal cannulas, were used in a 4 x 4 latin square with a 2 x 2 factorial treatment design to investigate the influence of K level on the metabolic action of monensin in feedlot animals. Each animal was fed a 70% concentrate, complete mixed diet. Four diets (Table 1) were fed: the basal (B) diet (calculated to provide .57% endogenous K); the basal diet plus 27 ppm monensin (M); the

basal diet plus supplemental potassium (K) from potassium chloride to provide .95% K; and the basal ration supplemented with both K and monensin (KM) (with .95% K and 27 ppm monensin). The K analysis was .65%, .87%, .62%, and .85% K for the B, K, M, and KM diets, respectively. Animals were stalled in metabolism crates and fed twice daily at 0800 and 2000 h. Daily dry matter intake of each animal was restricted to 1.25% of the animal's initial shrunk weight.

Periods lasted 21 days with sampling on days 19-21 of each of the 4 periods. Feed samples were collected on days 19-21 and composited within each diet and period. On day 19, rumen samples were withdrawn via cannula before feeding and 1, 3, 6, and 9 h post-feeding. The pH was measured; then rumen fluid was strained through four layers of cheesecloth and 1 ml of 20% H_2SO_4 was added per 100 ml strained fluid. For ammonia and VFA analyses, 100 ml of the strained rumen fluid was refrigerated until 45 g of each sample was centrifuged at 10,000 g for 10 minutes. The supernatant, decanted through two layers of cheesecloth, was frozen and later analyzed for ammonia-nitrogen (Broderick and Kang, 1980). One ml of 25% 2-ethylbutyric acid, an internal standard, was added to 5 ml of the thawed supernatant. This solution was centrifuged at 25,000 g for 20 minutes, and volatile fatty acid concentrations were determined gas chromatographically (Sharp et al., 1982).

To estimate buffering capacity, 40 ml of strained rumen fluid obtained 3 h postfeeding was increased to pH 7.0 by the addition of .1 N NaOH. Buffering capacity was measured by titrating this mixture with .1 N HCl in pH increments of .5 to a final pH of 3.

For Na analyses, an aliquot of the rumen fluid supernatant was

diluted with a 2000 ppm KCl solution. The same solution was used to prepare standards of 0, .2, .4, .6, .8, and 1.0 ppm Na from NaCl. Sodium concentrations were determined by regression of the atomic absorption readings against the standard curve. For K analysis, another aliquot of the supernatant was diluted with a 2000 ppm NaCl solution. The samples and standards (0, .5, 1.0, 1.5, 2.0 and 2.5 ppm KCl) diluted with the same NaCl solution were analyzed by atomic absorption. Regression analysis was used to calculate the K concentration of the samples.

To determine effects on ruminal digestion rate, beginning day 19, one blank dacron bag and duplicate dacron bags containing 1.5 g of soybean meal or corn ground through a 2mm screen were suspended in the rumen for 4, 12, or 24 h for each of the diets. Bag construction, incubation, and washing procedures were described by Weakley et al. (1983) and included securing the bags with plastic covered steel wire on a weighted string. After removal from the rumen, the bags were washed for 4 min, dried for 24 h at 105 C, weighed, and Kjeldahl nitrogen (AOAC, 1975) was determined on each blank bag and each bag plus its soybean meal residue to determine nitrogen (N) disappearance.

For estimating passage rates from the rumen, whole rumen fluid was sampled at 0800 on day 20, and 150 g of ytterbium-labeled milo and 100 g of dysprosium-labeled prairie hay were added to the diet for each animal. A fluid marker (100 ml of CoEDTA plus 150 ml distilled water) was then intraruminally dosed. Whole rumen fluid samples were withdrawn 6, 12, 18, 24, 30, and 36 hours post-dosing. Dry matter was calculated on a measured volume of each whole rumen fluid sample dried for 48 h in a 60 C oven to determine the weight:volume ratio.

Feed and dried rumen samples were ground in a Wiley mill fitted with a 2 mm screen and were stored at room temperature for future analysis. Feed samples were analyzed for dry matter (DM; 105 C for 24 h), ash (600 C for 12 h), Kjeldahl nitrogen (N; AOAC, 1975), starch (MacRae and Armstrong, 1968), and Na and K concentrations (as described in Chapter IV).

Ground rumen samples were analyzed for dry matter (DM; 105 C for 24 h) and ash (600 C for 12 h). Two grams of the rumen samples were prepared as described by Ellis et al. (1982) and Co, Yb and Dy concentrations were determined by atomic absorption spectroscopy.

Treatment means were compared by a general linear model program for a 4 x 4 latin square designed as a 2 x 2 factorial arrangement of treatments (Statistical Analysis System, 1982). Treatment effects were divided to test effects of K level, ionophore level, and the interactions of these two factors. Classes in the model therefore included steer effects, period effects, K effect, ionophore effect, and the K x ionophore interaction. In situ digestion rates were calculated by regressing either the amount of residual nutrient or the natural logarithm of this value against ruminal exposure time. Simple correlations among certain factors were calculated by linear regression of the two factors.

Results and Discussion

Intakes of various nutrients are presented in Table 2. Feed intake was limited and equal for all diets. Differences in starch and Na intake primarily reflect feed sampling errors. Sodium intake was 30% greater ($P < .05$) with the K or K-monensin diets. Since monensin was

added in the form of monensin sodium, a small portion of the added Na would come from this combination. The commercial KCl added was estimated to contain between 15000 and 25000 ppm Na. Part of the discrepancy in Na intake may be due to variation in the Na content of the added KCl. The dietary K:Na ratio was not affected by treatment but tended to be greater with the KCl-supplemented diets.

The means for the K and Na concentrations of the rumen fluid (Table 3) were averaged over 5 sampling times. Rumen fluid concentrations of cations did not differ significantly with diet ($P > .05$) though ruminal K concentration tended to be greater with elevated K intake. Spears and Harvey (1985) observed that increasing dietary K levels in the presence of lasalocid increased the soluble K concentrations in the rumen fluid of steers. The surprising consistency in ruminal concentrations of K and Na across all treatments may reflect recycling of these ions via the saliva or through the rumen wall. Parthasarathy (1952) noted an influx of ions into the rumen when the K and Na concentrations of the rumen were lower than the blood concentrations. In this trial, blood concentrations of minerals was not measured. Recycling of Na and K to the rumen was equal to 19 to 26 g Na and 3 to 10 g K daily in another study (Doran et al., 1985). In the current trial, the amount K recycling into the rumen was calculated to be from 65 to 88 g daily.

Monensin supplementation did not alter ($P > .05$) ruminal concentrations of either K or Na. Previous reports concerning the effects of monensin supplementation on ruminal fluid K levels have been contradictory. Lemenager et al. (1978) found that monensin supplementation increased ruminal fluid K concentration by 21.6%, while Starnes et al. (1984) noted that monensin supplementation decreased the

ruminal K concentrations of steers. Kirk et al. (1985) reported no significant differences could be attributed to monensin supplementation. In all of these above studies, ruminal fluid Na levels did not appear to be altered by monensin supplementation.

The ruminal K:Na ratio did not differ ($P>.05$) among treatments, but as the ruminal K levels increased, ruminal Na levels tended to decrease. Ruminal fluid concentrations of K and Na were inversely related ($r=-.96$; $P<.0001$). Spears and Harvey (1985) reported that dietary K added in the presence of lasalocid reduced ruminal fluid Na concentrations. This tendency was apparent in our study, also.

Liquid passage rate tended to be faster ($P<.07$) by 15% with K supplementation. Ruminal outflow was correlated positively with the ruminal ratio of K:Na ($r=.47$; $P<.07$). Fluid dilution rate and particulate passage rate for Yb-labeled milo were related positively ($r=.59$; $P<.02$). Potassium intake may have increased urinary K excretion which in turn increased water intake and rumen fluid dilution rate. Urinary K excretion was not measured in this trial. Ward (1966) suggested that fluid passage rate might be increased by dietary K level, and Schneider et al. (1984) noted that fluid passage rates in hyperthermic dairy cows increased with addition of K to levels greater than 1.7% of the diet. Whether the increased passage rate in the cows was due to greater water intake or reduced rumen volume was not determined in their study. In the current study, ruminal K:Na ratio was positively correlated with rumen volume ($r=.52$; $P<.04$). Thus, the faster rate of passage may have been due to increased water intake as ruminal outflow (Figure 1) was related positively with rumen volume ($r=.53$, $P<.03$).

Solid passage rate was estimated independently for milo and prairie hay. Passage of both feed ingredients tended to increase with K supplementation with the fastest passage with the K-monensin diet, and solids passage rate tended to increase with added monensin. This contradicts previous reports (Schelling, 1984; Ricke et al., 1984) in which monensin decreased passage rates of both the liquids and solids. Differences in passage rates among these trials may be due to additional factors such as rumen volume or K:Na ratios in the rumen. Particulate passage rate for milo and the ruminal K:Na ratio were negatively correlated ($r=-.52$; $P<.04$).

Estimated rumen volumes in this trial tended to be reduced with monensin supplementation ($P<.12$). Lemenager et al. (1978) observed a reduction in rumen volume with steers fed harvested low quality range grass plus monensin, but no change in steers fed concentrate diets with monensin. Ricke et al. (1984) also noted that while rumen volumes of lambs did not differ significantly, they tended to be reduced with monensin. In the current trial, as rumen volume tended to increase (Figure 2) the particulate passage rates of milo and prairie hay tended to decrease ($r=-.45$; $P<.08$ and $r=-.29$; $P<.28$). The correlation between fluid passage rate and rumen volume was not strong ($r=-.16$; $P<.56$).

With all four diets, the passage rate of Yb-labeled milo was slower than Dy-labeled prairie hay. This is inconsistent with the particle size model of Poppi et al. (1980) in which small particles exit most rapidly from the rumen. One explanation for this deviation is lodging of milo particles in the ruminal raft which could lower the passage rate constant for milo. Marker migration also could be involved.

Ruminal ammonia-N levels were lower ($P<.03$) with the monensin-

supplemented diets. Ricke et al. (1984) noted that rumen ammonia-N levels were reduced by 17% with monensin feeding of sheep. Monensin has been reported to reduce the ruminal degradation of dietary protein and decrease the rate of degradation of free amino acids in rumen fluid. Ruminal ammonia decreases are consistent with depressed deamination and proteolysis (Schelling, 1984). No K x monensin interaction was evident ($P > .05$) for ruminal ammonia. This agrees with data of Funk et al. (1985) who noted no K x lasalocid interactions for ruminal escape of feed N, nitrogen balance, or efficiency of microbial protein synthesis in the rumen of lambs. Mean ruminal ammonia-N levels and ruminal pH were negatively correlated ($r = -.47$; $P < .07$). At higher pH levels, as occurred with monensin supplementation, ruminal ammonia-N is more rapidly absorbed from the rumen. Hence, ruminal ammonia-N levels would be reduced. The other possibility is that less NH_4^+ diffuses into the rumen with a higher pH in the rumen, which in turn reduces ruminal ammonia-N levels.

Ruminal pH tended to be higher with monensin supplementation ($P < .11$). This may be due to decreased production of volatile fatty acids or lactate. The pH values across all diets were higher than expected for a 70% concentrate diet and may be attributed to the low feed intakes which reduce the ruminal VFA concentrations.

Buffering capacity was calculated as the ml of .1 N HCl needed to titrate 40 ml of rumen fluid from a high (7) to a low, yet physiological pH (5). Buffering capacity was not altered by treatment ($P > .05$). Treatments with the higher initial pH and lower volatile fatty acid concentrations tended to have the lower buffering capacities. Mean ruminal pH and buffering capacity were negatively related ($r = -.56$;

$P < .02$); whereas, total volatile fatty acid concentration and buffering capacity were positively correlated ($r = .49$; $P < .05$). This tends to refute the theory that added K acts to buffer digesta. Instead, ruminal pH and buffering capacity appear attributable primarily to VFA concentrations within a level of dietary concentrate.

Total volatile fatty acid concentration (Table 4) was similar ($P > .05$) across treatments. Lemenager et al. (1978), Ferrell et al. (1983), Ricke et al. (1984), and Kirk et al. (1985) also reported no differences in total ruminal volatile fatty acid concentrations with monensin supplementation. However, ratios were altered with monensin supplementation. The molar proportion of propionate increased ($P < .01$) by 31.3% with the monensin supplementation while molar proportions of acetate and butyrate tended ($P < .06$) to decrease (4.6% and 18.3%). These results agree with previous reports (Lemenager et al., 1978; Ricke et al., 1984; Greene et al., 1985; Kirk et al., 1985).

Addition of K did not significantly alter ($P > .05$) the acetate:propionate ratio similar to the results of Greene et al. (1985). However, K supplementation decreased the molar percentage of isobutyrate. Monensin supplementation also decreased the molar proportion of valerate ($P < .01$) which supposedly is derived from propionate and valine and increased the molar percentage of isovalerate ($P < .05$) which is derived from isoleucine. A K x monensin interaction further depressed the molar proportion of valerate ($P < .04$). These alterations may reflect protein degradation differences which would be increased with monensin and decreased with K. The molar proportion of isobutyrate was positively related with fluid passage rate and ruminal ammonia-N ($r = .45$; $P < .08$; and $r = .50$; $P < .05$). Sharp et al. (1982) also

noted an increased concentration of isobutyric, acetic, butyric, and valeric acid with a faster rate of passage of whole than ground corn in steers, but the faster passage rate decreased the concentration of propionate.

Potential dry matter and nitrogen digestibility in the rumen from in situ data were calculated two ways. Logarithmic and linear intercepts and slopes of the residues were computed for each steer on each treatment. These values for each steer were compared using the general linear model program to determine treatment differences. Values are reported in Table 5 are classified by calculation method. Logarithmic values of the digestible DM available are reported as the antilogs. The linear slopes of the residue components are negative, reflecting disappearance of residue over time. In all cases, the digestible dry matter and digestible nitrogen calculated logarithmically were greater than the linear values. This was especially noticeable with the nitrogen values. It is physically impossible to have greater than 100% initially; hence, values exceeding 100% reflect a lag time in digestion.

In situ dry matter disappearance of corn (Table 5) at various incubation times was not affected by diet ($P > .05$). Dry matter disappearance from ground corn at 12 hours was related positively with liquid and solids passage rates ($r = .57$, $P < .02$; and $r = .46$, $P < .07$). Liquid and solids (Yb) passage rates were positively correlated ($r = .59$; $P < .02$). The K-containing diets tended to have the fastest fluid passage rates and in turn, the greater disappearance of corn at 12 hours. Possibly increased water intake or water movement into the rumen with added K tended to remove the soluble corn residues at a faster rate.

After 24 hours of incubation, disappearance remained weakly related with solids passage rate ($r=.25$, $P<.36$). The zero time intercept was about 25% (Figure 3) suggesting that soluble residues and small particles were lost rapidly the first four hours leaving only the less soluble and larger particle fractions to be digested thereafter.

Dry matter disappearance could be considered a relative index of dry matter digestibility assuming loss of dry matter parallels digestibility. Previous trials (Reffett and Boling, 1982; St. Omer and Roberts, 1967; Zinn and Owens, 1980) have detected no effect of K on in vivo dry matter digestibility. In contrast, previous research generally indicates that monensin increases in vivo dry matter digestibility (Dinius et al., 1976; Poos et al., 1979; Beede et al., 1980; Thornton and Owens, 1981). Rust et al. (1978) reported that monensin increased starch and dry matter digestibilities in cattle receiving a high grain diet which was low in protein. The discrepancy in digestion rates between in situ disappearance of this trial and previous in vivo trials may be due to several factors. First, in vivo dry matter digestion encompasses starch, nitrogen, and ADF. Corn is primarily starch, so in situ disappearance primarily reflects starch digestion. Yet, different components are being considered. Secondly, previous trials have primarily reported total tract dry matter digestibility while this trial relates only to ruminal dry matter digestibility. Earlier trials with monensin have yielded contradictory results of ruminal dry matter digestibility. Muntifering et al. (1981) noted monensin decreased apparent ruminal digestion of starch by 19%. This contrasts with Zinn et al. (1980) who noted monensin supplementation (150 mg/head/day) increased ($P<.05$) ruminal starch digestion by 24%.

Dry matter (Figure 4) and nitrogen disappearance (Table 5) of soybean meal were not significantly altered by diet ($P>.05$), though ruminal digestion of nitrogen (Figure 5) tended to be increased with addition of monensin to the diet ($P<.07$). This directly contradicts most earlier research in which monensin has been reported to reduce ruminal digestion of dietary protein and increase escape (Schelling, 1984). Usually, total flow of N to the small intestine is not increased despite the increase in flow of dietary protein. Microbial protein flow generally decreases. This change could be caused by compositional changes of ruminal microbes since microbial flow relies on markers to estimate microbial protein.

One explanation for the increased disappearance of soybean meal nitrogen with monensin supplementation is ruminal pH. Mean ruminal pH was correlated positively with dry matter and nitrogen disappearance of the soybean meal at 24 hour ruminal incubation ($r=.63$; $P<.009$ and $r=.71$; $P<.002$). The digestion rate of soybean meal residue and N residue were related positively with ruminal pH ($r=.48$; $P<.06$ and $r=.67$; $P<.005$). Okeke et al. (1983) reported a strong positive correlation between ruminal pH four hours after feeding and nitrogen disappearance from soybean meal placed in nylon bags and suspended in the rumen 24 hours. Loerch et al. (1983) reported that an increase in the pH of McDougall's artificial saliva from 5 to 7 resulted in a 3.6-fold increase in N solubility for soybean meal and that the decreased degradation of soybean meal in the rumen of animals fed high concentrate diets is due primarily to the resulting decrease in rumen pH. Whether the interaction of pH with protein degradation is due to (1) altered solubility of the feed protein, (2) reportioning of microbial

species, or (3) altered proteolytic activity of existing species remains undetermined (Weakley, 1983). A second explanation for increased disappearance of soybean meal nitrogen with monensin supplementation is the retention time. The particulate passage rates of the monensin-containing diets were greater. If the increased pH alters solubility, and the particulate passage rate of monensin-containing diets is increased, then the increased N disappearance of the soybean meal would be possible. Nitrogen digestibility at 24 hours was positively correlated with the passage rates of Yb-labeled milo and Dy-labeled prairie hay ($r=.53$; $P<.04$ and $r=.45$; $P<.08$).

In conclusion, results from this study indicate that K did not alter the effect of monensin on measured ruminal factors. Though some of the available literature indicates an effect of ionophores on K or vice versa (Lemenager et al., 1978; Starnes et al., 1984; Spears and Harvey, 1985; Kirk et al., 1985), other researchers (Ferrell et al., 1983; Funk et al., 1985; Greene et al., 1985; Kelly and Preston, 1985) have detected no K by ionophore interaction. An interaction would be expected if monensin causes alterations in ion transport. But ionophores have additional effects, some of which can be altered by K level. In this study, these changes include ruminal pH (increased with monensin, and possibly reduced by K), ruminal fluid dilution rate (increased by K and reduced by monensin) and particulate passage rate (increased by both K and monensin). Further research to examine the magnitude of these effects and their relationship with ruminal digestion of diets should provide information to optimize efficiency of ruminant production.

TABLE 1. COMPOSITION OF FINISHING DIETS WITH VARYING AMOUNTS OF DIETARY POTASSIUM AND MONENSIN

	IFN	Diets ^{ab}			
		B	K	M	KM
Dry rolled sorghum	4-20-894	63.43	62.70	63.41	62.68
Sun-cured prairie hay	1-03-191	15.00	15.00	15.00	15.00
Cottonseed hulls	1-01-599	15.00	15.00	15.00	15.00
Cottonseed meal	5-01-621	5.50	5.50	5.50	5.50
Trace mineralized salt ^c		.50	.50	.50	.50
Limestone	6-02-632	.57	.57	.57	.57
Potassium chloride	6-03-755	.00	.73	.00	.73
Monensin		.00	.00	.02	.02

^aBasal (B) = .65% K analyzed.

Potassium (K) = .87% K analyzed..

Monensin (M) = .62% K analyzed and 27 ppm monensin.

^bPotassium and Monensin (KM) = .85% K analyzed and 27 ppm monensin.

^bDiets analyzed 32.6% starch, and 10.2% crude protein.

^cContained, as a percentage:

NaCl - not more than 97.0%.

NaCl - not less than 92.0%.

Manganese - not less than .250%.

Iron - not less than .200%.

Sulfur - not less than .030%.

Copper - not less than .033%.

Cobalt - not less than .0025%.

Iodine - not less than .007%.

Zinc - not less than .005%.

TABLE 2. DIETARY INTAKE

Item	Diets ^a			
	B	K	M	KM
Organic matter, g/d	5284	5257	5286	5266
Starch, g/d	1850	1854	1776	1671
Nitrogen, g/d	88.7	90.8	88.7	89.3
Potassium, g/d	35.60	47.30	33.94	46.43
Sodium, g/d	5.50	7.28	5.93	7.58
Dietary K:Na ratio	6.59	6.66	5.72	7.49

^aBasal (B) = .65% K.

Potassium (K) = .87% K.

Monensin (M) = .62% K and 27 ppm monensin.

Potassium and Monensin (KM) = .85% K and 27 ppm monensin.

TABLE 3. RUMINAL MEASUREMENTS

Item	Diets ^a				SE ^d	Probability of the Effects of		
	B	K	M	KM		K	Monen- sin	Inter- action
Ruminal fluid								
Potassium, ppm ^b	2447	2551	2283	2747	170	.15	.93	.33
Sodium, ppm	2018	2050	2394	1848	180	.20	.65	.16
Potassium:Na ratio	1.51	1.62	1.15	1.70	.22	.19	.55	.37
Ruminal passage rate								
Liquid, (Co) %/h	2.95	3.39	2.87	3.32	.20	.07	.71	.99
Solids								
Milo, (Yb) %/h	1.73	1.96	1.98	2.24	.22	.32	.28	.96
Prairie hay, (Dys) %/h	2.48	2.22	2.50	2.57	.29	.74	.54	.59
Rumen volume, l ^c	71.7	65.8	62.0	60.8	4.02	.41	.12	.59
Rumen outflow, l/h	2.1	2.2	1.8	2.0	.17	.38	.16	.86
Ammonia-N, mg/dl ^b	6.59	5.55	4.25	4.02	.71	.40	.03	.58
pH ^b	6.26	6.13	6.39	6.32	.08	.27	.11	.73
Buffering capacity (ml HCl needed to change from pH 7.0 to pH 5.0)	45.9	48.6	46.8	45.2	1.18	.67	.32	.12

^aBasal (B) = .65% K.

Potassium (K) = .87% K.

Monensin (M) = .62% K and 27 ppm monensin.

Potassium and Monensin (KM) = .85% K and 27 ppm monensin.

^bThese means are the average of 5 sampling times per animal.

^cRumen volumes were estimated from relative zero time intercepts of Co.

^dStandard error of the means.

TABLE 4. VOLATILE FATTY ACID MEASUREMENTS

Item ^b	Diets ^a				SE ^e	Probability of the Effects of		
	B	K	M	KM		K	Monen- sin	Inter- action
Total volatile fatty acids, mM/l	96.1	110.8	98.5	95.4	5.33	.32	.27	.15
Acetate, %	67.6	70.0	65.2	66.0	1.35	.28	.06	.59
Propionate, %	16.1	15.9	20.8	21.2	1.43	.98	.01	.83
Butyrate, %	12.9	11.0	9.8	9.8	.96	.36	.06	.35
Isobutyrate, %	.51	.43	.52	.23	.06	.02	.18	.12
Valerate, %	.92	.97	.86	.70	.04	.25	.01	.04
Isovalerate, %	1.93	1.76	2.85	2.07	.24	.10	.05	.25

^aBasal (B) = .65% K.

Potassium (K) = .87% K.

Monensin (M) = .62% K and 27 ppm monensin.

^bPotassium and Monensin (KM) = .85% K and 27 ppm monensin.

^cThese means are the average of 5 sampling times per animal.

^eStandard error of the means.

TABLE 5. EFFECT OF POTASSIUM AND MONENSIN ON RATE OF IN SITU DRY MATTER AND NITROGEN DISAPPEARANCE

Item	Diets ^a				SE ^b
	B	K	M	KM	
Corn dry matter disappearance, %					
4 hr	29.65+1.26 ^b	29.45+1.13	30.12+.85	30.14+1.41	
12 hr	39.40+.90	41.176+1.37	40.95+1.82	43.71+3.52	
24 hr	57.58+5.36	54.72+1.33	56.56+2.59	60.19+4.21	
Digestible DM available, %					
Logarithmic	80.6	76.6	77.6	79.0	1.87
Linear	76.5	74.7	75.1	75.2	1.19
Residue slopes, %/h					
Logarithmic	-2.71	-2.21	-2.42	-2.91	.48
Linear	-1.41	-1.25	-1.32	-1.49	.16
Soybean meal dry matter disappearance, %					
4 hr	38.09+1.52	36.83+.37	42.50+2.95	39.66+.75	
12 hr	51.91+3.01	56.28+3.86	64.99+4.44	63.32+4.65	
24 hr	72.49+7.26	74.79+6.19	85.53+4.03	77.27+8.40	
Digestible DM available, %					
Logarithmic	80.3	79.0	84.3	79.0	8.64
Linear	68.8	69.0	63.9	64.2	2.81
Residue slopes, %/h					
Logarithmic	-4.91	-5.12	-7.77	-6.26	1.47
Linear	-1.72	-1.87	-2.12	-1.82	.30
Soybean meal N disappearance, %					
4 hr	19.33+3.95	19.72+1.65	29.62+2.24	22.35+2.33	
12 hr	32.59+4.55	38.77+5.54	54.07+5.46	48.25+7.49	
24 hr	59.01+13.19	53.45+10.31	80.82+7.68	75.09+12.36	
Digestible N available, %					
Logarithmic	112.9	91.5	132.2	113.4	20.4
Linear	89.7	84.7	79.0	86.2	2.79
Residue slopes, %/h					
Logarithmic	-5.31	-3.28	-9.50	-7.52	1.97
Linear	-2.00	-1.65	-2.53	-2.61	.33

^bStandard error of the mean.

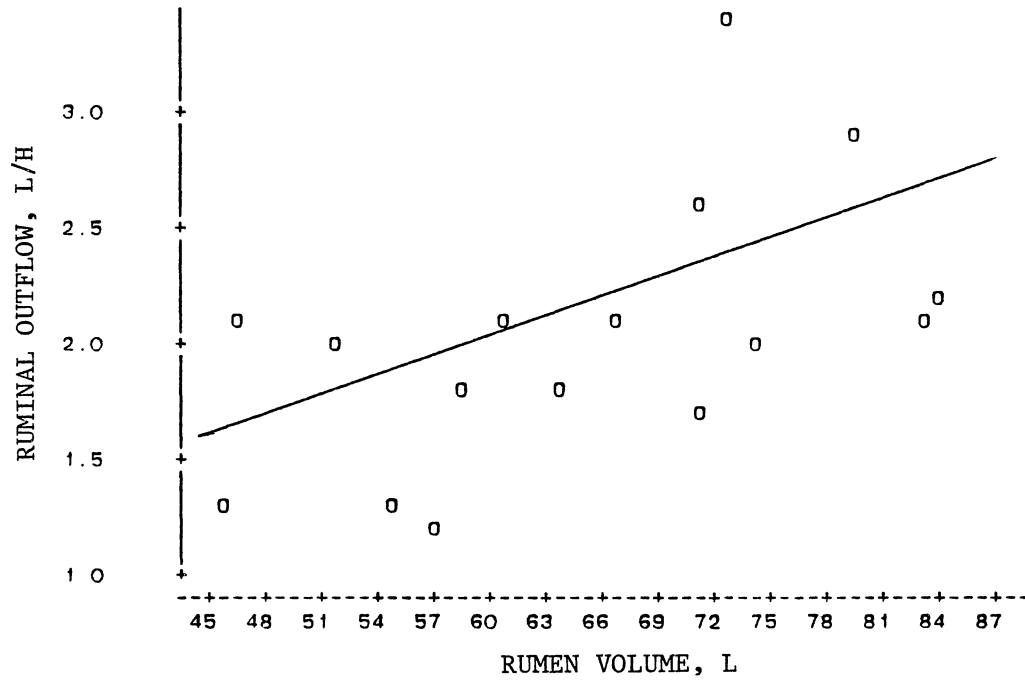


Figure 1. Ruminal Outflow (O) vs Ruminal Volume
 $O = .388 + .025 (\text{Rumen Volume, L})$; $r = .53$; $P < .03$.

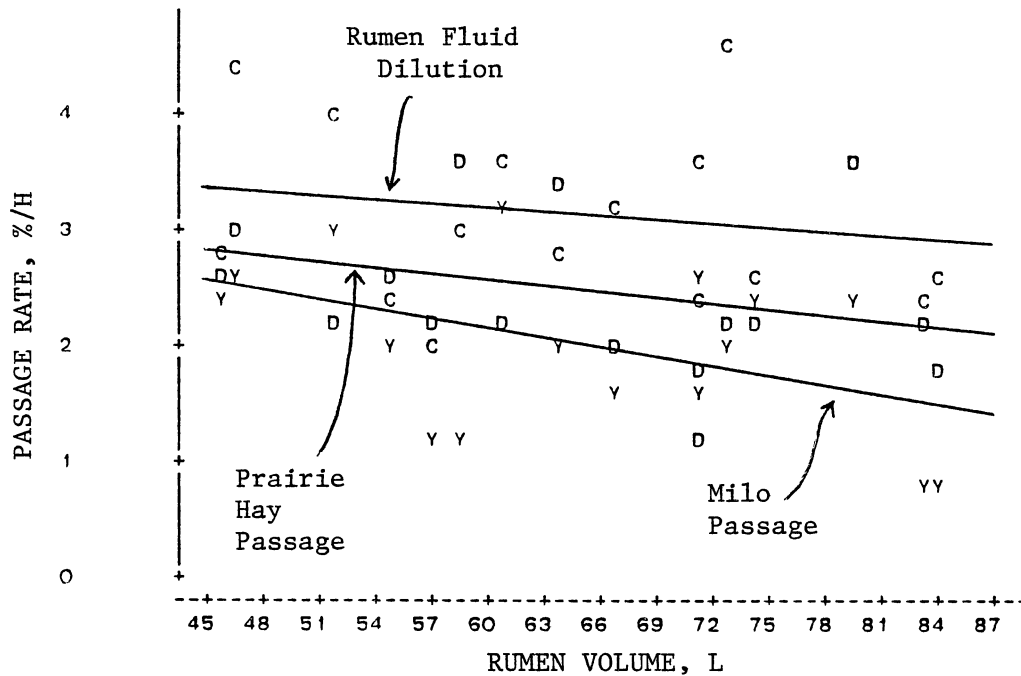


Figure 2. Rumen Fluid Dilution (C), Milo Passage Rate (Y), and Prairie Hay Passage Rate (D) vs Rumen Volume
 $C=3.782-.010$ (Rumen Volume, L); $r=-.16$; $P<.56$.
 $Y=3.758-.027$ (Rumen Volume, L); $r=-.45$; $P<.08$.
 $D=3.507-.016$ (Rumen Volume, L); $r=-.29$; $P<.28$.

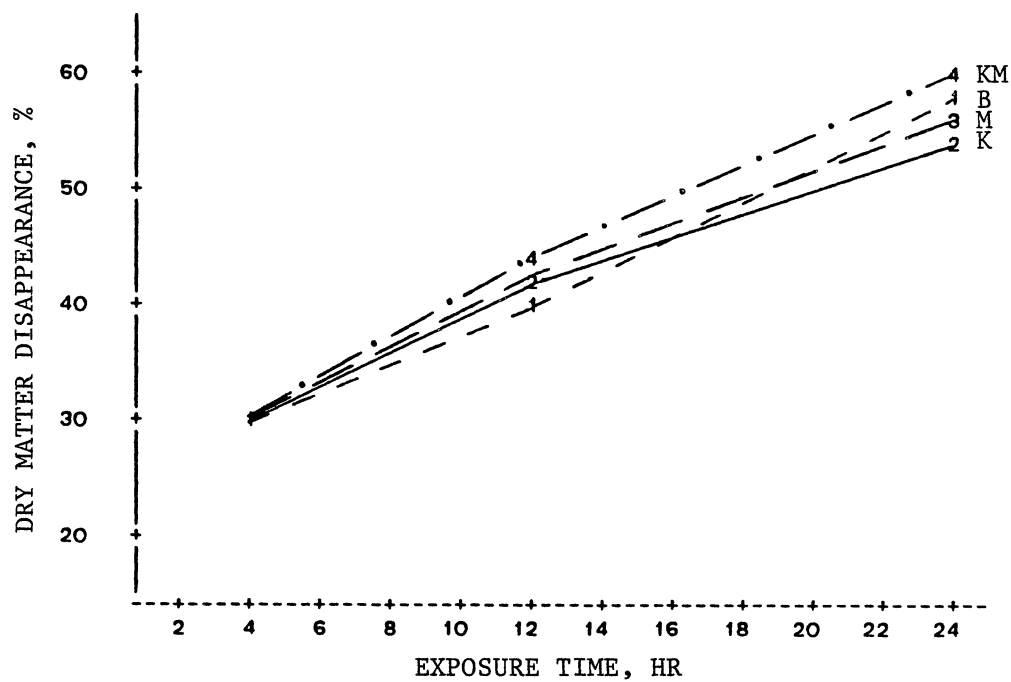


Figure 3. Dry Matter Disappearance of Ground Corn From Dacron Bags Suspended in the Rumen of Beef Animals for 4, 12, or 24 Hours

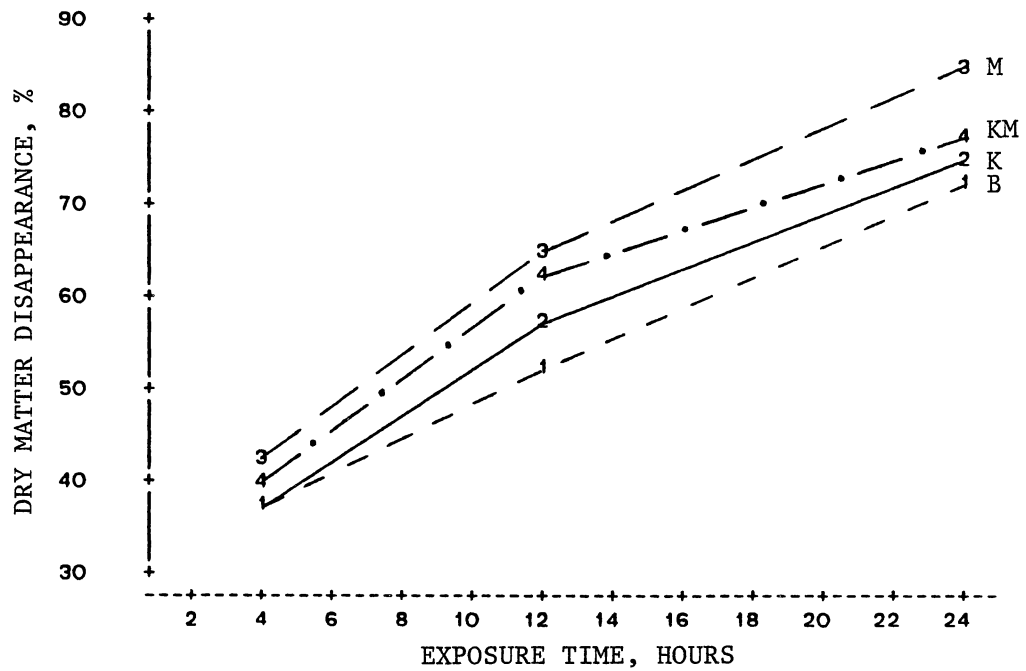


Figure 4. Dry Matter Disappearance of Soybean Meal from Dacron Bags Suspended in the Rumen of Beef Animals for 4, 12, or 24 Hours

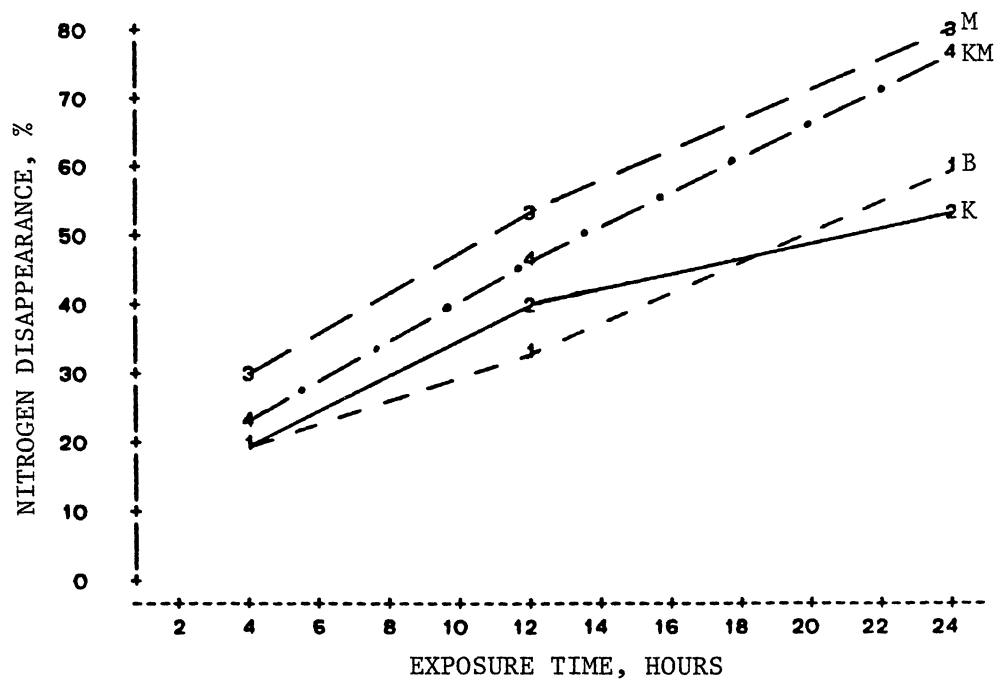


Figure 5. Nitrogen Disappearance of Soybean Meal from Dacron Bags Suspended in the Rumen of Beef Animals for 4, 12, or 24 Hours

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APPENDICES

APPENDIX A

COMPOSITION OF DIETS FED TO HEIFERS SAMPLED TO OBTAIN
IN VITRO RUMEN FLUID

	IFN	Diet	
		1st Heifer ^a	2nd Heifer ^b
		Percentage of Dry Matter	
Dry whole corn	4-02-931	83.04	83.37
Cottonseed hulls	1-01-599	10.00	.00
Sun-cured prairie hay	1-03-191	.00	10.00
Soybean meal	5-20-637	4.81	4.63
Limestone	6-02-632	1.25	1.20
Trace mineralized salt		.50	.50
Chromic oxide		.30	.30

^aDiet was fed at a level of 70 g dry matter/kg metabolic body weight.

^bDiet was fed at a level of 65 g dry matter/kg metabolic body weight.

APPENDIX B

POTASSIUM AND SODIUM STANDARD DILUTIONS

Summary

Detection of Na and K by atomic absorption with a dual lamp was tested. Sodium interfered with K analysis and vice versa. To counteract interference effects, it was determined that solutions of 2000 ppm potassium and 2000 ppm sodium should be used to dilute the samples to analyze sodium and potassium concentrations, respectively.

(Key Words: Potassium, Sodium, Interference, Atomic Absorption, Standard Dilutions)

Introduction

When the potassium and sodium analyses were begun, standard dilutions were conducted on approximately twelve samples to detect any interference in reading the absorbance of each element. Interference was detected. As the trials progressed, further problems in the analyses indicated that it was important and informative to conduct standard dilutions on the two elements. The objectives were (1) to determine the minimum concentration of the diluting solution which would optimize the atomic absorption reading and (2) to determine if increased concentrations of this diluting solution would affect these readings.

Materials and Methods

Standard concentrations of 12, 24, 36, 48, and 60 ppm sodium were prepared by diluting a stock solution of 1000 ppm sodium with deionized distilled water. One-half ml of each standard was diluted with 9.5 ml of deionized water, 500, 1000, 2000, or 4000 ppm potassium solutions. From this 10 ml, 1/2 ml of this solution was rediluted with one ml of the water and potassium solutions. The final standard concentrations of sodium were 0, .2, .4, .6, .8, and 1.0 ppm; each was diluted with final concentrations of 0, 492, 983, 1967, or 3800 ppm potassium.

In a similar fashion, interference of Na with K detection was examined. Standard concentrations of 25, 50, 75, 100, and 125 ppm potassium were prepared by diluting a stock solution of 1000 ppm potassium with deionized-distilled water. One-tenth ml of each potassium standard was diluted with 4.9 ml of distilled-deionized water, 500, 1000, 2000, or 4000 ppm sodium solutions. Final standard concentrations of potassium were 0, .5, 1.0, 1.5, 2.0, and 2.5 ppm; each was diluted with 0, 490, 980, 1960, or 3920 ppm sodium.

Duplicate samples were analyzed by atomic absorption. Data were analyzed by linear regression of optical density against the known concentration of the element present within each level of the interfering mineral using a general linear model procedure.

Results and Discussion

Both sodium and potassium are partially ionized in the air-acetylene flame which tends to reduce the absorption of incident light. The effects of ionization may be substantially overcome by the

addition of an excess (1000-2000 ug/ml) of another alkali to the samples and standards (Perkin-Elmer, 1976).

Results from the potassium standard dilutions (Figure 1) indicate that 490 ppm sodium in the diluting solution increased the slope of the potassium concentrations by 46% above the slope of diluting with water. Levels of sodium beyond 490 ppm did not appear to further shift the potassium slope (Table 1).

With the 492 ppm potassium solution, the sodium slope (Figure 2) was increased 49% above the slope of the water dilution. The sodium slope did not appear to shift beyond dilutions of 492 ppm potassium (Table 2).

For the Perkin Elmer model 4000 atomic absorption system for Na and K detection with a dual lamp and detection at 589 nm for Na and 766.5 nm for K, dilutions to 490 ppm sodium and 492 ppm potassium tended to maximize the readings and slopes for potassium and sodium. Since concentrations of Na and K vary in feeds and digesta, interference is variable which complicates interpretation of atomic absorption data. Whether similar interference occurs in other systems using different machines and samples is unknown but needs to be examined.

TABLE 1. SLOPES OF POTASSIUM CONCENTRATIONS WITH DIFFERENT CONCENTRATIONS OF SODIUM DILUTING SOLUTIONS

Concentration of Sodium Diluting Solution, ppm	Potassium Slope	Standard Error of Slope
0	.067	\pm .006
490	.098	\pm .006
980	.095	\pm .005
1960	.113	\pm .004
3920	.093	\pm .006

TABLE 2. SLOPES OF SODIUM CONCENTRATIONS WITH DIFFERENT
CONCENTRATIONS OF POTASSIUM DILUTING SOLUTIONS

Concentration of Potassium Diluting Solution, ppm	Sodium Slope	Standard Error of Slope
0	.115	<u>+.019</u>
492	.231	<u>+.007</u>
983	.214	<u>+.012</u>
1967	.211	<u>+.004</u>
3800	.228	<u>+.010</u>

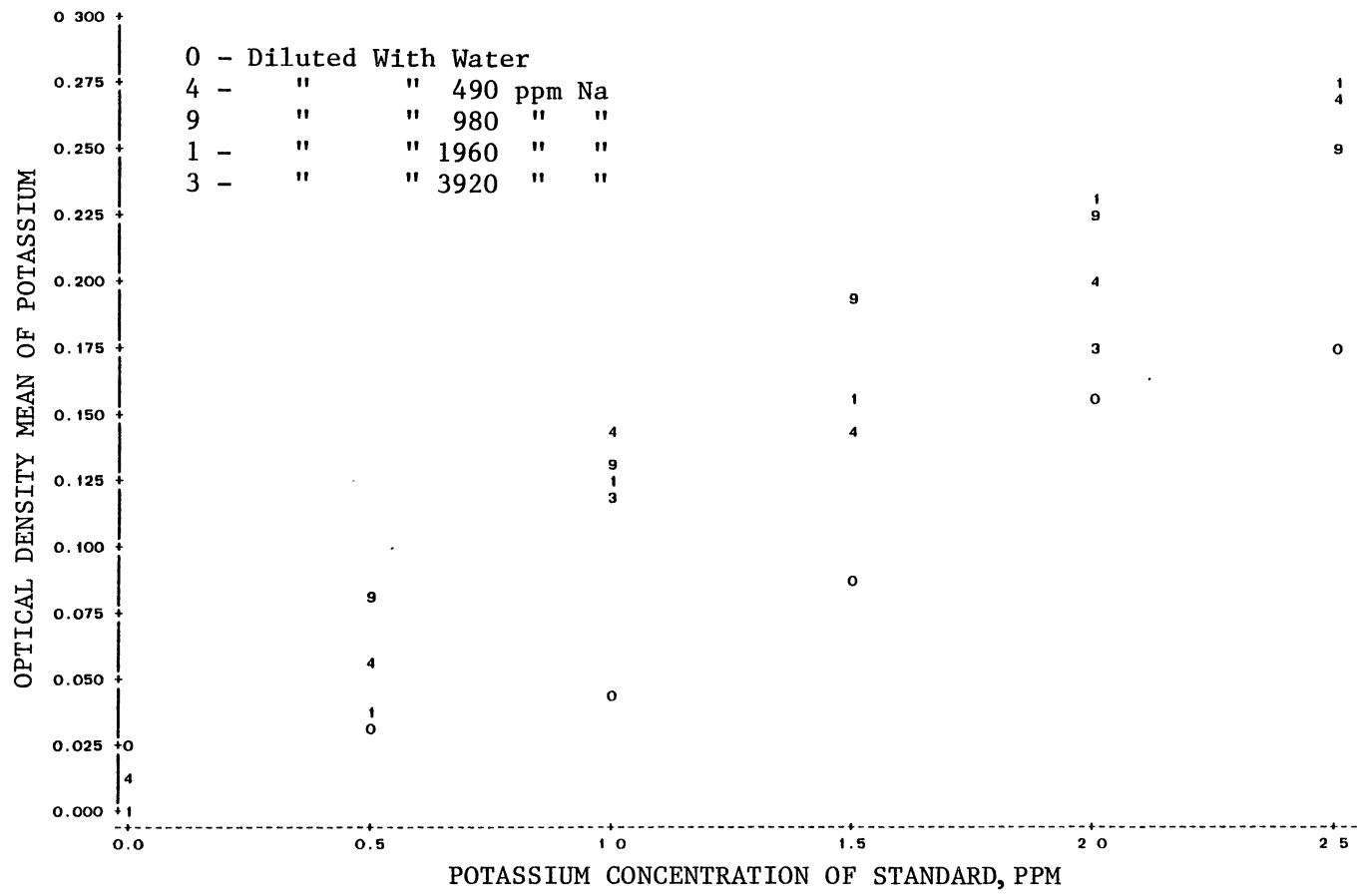


Figure 1. Optical Density Values of Potassium Standards vs Concentration of the Sodium Diluting Solution

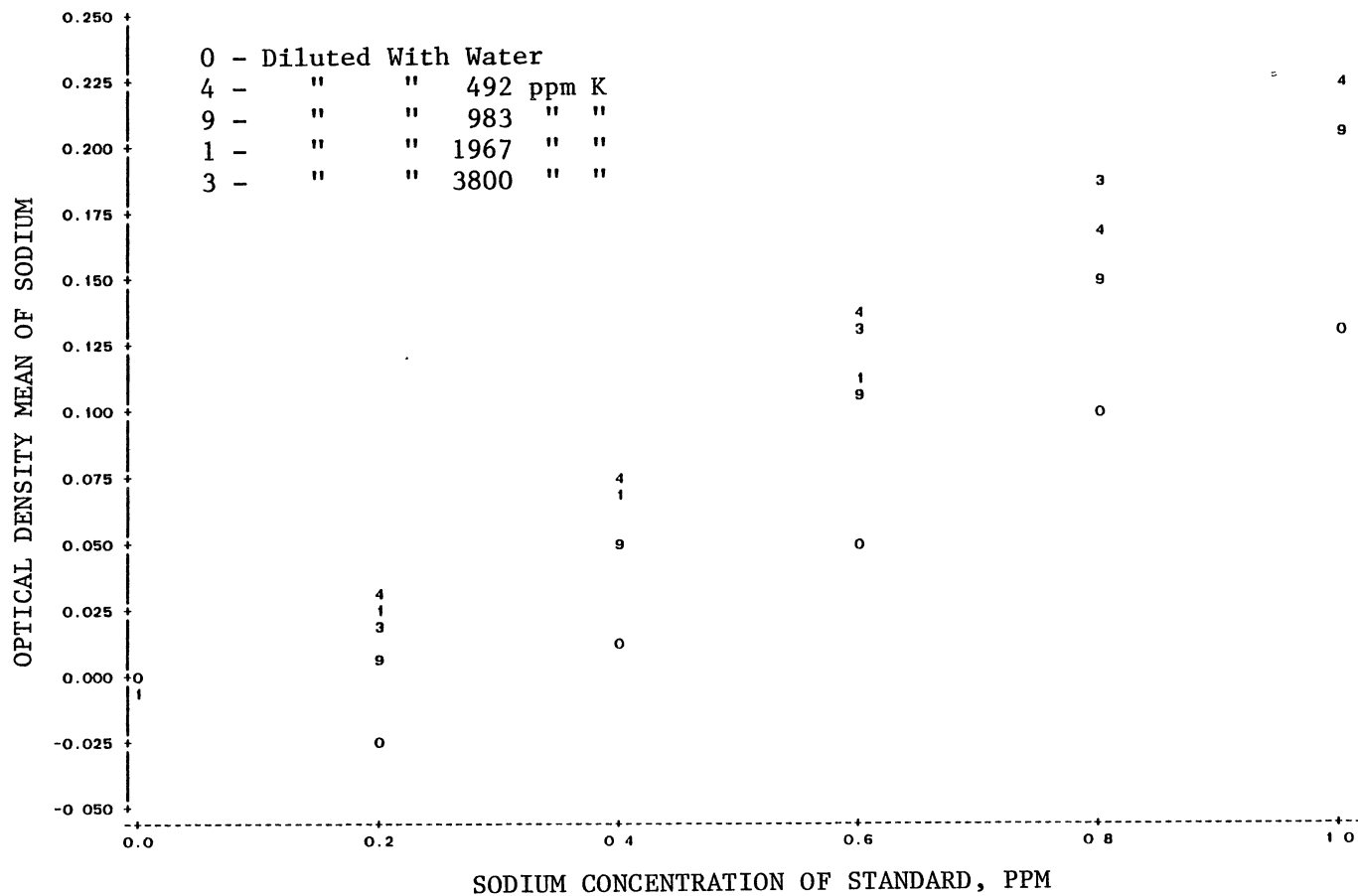


Figure 2. Optical Density Values of Sodium Standards vs Concentration of the Potassium Diluting Solution

VITA ~

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