To the Graduate Council:

I am submitting herewith a thesis written by Stephen R. Banet entitled "Effects of Potassium Fertilization of Fescue Pastures and Oral Administration of Potassium Chloride on Metabolism of Magnesium, Calcium and Potassium in Lactating Beef Cows." I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Animal Science.

M. C. Bell

M. C. Bell, Major Professor

We have read this thesis
and recommend its acceptance:

E. R. Sidwell

John H. Reynolds

Accepted for the Council:

The Graduate School

EFFECTS OF POTASSIUM FERTILIZATION OF FESCUE PASTURES AND ORAL ADMINISTRATION OF POTASSIUM CHLORIDE ON METABOLISM OF MAGNESIUM, CALCIUM AND POTASSIUM IN LACTATING BEEF COWS

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

Stephen R. Banet

December 1983

Ag-VetMed
Thesis
83
.B262

ACKNOWLEDGMENTS

The author wishes to express his gratitude and appreciation to the following:

To the Animal Science Department at The University of Tennessee, Knoxville, for the opportunity to pursue the Master's degree.

To his major professor, Dr. M. C. Bell, for his guidance and advice throughout the author's graduate studies.

To Dr. John Reynolds and Professor E. R. Lidvall for serving on the thesis committee and thesis review.

To Dr. Haley Jamison for his sincere interest, guidance, and advice.

To co-workers Kathy McKim, Tina Johnson, Nancy Ramsey, Linda Miller, and all supporting staff for their assitance in collection and chemical analysis of data.

To John Burnett, Jim Tweed, and Larry Burnett for their assistance in care and handling of experimental animals.

To Jimmie Patterson and Ann Lacava for their technical assistance in preparation and typing of this manuscript.

To my parents, Richard and Elizabeth Banet, for their support and encouragement throughout the author's collegiate studies.

Finally to his wife Holly for all her patience, support, and encouragement during the author's studies.

ABSTRACT

The purpose of this study was to compare the effects of potassium (K) fertilization of fescue pasture and oral administration of potassium chloride (KCl) on plasma Mg, Ca and K in lactating beef cows. Four pasture trials were conducted in February and March of 1982 and 1983. Pasture I (control pasture) was fertilized at a rate of 112 kg of N/ha and 169 kg of P/ha. Pasture II (K pasture) was fertilized at the same rate as pasture I plus 224 kg of K/ha. One half of the animals on each pasture were given 280 grams of KCl via gelatin bolus. Milk, urine and fecal samples were collected twice daily. The Cr_2O_3 acid detergent lignin (internal-external) technique was used to estimate fecal dry matter output. The weigh-suckle-weigh method was used to estimate daily milk production. Creatinine ratios were used to determine urine volume.

Plasma Mg was depressed by pasture fertilization in both years. Potassium chloride bolusing had no effect on plasma Mg. Sixteen of 24 animals on the K fertilized pasture became hypomagnesemic. Plasma Ca was variable and inconsistent. Plasma K was not affected by treatment. However, plasma K of animals in trial II in both years had significantly higher plasma K. This may be a reflection of the higher forage K in trial II. From these results it seems the incidence of hypomagnesemic tetany may be increased by heavy K fertilization of fescue pastures and not by KCl intake.

TABLE OF CONTENTS

CHAPTER		PAGE
I.	INTRODUCTION	1
II.	REVIEW OF LITERATURE	2
	Metabolic Function of Magnesium	2
	Factors Affecting Maximum Utilization of Magnesium	7
	Prevention and Treatment of Hypomagnesemia	12
III.	EXPERIMENTAL PROCEDURE	18
	General Design	18
	Management and Procedure	19
	Data Collected	19
	Chemical Analysis	21
	Statistical Analysis	22
IV.	RESULTS AND DISCUSSION	24
	Forage and Excretions	24
	Plasma Data	37
	General Discussion	56
V.	SUMMARY	58
LITERATURE CITED		59
VITA		70

LIST OF TABLES

TABLE		PAGE
1.	Magnesium Requirement of Ruminants.	12
2.	Experimental Design	18
3.	Forage Analysis During Collection Periods in 1982	25
4.	Forage Analysis During Collection Periods in 1983	25
5.	Fecal DM Output (kg/day).	26
6.	Urine Volume Production (l/day)	27
7.	Milk Production (kg/day).	28
8.	Fecal Mg Excretion (g/day).	29
9.	Urinary Mg Excretion (g/day).	30
10.	Mammary Mg Excretion (g/day).	30
11.	Fecal Ca Excretion (g/day).	32
12.	Urinary Ca Excretion (g/day).	33
13.	Mammary Ca Excretion (g/day).	34
14.	Fecal K Excretion (g/day)	35
15.	Urinary K Excretion (g/day)	36
16.	Mammary K Excretion (g/day)	37
17.	Effect of Year on Plasma Minerals (mg/dl)	39
18.	Effect of Trial on Plasma Minerals in 1982 (mg/dl).	39
19.	Effect of Trial on Plasma Minerals in 1983 (mg/dl).	39
20.	Effect of Treatment on Plasma Mg (mg/dl) in Trial I and II in 1982.	44
21.	Effect of Treatment on Plasma Mg (mg/dl) in Trial I and II in 1983.	44

TABLE PAGE

22. Summary of the Incidences of Hypomagnesemia 45

23. Effect of Treatment on Plasma Ca (mg/dl). 47

24. Effect of Treatment on Plasma K (mg/dl) in Trial I and II in 1982. 54

25. Effect of Treatment on Plasma K (mg/dl) in Trial I and II in 1982. 54

LIST OF FIGURES

FIGURE		PAGE
1.	Changes in Plasma Mg: Trial I, 1982.	40
2.	Changes in Plasma Mg: Trial II, 1982	41
3.	Changes in Plasma Mg: Trial I, 1983.	42
4.	Changes in Plasma Mg: Trial II, 1983	43
5.	Changes in Plasma Ca in 1982.	48
6.	Changes in Plasma Ca in 1983.	49
7.	Changes in Plasma K: Trial 1, 1982	50
8.	Changes in Plasma K: Trial 2, 1982	51
9.	Changes in Plasma K: Trial 1, 1983	52
10.	Changes in Plasma K: Trial 2, 1983	53

CHAPTER I

INTRODUCTION

Magnesium (Mg) is a macroelement required by livestock (N.R.C., 1975, 1976, 1978). A depression of plasma Mg (hypomagnesemia) below 1.0 mg/100 ml often results in tetanic convulsions known as grass tetany (Sjollema, 1932). Kemp (1958) estimated death loss caused by grass tetany is 1-2% of the mature cows annually. This estimate does not include the loss in production caused by a decrease in milk production or the loss of revenue caused by the decrease in gain of calves nursing chronic hypomagnesemic cows.

It is evident that there is no single causative agent involved in the incidence of grass tetany. Instead, it appears that a combination of environmental factors such as pasture fertilization, intake of Mg, age and sex of the animal is required to produce tetany. Hypomagnesemia is not a malady of mismanaged cattle but often occurs on farms of superior management (Fontenot, 1979).

It is clear that there is no obvious answer to the problem of hypomagnesemia. This is evident by the vast amount of literature available on the subject of grass tetany. Therefore, the purpose of this study was to evaluate the effects of oral potassium (K) and K fertilization of fescue pastures on Mg, Calcium (Ca), and K metabolism of mature lactating beef cows.

CHAPTER II

REVIEW OF LITERATURE

I. METABOLIC FUNCTION OF MAGNESIUM

Physiological Function

Magnesium is widely dispersed among the tissues of the body. Approximately 60% of the Mg in the body is found in the bone (Rook and Storry, 1962). The Mg is held tightly within the "hydration shell of hydroxyapatite" and is not readily available (Care, 1967). This author also reported that only .9 to 2.3% of the Mg present in the bone was exchangeable with the extracellular fluids. Magnesium is also important in soft tissues of the body, where the remaining 40% of Mg is located (Rook and Storry, 1962). Magnesium is an important cofactor in many enzymatic reactions, especially those involved with the utilization of energy. Magnesium also plays a major role in nerve transmission, conduction and muscle contraction (Wilson, 1964).

Plasma and Serum

Hypomagnesemia is by definition a below normal plasma Mg level. In a study of 200,000 cows, Ross and Halliday (1975) found the mean plasma Mg level to be 2.1 mg/dl. Sjollema (1932) reported a range of 1.3 to 2.0 mg/100 ml. Blood samples taken from cows with clinical signs of tetany showed a mean plasma Mg of 0.79 ± .10 mg/dl (Hall and Reynolds, 1972). Kemp (1960) reported that clinical signs of hypomagnesemic tetany are seen when plasma Mg levels fall below 1.0 mg/dl.

He also noted that clinical signs are not seen in every case of hypomagnesemia. Kemp et al. (1961) showed that when plasma Mg levels dropped below 1.8 mg/100 ml, urine excretion of Mg became negligible. Results of several studies have shown a high correlation between the drop in urinary Mg and subsequent depression of plasma Mg (Rook and Balch, 1958; Kemp et al., 1961; Fisher et al., 1978). Rook and Storry (1962) found the normal lower limit of plasma Mg to be 1.7 mg/100 ml, which is thought to be close to renal threshold. The severity of hypomagnesemia differs between age groups (Chicco et al., 1973; Fisher et al., 1978). It appears that younger animals are able to mobilize up to 30% of skeletal Mg while the mature animal can mobilize only 2% of its Mg stored in the bone (Rook and Storry, 1962).

Hypomagnesemia is more prevalent in late winter and early spring. Ramsey et al. (1979) reported a decrease in plasma Mg when cows were switched from a wintering ration to lush spring pasture. Kemp (1960) stated that low serum Mg values occur when the Mg content of the forage drops below .20%. Also, as plasma Mg level decreases, intake decreases, compounding the problem of hypomagnesemia (Herd, 1966).

Absorption

The literature reviewing the site of Mg absorption is voluminous and often contradictory. Care and Van't Klooster (1965) reported little or no Mg absorption from the forestomach. In a study by Tomas and Potter (1976a), sheep were fitted with cannulas located in different parts of the digestive tract. They found the rumen to be a major site

of Mg absorption with little absorption in the abomasum or omasum. Ben-Ghedalia et al. (1975) reported that large portions of ingested Mg were absorbed before the duodenum. This is in agreement with work done by Grace et al. (1974). Tomas and Potter (1976b) showed that infusions of Mg into the abomasum and omasum had a net recovery of 100% in the duodenum. In this same study infusions of Mg into the rumen resulted in a 36-61% recovery in the duodenum, indicating Mg was absorbed from the forestomach. Excretion of Mg in the urine was greater from ruminal infusions than from infusions into the duodenum (Field and Munro, 1977). Stewart and Moodie (1956) found that after oral dosing of large amounts of magnesium, absorption occurred in the rumen, omasum, small intestine and cecum. In contrast, several workers have noted a net secretion of endogenous magnesium into the small intestine (Care and Van't Klooster, 1965; Grace et al., 1974; Ben-Ghedalia et al., 1975). Tomas and Potter (1976a), using cannulated sheep, indicated that the large intestine has the propensity to absorb magnesium but to a lesser extent. Rectal infusions of magnesium chloride ($MgCl_2$) have shown a rapid increase in plasma Mg in both normal and hypomagnesemic cattle and sheep (Meyer and Busse, 1975; Bell et al., 1978).

Movement of Mg across the epithelium of the digestive tract is thought to be an active transport mechanism (Field and Munro, 1977; Brown et al., 1978; Martens et al., 1978). Care and Van't Klooster (1965) hypothesized that the concentration of Mg in the rumen is too low to overcome the electrochemical gradient of the rumen epithelium.

In contrast, uptake postruminally of Mg is inadequate to sustain normal Mg levels of the animal (Tomas and Potter, 1976a).

Excretion

Fecal excretion of Mg has been shown to vary greatly from animal to animal. It has been suggested that the variability is due to differences in absorptive capacity of individual animals (Rook and Balch, 1958). Rook and Storry (1962) reported fecal excretion of Mg in calves to be 3.5 mg of Mg per kg of body weight, 1.5 mg of Mg per kg of body weight in mature cows and 100 to 250 mg of Mg per animal in sheep. Fecal Mg excretion expressed as a percent of daily intake was 83%. Kemp et al. (1961) found similar results with a range in fecal magnesium of 67-93% for freshly cut forages.

As would be expected, fecal Mg varies with intake. Field (1962) showed intake and fecal Mg excretion to be a rectilinear relationship. In sheep, plasma Mg varies with fecal Mg. It was also found that an increase in fecal excretion was generally greater for those sheep receiving a natural diet than those receiving an artificial diet (Allsop and Rook, 1972). These authors hypothesized that an increase in plasma Mg decreased Mg absorption and therefore increased fecal excretion of Mg. Fyre et al. (1975) confirmed this hypothesis when dietary Mg was increased from 0.5 to 4.1 g/day plasma Mg of the sheep increased along with fecal excretion. Fontenot et al. (1960) reported that a high level of K in the diet would significantly increase fecal Mg excretion. It was suggested that the K in the diet interfered with the

retention and absorption of Mg. This theory was substantiated by Field and Suttle (1979), who showed fecal excretion of Mg to be increased and retention decreased at elevated levels of K intake.

Magnesium in excess of requirement is excreted primarily through the urine (Smith, 1959). When magnesium sulfate ($MgSO_4$) was injected subcutaneously, fecal Mg excretion and plasma Mg remained unchanged, while urinary excretion of Mg increased (Smith, 1959). Jesse et al. (1981) injected 3.0 g of Mg intravenously into nonlactating cows. Recovery of the Mg was almost 100% in the urine. Several studies have reported that above renal threshold level there exists a linear response between excess Mg in the diet and excretion in the urine (Kemp et al., 1961).

Wilson (1964) calculated the threshold concentration of Mg in the plasma to be 2 mg of Mg per 100 ml. However, other work has shown urinary excretion of Mg at plasma Mg concentration below 2 mg/100 ml (O'Kelley and Fontenot, 1969). Kemp et al. (1961) reported that animals consuming diets low in Mg showed a decrease in urinary Mg before a drop in plasma Mg was observed. Urinary excretion of Mg may therefore be a better indicator of the Mg status of an animal than is plasma Mg (Kemp et al., 1961; Fisher et al., 1978). Kemp et al. (1961) also reported that hypomagnesemia did not occur in cattle unless urinary excretion of Mg was less than 1 gram of Mg/day.

II. FACTORS AFFECTING MAXIMUM UTILIZATION OF MAGNESIUM

Potassium fertilization and K content of the forage have been identified as contributing factors in the etiology of grass tetany. Early work with tobacco plants (Garner et al., 1923) first noted a Mg deficiency was increased by utilization of large amounts of K fertilizer. Several researchers have noted that the occurrence of grass tetany is greatly increased when animals are grazed on pastures containing a high K content (Dryerre, 1932; Nicholson and Shearer, 1938; Hodge, 1981). Ellis (1979) postulated that grass tetany occurs on soils that were high in exchangeable K. This author also indicated that the correlation between grass tetany and Mg in the soil is low for several reasons. First, the Mg requirement by the forage for maximum yield is often lower than the level of Mg needed in the forage to prevent grass tetany. Secondly, factors controlling the ability of animal to utilize Mg in the forage are not related to the soil content of Mg. It is interesting to note that Metson (1974) reported a high correlation between grass tetany and soil type. From this study, he suggested that soils which originate from volcanic ash had a higher incidence of grass tetany. This is in agreement with the report of a high incidence of grass tetany after the eruption of Mt. St. Helens (M. C. Bell, personal communication).

In a summary of mineral content of pasture samples collected throughout the world, Butler (1963) stated that tetany prone pastures

were usually low in Mg and sodium (Na), high in K and had a trend towards higher phosphorus (P) levels. Work done by Smyth et al. (1958) and Powley et al. (1978) have shown K fertilization will increase K content of the forage. Hodge (1981) reported similar results. Two adjacent fescue pastures were fertilized with 0 and 224 of K per hectare, respectively. The K content of the control pasture was 2.3% versus 3.0% for the potassium fertilized pasture. Time of fertilizer application will also influence Mg concentration of the forage (Lidgate, 1976). This author showed that the depression in forage Mg was greater for spring applied K fertilizer than for fertilizer applied in the fall. Potassium pasture fertilization has resulted in lowered plasma Mg levels in cattle grazing these pastures (Kemp, 1960; Hodge, 1981). DeGroot (1967) noted that not only was plasma Mg depressed by K fertilization, but when pastures were also fertilized with nitrogen (N), plasma Mg was depressed even further. In contrast, several studies have reported no effect of K fertilization on plasma Mg (Smyth et al., 1958; Hemingway et al., 1963). Sims and Crookshank (1956) fertilized wheat pastures with 0, 100 and 200 lbs of muriate of potash/acre. Plasma Mg of ewes and cows was not affected by the pasture fertilizer treatment. Also, no change was noted in mineral composition of the forage. In pastures composed largely of clovers, K fertilization had no effect on plasma Mg. However, a severe depression in plasma Mg was noted in cattle grazing pastures containing substantially less clover (Bartlett et al., 1954).

Mineral Ratios

Kemp and t'Hart (1957) reported a significant correlation between grass tetany and the ratio of K/Ca + Mg concentrations in the forage. When the ratio was greater than 2.2, there was a high incidence of grass tetany (6.6%). When the ratio was less than 2.2, the incidence of grass tetany was significantly decreased (.73%). This was in agreement with Butler (1963). It seems that under conditions of high soil moisture and high soil K, the ratio is increased. As soil moisture increases the proportion of the divalent ions decrease, and monovalent ions are increased. Since K is usually the most dominant monovalent ion, changes in the weather (rain) and soil moisture will be reflected by changes in the ratio (Ellis, 1979).

Inclusions of K in the diet of livestock has also had varying results. In general, addition of K to the diet has reduced plasma Mg (Kunkel et al., 1953; Suttle and Field, 1969; Frye et al., 1975), increased fecal Mg excretion and decreased urinary Mg excretion (Suttle and Field, 1969; Newton et al., 1972; House and Van Campen, 1971). Potassium also seems to decrease Mg availability (Kemp et al., 1961) and Mg absorption (House and Van Campen, 1971; Frye et al., 1975). Greene et al. (1983) fed four different levels of K (.6, 1.2, 2.4, 4.8%) to sheep. These authors reported as K in the diet increases, Mg absorption decreases and fecal Mg excretion increased linearly. Plasma Mg followed a similar linear decrease.

Heavy N fertilization of pastures has been shown to interfere with the utilization of Mg (O'Kelley and Fontenot, 1969; Moore et al.,

1972a; Moore et al., 1972b; Henry et al., 1977). Stewart et al. (1981) reported N fertilization increased the tetany producing potential of winter wheat. Animals grazing pasture fertilized with K showed a depression of plasma Mg (O'Kelley and Fontenot, 1969; Kemp, 1960), decrease in urinary Mg and an increase in fecal excretion of Mg (Stillings et al., 1964). Nitrogen fertilization of pastures results in an increase in protein content (Kemp, 1960), which in turn will result in an increase in ammonia concentration in the rumen (Wilcox and Huff, 1974). The latter authors suggested that the increase in ruminal ammonia may interfere with Mg utilization and availability. Inclusion of ammonia and urea in the rations of sheep has reduced serum Mg and reduced the retention of Mg (Moore et al., 1972b).

Application of both N and K fertilizer seems to have an additive effect on depressing Mg utilization (Smyth et al., 1958; Fontenot et al., 1960; Kemp, 1960; Moore et al., 1972a). Fontenot et al. (1960) fed diets to sheep similar in K content to that found in rapidly growing wheat pastures. Animals on a high protein and high K diet showed a 38% increase in fecal Mg excretion. However, Moore et al. (1972a) reported a significant increase in fecal Mg at elevated levels of dietary K regardless of protein content.

Aluminum (Al) has been identified as being another causative agent in the etiology of grass tetany (Allen, 1979; Ellis, 1979). Ellis (1979) suggested that on highly acidic soils the trivalent Al ion disrupts the uptake of Mg by the plant. Allen (1979) reported excessively high levels of Al in the rumen contents of tetany animals. A mean Al

concentration of the samples from tetany animals was 2373 ppm versus 405 ppm for non-tetany animals. Aluminum content of forage samples taken from tetany prone pastures was quite variable. The one consistent characteristic was the high concentration of Al found in areas that often had standing water.

Organic Acids

High concentration of organic acids has been related to the incidence of grass tetany (Burau and Stout, 1965; Bohman et al., 1969; Scotto et al., 1971). Stout et al. (1967) found trans-aconitate in large concentration in early spring, tetany-prone pastures. Oral administration of trans-aconitate has not shown any adverse side effects (Camp et al., 1968; Kennedy, 1968). The authors suggested trans-aconitate may be a compounding factor in the etiology of grass tetany and not a direct cause of the malady. Citric acid has also been identified as a potential component in grass tetany (Burt and Thomas, 1961; Bohman et al., 1969). Citric acid has been shown to decrease plasma magnesium in young calves (Burt and Thomas, 1961). Kennedy (1968) reported no significant effect on plasma or urinary Mg due to an increase in citric acid intake. Bohman et al. (1969) induced clinical tetany at very high levels of K (150 g/100 kg body weight) and citric acid (157 g/100 kg of body weight). It was interesting to note that tetany was only induced when the two were used in combination.

Meteorology

Allcroft (1954) reported the probability of grass tetany is greater on cold, wet mornings during a period of rapid forage growth. Kemp and t'Hart (1957) have correlated the incidence of grass tetany with an increase in temperature. These authors suggest that 5 days after a rise in temperature above 14°C an increase in the number of tetany cases occur. It was hypothesized that the change in temperature caused a change in the forage composition.

III. PREVENTION AND TREATMENT OF HYPOMAGNESEMIA

The National Research Council has published Mg requirements for most species of livestock (Table 1) (N.R.C., 1975, 1976, 1978).

Table 1. Magnesium Requirement of Ruminants

Class	Requirement (%)
Sheep (adult)	.06
Beef Cattle	
lactating cows	.18
growing and finishing	.04 - .10
Dairy Cattle	
lactating cows	.20
dry pregnant cows	.16
growing heifers and bulls	.16

O'Kelley and Fontenot (1969) reported that in order to maintain a plasma Mg level of 2.0 mg of Mg/dl, a mature lactating beef cow would have to consume 20 gm of Mg/day. In studies to determine a threshold level of Mg in the urine, Kemp et al. (1961) reported a requirement of 2.5 g of Mg plus .12 gram of Mg for each kilogram of milk produced.

Dietary requirement of Mg during gestation has been estimated to be approximately one-half the amount required during lactation (O'Kelley and Fontenot, 1969). These authors concluded that at 145, 200 and 255 days of gestation the Mg requirements were 8.5, 7.0 and 9.0 gram/day/cow, respectively.

During most of the year, Mg provided in the forage will be sufficient to meet the requirements of the animal. When rapid, lush growth of the forage coincides with the initiation of lactation, all the requirements for Mg by the animal may not be met. The concentration of Mg in the forage may be too low to meet the needs of the animal. Various regimes of supplying additional Mg have been used. Bartlett et al. (1954) reported a 64% increase in the Mg level of forage, after 2500 lb of magnesite/acre was applied. In a comparison of magnesium limestone and calcined limestone, Parr and Allcroft (1957) showed that both application of 2.5 tons of limestone and 1000 lb of magnesite/acre increased the Mg content of the forage, with the response being greater for the magnesite. During the next 6 years, there were no clinical signs of grass tetany on these pastures. However, on comparable unfertilized plots tetany did occur. Plasma Mg levels for cows

on a pasture fertilized with 325 lb of Mg/acre were increased significantly. These animals had a mean plasma Mg level of 2.38 mg of Mg/dl compared to 1.5 mg of Mg/dl for the controls (Smyth et al., 1958). A major disadvantage to Mg fertilization of pastures is that extremely large amounts of fertilizer are required to increase the level of Mg in the forage (Grunes et al., 1970). Mayland and Grunes (1979) found that 600 kg of Mg/ha was needed to increase the Mg level of wheat pasture to .20%.

An alternative to pasture fertilization is foliar applications of Mg as a dust or slurry. Todd and Morrison (1964) applied 28 lb of calcined magnesite/acre as a dust. Two days after the dust was applied forage Mg had been increased from .16% to .31%. A 20% $MgSO_4$ spray has been shown to increase forage Mg to .7% immediately after application. Although the forage content was raised substantially, plasma Mg level was elevated only slightly (McConaghy et al., 1963). Adhesion and persistence of magnesium to the forage is very dependent on wind and amount of rainfall (Wilkinson et al., 1972). Young (1975) reported that when rainfall was greater than 40 mm redusting was necessary. Meyer (1976) reported 40% recovery when the Mg dust was applied to "dew-wet grass." In attempt to increase adhesion, several workers have increased forage Mg by applying a magnesium-bentonite slurry (Wilkinson and Stuedemann, 1979; Reynolds, 1980). The former authors applied 20 to 30 kg of MgO/ha as a slurry and stated it was effective in controlling grass tetany, but it too was very dependent on wind and rainfall. Reynolds (1980) indicated that the MgO-bentonite slurry

was more resistant to rainfall than dusting. The slurry could be expected to last up to 4 weeks providing that rainfall was not excessive. Several researchers have reported little problems with consumption of foliar applied Mg (Rogers and Poole, 1971; Stuedemann et al., 1973; Young, 1975). Oral drenching with Mg compound has had limited success (Bell, 1980). Allcroft (1954) increased plasma Mg level of cows drenched with 2 oz of MgO/day. In this same study when the Mg supplementation was terminated, plasma Mg level fell rapidly. Inclusion of 2 ounces of calcined magnesite in the diet of dairy cows provided protection against grass tetany. When the lowest plasma Mg levels of cows on the two treatments were compared, the supplemented cows had a low of 1.1 mg of Mg/100 ml versus 0.5 mg of Mg/100 ml (Line et al., 1958). Rogers et al. (1977) reported that drenching cattle with 4 ounces of MgO on every other day was inadequate to prevent occurrence of grass tetany. Addition of Mg to the drinking water of cattle has met with some success. Rogers and Poole (1976) reported an increase in plasma Mg of 2.1 and .34 mg/100 ml by the addition of 15.5 g of Mg/cow/day as MgO and magnesium acetate, respectively. In areas down wind from Mt. St. Helens in Washington, ranchers have successfully added $MgCl_2$ to the water to control grass tetany in their herds (M. C. Bell, personal communication). A major problem with supplemental Mg in the water is that during times of excess rainfall cattle will drink the pooled rain water and thus may not receive adequate intake of supplemental Mg (Wilkinson and Stuedemann, 1979; M. C. Bell, personal communication). The latter reported several cows were lost

due to grass tetany after heavy rains. Low-lying areas containing the runoff were fenced off, and no other cases of grass tetany were reported. Mineral mixes that include Mg in a palatable supplement have been shown to provide adequate protection against grass tetany (Allcroft and Green, 1938; Fontenot et al., 1965; Ritchie and Fishwick, 1977). Palatability remains the major problem with adequate intake of Mg. Addition of dry and wet molasses has been shown to increase consumption of magnesium-salt mixtures (Todd et al., 1966; Horvath et al., 1967). McLaren et al. (1975) reported an intake of 9.6 gm of Mg/cow/day. The mix was composed of MgO, salt molasses and cottonseed meal in a 1:1:1:4 ratio. Magnesium molasses blocks have resulted in excellent consumption of Mg. Convenience plays a major role in the use of magnesium blocks on many farms (Horvath and Todd, 1968).

Slow-release, magnesium bullets have been shown to provide adequate protection against grass tetany (Ritchie et al., 1962). The Mg bullets are composed of magnesium alloy containing 2% Cu, 12% Al, and 86% Mg. The bullets weigh approximately 100 grams, are 7.5 cm long and 2.5 cm in diameter. This specific type was designed to release 1 gm of Mg/day and last for 150 days (Ritchie and Hemmingway, 1968).

Several problems have arisen from using bullets. Foot et al. (1969) reported a case of reticulitis presumably caused by a roughened bullet. The former, along with Kemp and Todd (1970), stated that there were circumstances in which Mg bullets did not provide adequate protection. A wide variation in decomposition of the bullets exists between animals which may lead to inconsistent release of Mg and inadequate protection (House and Mayland, 1976).

Meyer and Busse (1975) reported an increase in plasma Mg from rectal infusion of a 30% MgCl solution. Bell et al. (1978) successfully treated hypomagnesemic cows with a rectal infusion of magnesium chloride. The increase in plasma Mg lasted approximately 24 hours in hypomagnesemic animals versus only 8 hours for control animals. $MgSO_4$ solutions did not increase plasma Mg.

The usual treatment for grass tetany is intravenous (I.V.), intramuscular (I.M.) or intraperitoneal (I.P.) injections of Mg-Ca solutions. Two-hundred fifty g of Ca borogluconate and 50 g of Mg borogluconate or sulfate is dissolved in 1 liter of distilled water. Dosages for cattle and sheep are 400 to 800 ml and 100 ml respectively, given I.V., I.P. or subcutaneously (Merck, 1979).

CHAPTER III

EXPERIMENTAL PROCEDURE

I. GENERAL DESIGN

Two pasture studies were conducted in February and March of 1982 and 1983. Twelve lactating Angus cows were allotted to a two-by-two factorial arrangement in each trial. Treatments consisted of a control pasture and a high K fertilized pasture. Each tall fescue pasture was fertilized the preceding fall. The control pasture was fertilized at a rate of 112 kg of N/ha, 169 kg of P/ha with no K fertilization. The high potassium pasture was fertilized at this same rate plus 224 kg of K/ha was applied. Six animals were placed in each pasture at a stocking rate of 1.25 animal/ha at the beginning of each trial. These six animals were then subdivided into KCL treatments. KCL treatments consisted of three animals receiving 280 grams of KCL/day with the remaining three animals receiving 0 grams of KCL/day. Treatment assignments are illustrated in Table 2. Animals were allotted to

Table 2. Experimental Design

KCl Bolus Treatment g/day	Pasture Treatment	
	Control	High K
0	1-12*	3-12
280	2-12	4-12

*Treatment number and total number of animals in each treatment respectively.

their respective treatments by age, plasma Mg, weight and age of calf. Each trial consisted of a one-week preliminary and a one-week collection.

II. MANAGEMENT AND PROCEDURE

Animals utilized in this study were selected from the herd at Holston farm. Average age of the animals used was eight years, with a range of five to fifteen years. All cows had calves at side, ranging in age from one to seven weeks. Cows averaged 460 kg at the beginning of the trial. In year 1, cows were offered a 1:1 mineral mix of limestone and dicalcium phosphate plus salt. All cows were bled via jugular puncture during the last week in January and again two weeks prior to the start of the first trial. These samples were used to allot cows to treatments.

III. DATA COLLECTED

Cows were stanchioned twice daily at approximately 0800 hr and 1600 hr in metabolism stalls similar to those described by Ritter (1979). Beginning on day 0, all animals were bolused with 2.00 gm Cr as chromium sesquoxide. Bolusing with KCl was initiated on day 6 and was administered in a gelatinous capsule via a balling gun. These KCl boluses were given in split applications with 140 gm per cow given each A.M. and P.M. Calves were separated from their dams at the beginning of each trial. Calves were allowed to nurse twice daily. Beginning on day 7, calves were weighed before and after nursing (weigh-suckle-weigh) to determine milk production of their dams. Midway through the

nursing period, approximately 50 ml of milk were obtained, stored and frozen at -4°C for later chemical analysis. Due to the impracticality of total fecal and urine collection, dry matter intake, fecal dry matter output and urine volume were estimated by using indirect measures. Urine volume was estimated by using creatinine ratios (Albin and Clanton, 1966; Field, 1964). Urine volume was calculated from equation 1.

(1) Urine volume (ml/day) = 50.9 x Wt (kg)/concentration of creatinine in urine sample (The constant, 50.9 mg of creatinine/kg BW was determined by Ritter (1979)).

Dry matter intake and fecal dry matter output was estimated by using the internal-external indicator method (Linkous et al., 1955). The calculation of fecal dry matter output and dry matter intake are represented in equations 2 and 3 respectively (Crampton and Harris, 1969).

(2) Fecal DM output (g/day) = g Cr in feed x 100/%Cr in fecal sample.

(3) Forage DM intake (g/day) = (Fecal DM output x % Fecal ADL)/(% ADL in Forage).

Grab urine and fecal samples were collected twice daily. If midway through the nursing period fecal samples had not been obtained, cows were rectally palpated to promote defecation. If necessary, urination was induced by palpating the cow between the udder and vulva.

Urine samples were composited by day and separated into two portions for mineral and creatinine analysis. In year two, urine samples were centrifuged and the supernatant removed for immediate mineral analysis. Urine and fecal samples were stored in the same way as the milk samples. Blood samples were obtained on days 0, 3, 5, 7, 9, 11 and 13. Blood samples were centrifuged and plasma was removed for immediate mineral analysis; grab forage samples were obtained by walking diagonally across the pastures. Samples were obtained on days 0, 7 and 13 of the trial.

IV. CHEMICAL ANALYSIS

Plasma

Plasma samples were prepared and diluted to a known volume according to standard procedure. An Instrumentation Laboratory Model 551 Atomic Absorption/Atomic Emission Spectrophotometer was used to analyze all samples for Ca, Mg and K.

Forage and Feces

Forage and fecal dry matter were determined using standard procedures from A.O.A.C. (1975). Dried fecal and forage samples were then ground through a 1 mm screen in a Wiley mill. Next, fecal samples were composited by day. Approximately .5 grams of forage and fecal samples were ashed at 600°C in a muffle furnace for 4 hours. The ashed samples were then dissolved in three ml of 6N HCL and brought to a known volume and analyzed for Ca, Mg and K. Forage and fecal acid detergent lignin was determined by the method described by Van Soest and Vine

(1967). The procedure of Williams et al. (1962) was used for analysis of Cr.

Urine and Milk

In year one, five ml of urine was ashed for 4 hours at 600°C. Milk was handled in the same way in both year 1 and year 2. These samples were then processed in the same manner as the fecal and forage samples. In year 2, urine samples were run fresh, similar to the method used for plasma analysis. Creatinine concentration was determined by using Creatinine II™ test.[1]

V. STATISTICAL ANALYSIS

In the statistical analysis of data, programs prepared by S.A.S. Institute (1982) were utilized. General linear models procedure was used to test the variation in dependent variables. The model used was:

$$Y_{ijk} = U + Y_i + K_j + t_k + e_{ijk}$$

where

Y_{ijk} = dependent variables

M = theoretical population mean

Y_i = effect of year, i = 1-2

[1]Boehringer Mannheim Diagnostics. Houston, TX 77063.

K_j = effect of trial, j = 1-2

t_k = effect of treatment, k = 1-4

e_{ijk} = random error.

If significant differences were found by the above procedure, Student Neuman-Keuls' test (Sokal and Rohlf, 1969) was used to separate the least square means.

CHAPTER IV

RESULTS AND DISCUSSION

I. FORAGE AND EXCRETIONS

Chemical analysis of forage samples collected during the balance trials are presented in Tables 3 and 4. Magnesium content of both pastures was lower in 1983 than in 1982. Differences between pasture fertilization on Mg content of the forage were variable and inconsistent. Pastures fertilized with excess K were lower in Ca and higher in K. Acid detergent lignin content was higher in the control pasture.

As previously outlined in another chapter, indirect indicators were used to estimate dry matter intake (DMI), fecal dry matter (FDM) excretion and urine volume. Inherent errors exist in many of these methods. When DMI was calculated, estimates were approximately two times higher than expected. Crampton and Harris (1969) emphasized the importance of collecting representative samples of the consumed forage. When the forage data was compared to samples collected from similar pastures and from data collected on these same pastures in previous years, it becomes obvious that the ADL content of the pastures was lower than would be expected. This deflation in forage ADL content consequently led to an inflation in the estimation of DMI. Since these data appear to be unrealistic, it was omitted from this discussion.

Table 5 contains means of FDM excretion. In general, animals on the K fertilized pasture had lower FDM excretion than did animals

Table 3. Forage Analysis During Collection Periods in 1982

	Trial I		Trial II	
Variable	Control	K Fertilized	Control	K Fertilized
Mg[a]	1.60	1.9	2.1	1.9
Ca[a]	4.6	4.2	4.1	3.5
K[a]	9.0	10.0	22.2	34.8
ADL[b]	4.35	3.47	3.31	3.14

[a]Expressed as mg/g DM basis.

[b]Expressed as a % DM basis.

Table 4. Forage Analysis During Collection Periods in 1983

	Trial I		Trial II	
Variable	Control	K Fertilized	Control	K Fertilized
Mg[a]	1.31	1.32	1.59	1.21
Ca[a]	4.15	3.73	4.92	4.18
K[a]	19.05	21.48	20.18	27.5
ADL[b]	3.26	2.93	3.41	3.01

[a]Expressed as mg/g DM basis.

[b]Expressed as % DM basis.

Table 5. Fecal DM Output (kg/day)

		Treatment			
	Trial	1	2	3	4
1982	1	6.69[a]	6.78[a]	5.19[a]	6.00[a]
1982	2	6.30[a]	5.30[b]	5.12[b]	4.82[b]
1983	1	5.15[a]	5.14[a]	3.64[b]	4.55[a]
1983	2	6.63[a]	6.48[a]	5.30[b]	5.08[b]

Means within rows with different superscripts are different ($P < .05$).

on the control pasture. In trial 1 of 1982 and 1983, animals in treatment 3 excreted significantly less ($P < .05$) FDM than those in treatments 1, 2 or 4. In trial 2 of 1982, animals in treatment 1 excreted significantly more ($P < .05$) FDM than did those in treatments 2, 3 or 4. In trial 2 of 1983, animals on the control pasture (treatments 1 and 2) excreted significantly more FDM than did animals on the K fertilized pasture (treatments 3 and 4).

Means of daily urine volume production are presented in Table 6. In trial 1 of 1982, animals in treatments 3 and 4 excreted more ($P < .05$) urine than did animals on treatments 1 and 2. In trial 2, cows in treatments 2, 3 and 4 excreted significantly more ($P < .01$) urine than treatment 1 cows. In 1983, no significant differences were noted between treatments in either trial.

Table 6. Urine Volume Production (l/day)

Year	Trial	Treatment 1	2	3	4
1982[1]	1	5.68[b]	5.86[b]	9.15[a]	11.51[a]
1982[1]	2	6.93[b]	14.17[a]	13.91[a]	13.25[a]
1983[2]	1	6.95[a]	6.04[a]	6.33[a]	6.58[a]
1983[2]	2	4.76[a]	7.14[a]	5.40[a]	6.17[a]

[1]Means within this row with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

Means of daily milk production are presented in Table 7. In 1983, animal 201 produced exceptionally low quantities of milk. This low milk production was attributed to poor health, unrelated to hypomagnesemia or grass tetany. Data for milk production and mammary mineral excretion for this animal were therefore omitted from analysis. Milk production within years showed little variation. In trials 1 and 2 of 1982, animals in treatment 3 produced more ($P < .01$) milk per day than did animals in any other treatment. Treatments 1, 2 or 4 did not differ significantly in kg of milk produced per day. In trial 1 of 1983, animals on the control pasture produced more ($P < .05$) milk than did animals on the K fertilized pasture. In trial 2, treatments 1, 3 and 4 produced more ($P < .01$) milk than did treatment 2. Cows were not

Table 7. Milk Production (kg/day)

Year	Trial	Treatment			
		1	2	3	4
1982[1]	1	5.90[b]	5.54[b]	6.74[a]	5.40[b]
1982[1]	2	5.50[b]	5.48[b]	6.74[a]	5.1[b]
1983[2]	1	9.42[a]	9.22[a]	8.07[b]	7.92[b]
1983[1]	2	7.23[a]	5.42[b]	7.05[a]	7.37[a]

[1]Means within rows with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

allotted to treatment based on past milk production so the small numbers and the inconsistent treatment effects may be due to genetic variation.

Daily fecal Mg excretion means are presented in Table 8. In 1982, the general trend was for animals on the K fertilized pasture to excrete the most Mg in their feces. This increase in fecal Mg excretion and reduced fecal DM excretion of these animals indicates a decrease in Mg absorption. House and Van Campen (1971) indicated that an excess of K in the diet could decrease Mg absorption. Conversely, in 1983 animals on the control pasture had higher fecal Mg excretion. The increase in fecal Mg excretion was mainly due to an increase in fecal DM and not a difference in percent of Mg in the feces. The effect of KCl bolusing on fecal Mg excretion was varied and inconsistent.

Table 8. Fecal Mg Excretion (g/day)

Year	Trial	Treatment			
		1	2	3	4
1982	1	16.65[b]	16.15[b]	16.33[b]	22.30[a]
1982	2	20.35[b]	17.82[b]	26.18[a]	24.73[a]
1983	1	15.32[b]	17.86[a]	9.38[d]	12.72[c]
1983	2	22.29[a]	27.17[a]	21.01[a]	21.06[a]

Means within rows with different superscripts are different ($P < .05$).

Table 9 contains means of daily urinary Mg excretion. In trial 1 of 1982, animals in treatment 3 excreted significantly less ($P < .05$) Mg in the urine than did treatments 1 and 4 cows. Urine volume was 9.15 liters for cows on treatment 3 compared with 5.68 liters for animals in treatment 1. Urinary Mg excretion for animals in treatment 2 were not statistically different from any treatment. In trial 2, no differences in urinary Mg excretion patterns were noted. In trial 1 of 1983, animals on the control pasture excreted more ($P < .01$) Mg in the urine than did animals on the K fertilized pasture. In trial 2, animals in treatment 3 excreted less ($P < .05$) Mg in the urine than cows on any other treatment. Daily urinary Mg excretions were low averaging 0.5 g or less per treatment group.

Means of daily mammary Mg excretion are shown in Table 10. In general, mammary Mg excretion was constant within individual trials

Table 9. Urinary Mg Excretion (g/day)

Year	Trial	Treatment 1	2	3	4
1982[2]	1	.13[a]	.08[ab]	.04[b]	.15[a]
1982[2]	2	.10[a]	.03[a]	.50[a]	.01[a]
1983[1]	1	.45[a]	.40[a]	.1[b]	.02[b]
1983[2]	2	.44[a]	.50[a]	.03[b]	.13[ab]

[1]Means within this row with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

Table 10. Mammary Mg Excretion (g/day)

Year	Trial	Treatment 1	2	3	4
1982[2]	1	.62[a]	.51[bc]	.56[ab]	.44[c]
1982[1]	2	.45[b]	.45[b]	.73[a]	.51[b]
1983[2]	1	1.0[a]	.94[a]	.92[a]	.76[b]
1983[1]	2	.79[a]	.59[b]	.82[a]	.81[a]

[1]Means within this row with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

but varied among trials. In trial 1 of 1982, animals in treatment 1 excreted more ($P < .05$) Mg in the milk than did treatments 2 or 4. Animals in treatment 3 excreted significantly more Mg in the milk than animals in treatment 4. Animals in treatment 3 were not statistically different from treatments 1 or 2. In trial 2, animals in treatment 3 excreted significantly more ($P < .01$) Mg in milk than any other treatment. In trial 1 of 1983, treatments 1, 2 and 3 excreted more ($P < .05$) Mg in their milk than did animals in treatment 4. In trial 2, animals in treatment 2 excreted significantly less Mg in their milk than did treatments 1, 3 and 4 cows. The differences seen in mammary Mg excretion was mainly due to differences in milk production.

Table 11 contains means of daily fecal Ca excretion. In trial 1 of 1982, animals in treatment 4 excreted more ($P < .05$) Ca in the feces than did treatment 3 cows. Fecal Ca excretion of treatments 1 and 2 cows did not differ significantly from either treatment 3 or 4 cows. In trial 2, cows on treatments 3 and 4 were significantly lower ($P < .01$) in fecal Ca excretion than were treatment 1 cows. In 1983, animals on the K fertilized pasture were generally lower in fecal Ca excretion. In trial 1, animals in treatment 1 excreted more ($P < .01$) Ca in the feces than did treatments 3 or 4 cows. Fecal Ca excretion was greater ($P < .01$) for animals in treatment 4 than for animals in treatment 3. In trial 2, animals in treatment 2 excreted significantly more ($P < .05$) fecal Ca than did any other treatment. Fecal Ca excretion for animals in treatments 3 and 4 did not differ statistically

Table 11. Fecal Ca Excretion (g/day)

Year	Trial	Treatment			
		1	2	3	4
1982[2]	1	72.16^{ab}	69.48^{ab}	54.02^{b}	98.69^{a}
1982[1]	2	96.67^{a}	84.54^{ab}	72.69^{b}	72.29^{b}
1983[1]	1	77.62^{a}	68.67^{ab}	40.11^{c}	58.37^{b}
1983[2]	2	116.45^{b}	140.6^{a}	72.28^{c}	74.20^{c}

[1]Means within this row with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

but were significantly lower ($P < .05$) than treatment 1 cows. Animals in treatments with the highest fecal DM output also had the highest concentration of Ca in their feces. Conversely, animals in treatments with low fecal DM output also had lower Ca concentration in their feces.

Daily mean urinary Ca excretions are presented in Table 12. Differences between treatments were minor and inconsistent. In 1982, no significant differences in urinary Ca excretion were noted. In 1983, animals in treatment 2 excreted significantly more ($P < .01$) in the urine than did any other treatment. Animals in treatments 1, 3 and 4 did not differ significantly in excretion of urinary Ca. In trial 2, animals on the K fertilized pasture were significantly lower

Table 12. Urinary Ca Excretion (g/day)

Year	Trial	Treatment			
		1	2	3	4
1982[2]	1	.48[a]	.43[a]	.17[a]	.49[a]
1982[2]	2	.17[a]	.22[a]	.28[a]	.25[a]
1983[1]	1	.23[b]	.57[a]	.05[b]	.09[b]
1983[1]	2	.57[a]	.48[a]	.06[b]	.09[b]

[1]Means within this row with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

($P < .01$) in their urinary excretion of Ca than were animals on the control pasture.

Daily means of mammary Ca excretion are presented in Table 13. Surprisingly, Ca excretion in the milk was not consistent across treatments. In trial 1 of 1982, animals receiving KCl boluses excreted significantly less ($P < .05$) Ca in their milk than did their non-bolused counterparts. In trial 2, animals in treatments 1, 2 and 4 excreted significantly less ($P < .01$) mammary Ca than did animals in treatment 3. Animals in treatments 1, 2 and 4 were not significantly different. In trial 1 of 1983, animals in treatment 1 excreted more Ca in their milk than did animals in treatment 4. In trial 2, animals on the K

Table 13. Mammary Ca Excretion (g/day)

Year	Trial	Treatment 1	2	3	4
1982[2]	1	7.2[b]	6.5[c]	8.9[a]	6.2[c]
1982[1]	2	5.8[b]	5.5[b]	8.7[a]	5.1[b]
1983[2]	1	10.61[a]	9.77[ab]	9.09[ab]	8.48[b]
1983[2]	2	8.35[c]	9.93[b]	11.4[a]	10.42[ab]

[1]Means within rows with different superscripts are different ($P < .01$).

[2]Means within rows with different superscripts are different ($P < .05$).

fertilized pasture excreted more Ca in their milk than did treatments 1 and 2.

Daily fecal K excretion means are presented in Table 14. Generally, animals in treatment 4 excreted the most fecal K. In trial 1 of 1982, treatment 4 cows excreted more K ($P < .05$) than any other treatment group, while animals in treatment 1 excreted the least ($P < .05$) amount of fecal K. Animals in treatments 2 and 3 were not significantly different in their amount of K excreted. In trial 2, animals in treatment 3 excreted significantly more ($P < .05$) K in the feces than did treatments 2 or 4. In trial 1 of 1983, animals in treatment 1 excreted significantly less ($P < .01$) K than did treatment 4

Table 14. Fecal K Excretion (g/day)

Year	Trial	Treatment 1	2	3	4
1982[1]	1	52.42^{c}	72.51^{b}	67.08^{b}	87.81^{a}
1982[2]	2	52.34^{ab}	39.62^{b}	60.88^{a}	42.40^{b}
1983[1]	1	40.51^{b}	49.12^{ab}	47.57^{ab}	56.20^{a}
1983[1]	2	55.5^{a}	74.75^{a}	83.43^{a}	96.94^{a}

[1]Means within this row with different superscripts are different ($P < .01$).

[2]Means within this row with different superscripts are different ($P < .05$).

cows. Animals in treatments 2 and 3 excreted significantly less fecal K than cows on treatment 4 and excreted significantly more ($P < .01$) than cows on treatment 1. In trial 2, no significant differences in fecal K excretion were noted.

Urinary K excretion means are presented in Table 15. Relatively few differences in urinary excretion of K were noted. In trial 1 of 1982, animals in treatment 4 excreted significantly more ($P < .05$) K in the urine than did treatments 1, 2 or 3. In trial 2 of 1982 and trial 1 of 1983, no significant differences in urinary K excretion were noted. In trial 2 of 1983, animals receiving KCl boluses (treatments 2 and 4) excreted more ($P < .05$) K in the urine than did their non-bolused counterparts. Excess K in the diet is rapidly excreted

Table 15. Urinary K Excretion (g/day)

Year	Trial	Treatment			
		1	2	3	4
1982[1]	1	22.44[b]	45.07[b]	33.48[b]	75.05[a]
1982[2]	2	40.03[a]	52.67[a]	88.02[a]	63.11[a]
1983[2]	1	33.90[a]	38.82[a]	35.40[a]	47.80[a]
1983[2]	2	34.51[b]	71.41[a]	48.73[b]	69.02[a]

[1]Means within rows with different superscripts are different ($P < .01$).

[2]Means within rows with different superscripts are different ($P < .05$).

from the body. Lentz et al. (1976) showed a rapid increase in plasma K through 60 minutes after intraruminal infusion with 550 g of KCl, but plasma K returned to normal within 120 minutes post infusion. The primary route of excretion appears to be the urine. The small differences in urinary K excretion was unexpected. It appears, as with plasma K, that synchronizing sampling time with rate of disappearance of K in the urine is critical. If the sampling of urine had coincided with rapid excretion, it is probable that large differences in urinary excretion of K would have been observed.

Table 16 contains daily means of mammary K excretion. In trial 1 of 1982, animals in treatments 1 and 3 excreted significantly more ($P < .01$) K in the milk than did cows in treatments 2 or 4. In trial

Table 16. Mammary K Excretion (g/day)

Year	Trial	Treatment			
		1	2	3	4
1982	1	4.9^{a}	2.0^{b}	5.7^{a}	1.5^{b}
1982	2	6.08^{a}	2.4^{b}	5.5^{a}	4.2^{ab}
1983	1	8.3^{a}	8.08^{a}	6.09^{b}	6.0^{b}
1983	2	6.33^{a}	4.2^{c}	5.5^{b}	5.5^{b}

Means within rows with different superscripts are different ($P < .01$).

2, animals in treatment 2 produced lower ($P < .01$) amounts of K in their milk than did animals in treatments 1 or 3. In trial 1 of 1983, animals that were on the K fertilized pastures excreted significantly less ($P < .01$) K in their milk than did their control pasture counterparts. In trial 2, animals in treatment 2 excreted significantly less ($P < .01$) K in the milk than did any other treatment. Conversely, animals in treatment 1 excreted significantly more ($P < .01$) K in the milk than did any other treatment. Animals in treatments 3 and 4 did not differ in mammary K excretion. Total milk K was usually but not always associated with total milk output.

II. PLASMA DATA

Preliminary statistical analysis showed significant differences between years for all plasma minerals. Significant differences were

also noted between trials for plasma Mg and plasma K. Therefore, data for plasma Mg and plasma K are presented by year and trial. Data for plasma Ca is pooled across years. The effect of year on plasma minerals is shown in Table 17. Plasma K was significantly lower ($P < .01$) in 1983 than in 1982. Plasma Ca and plasma Mg were higher ($P < .01$) in 1983. Differences between trials are represented in Tables 18 and 19. In 1982, plasma Mg and plasma K were significantly lower in trial 1 ($P < .01$) than in trial 2. There were no significant differences between trials for plasma Ca in either year. In 1983, plasma Mg was lower ($P < .01$) in trial 2 than in trial 1. In the same year, plasma K was higher ($P < .01$) in trial 2 than in trial 1.

Changes in plasma Mg over time are presented in Figures 1, 2, 3 and 4. Tables 20 and 21 contain plasma Mg means for 1982 and 1983, respectively. In all 4 trials, plasma Mg was lower for animals on the K fertilized pastures. KCl bolusing had no effect on plasma Mg in 1982. Potassium chloride bolusing was associated with depressed plasma Mg in 1983. In trial 1, animals on the control pasture that were bolused with KCl (treatment 2) had lower ($P < .05$) plasma Mg levels than did their counterparts receiving no KCl (treatment 1). No differences were observed due to KCl bolusing on the K fertilized pasture. In trial 2, animals on both pastures that were bolused with KCl had lower mean plasma Mg ($P < .05$) levels than did their non-bolused counterparts. In general, animals in treatments 3 and 4 showed a trend of decreasing plasma Mg throughout the trial. In contrast, cows in treatments 1 and 2 were more variable and inconsistent. Plasma Mg of

Table 17. Effect of Year on Plasma Minerals (mg/dl)

Year	Ca[a]	Mg[a]	K[a]
1982	10.025	1.7289	17.619
1983	10.329	2.0409	15.549

[a]Means within this column are different ($P < .01$).

Table 18. Effect of Trial on Plasma Minerals in 1982 (mg/dl)

Trial	Ca[b]	Mg[a]	K[a]
1	10.08	1.61	16.20
2	9.97	1.82	19.04

[a]Means within this column are different ($P < .01$).

[b]Means within this column are not different ($P < .05$).

Table 19. Effect of Trial on Plasma Minerals in 1983 (mg/dl)

Trial	Ca[c]	Mg[a]	K[b]
1	10.427	2.14	15.17
2	10.231	1.94	15.93

[a]Means within this column are different ($P < .05$).

[b]Means within this column are different ($P < .01$).

[c]Means within this column are not different ($P < .05$).

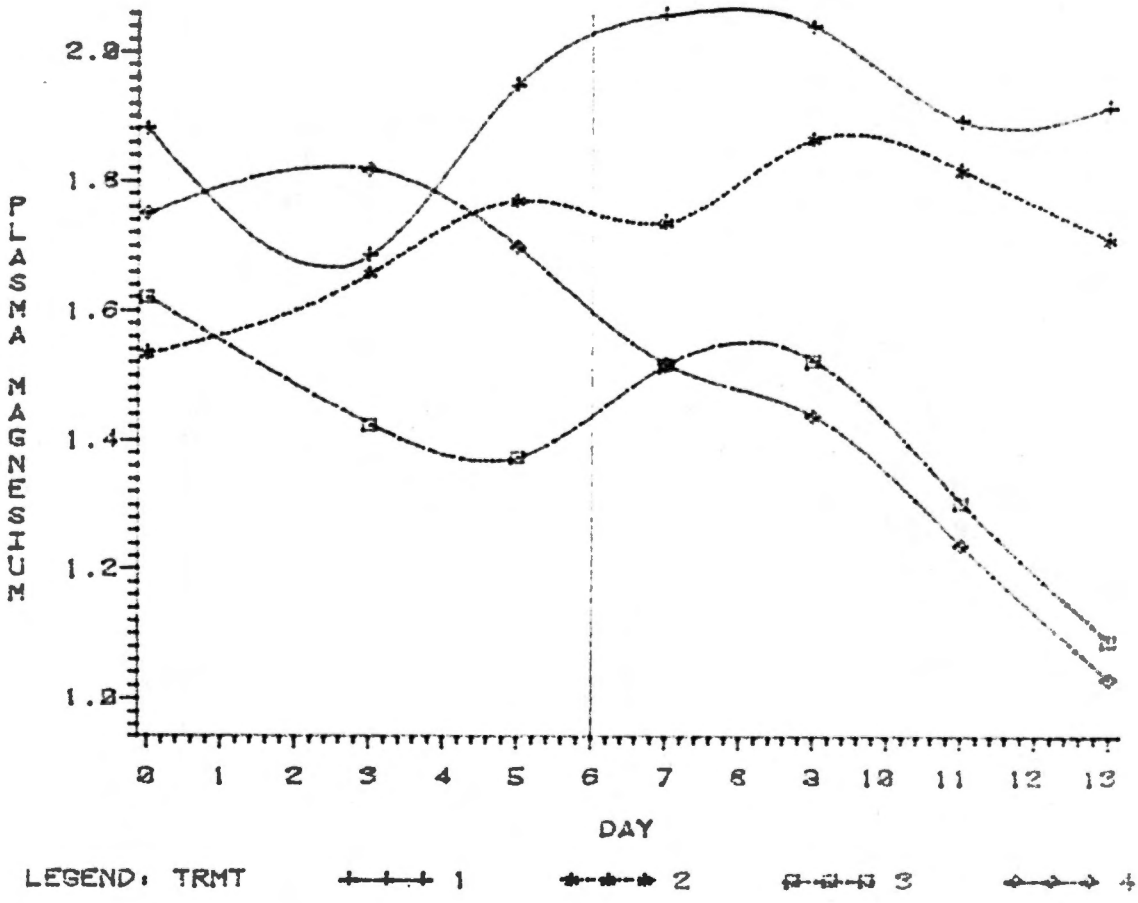

Figure 1. Changes in Plasma Mg: Trial I, 1982.

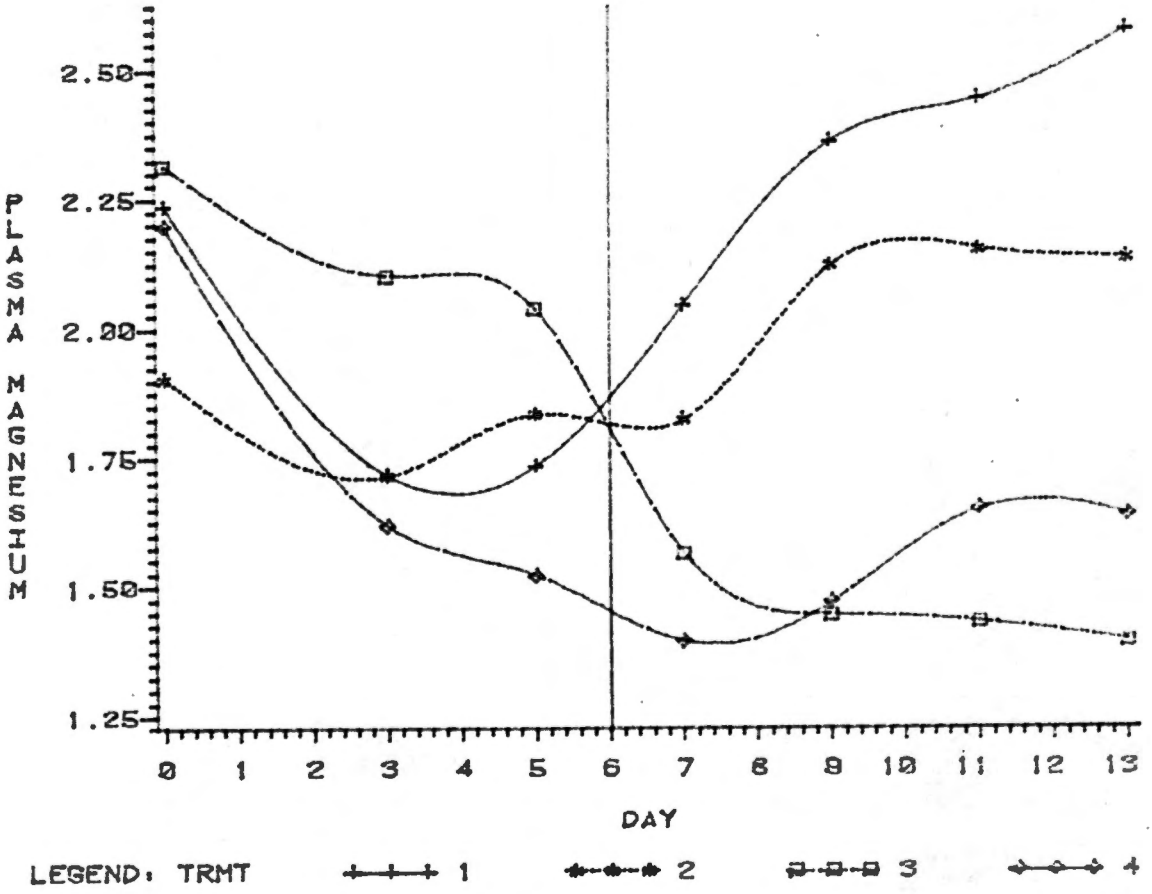

Figure 2. Changes in Plasma Mg: Trial II, 1982.

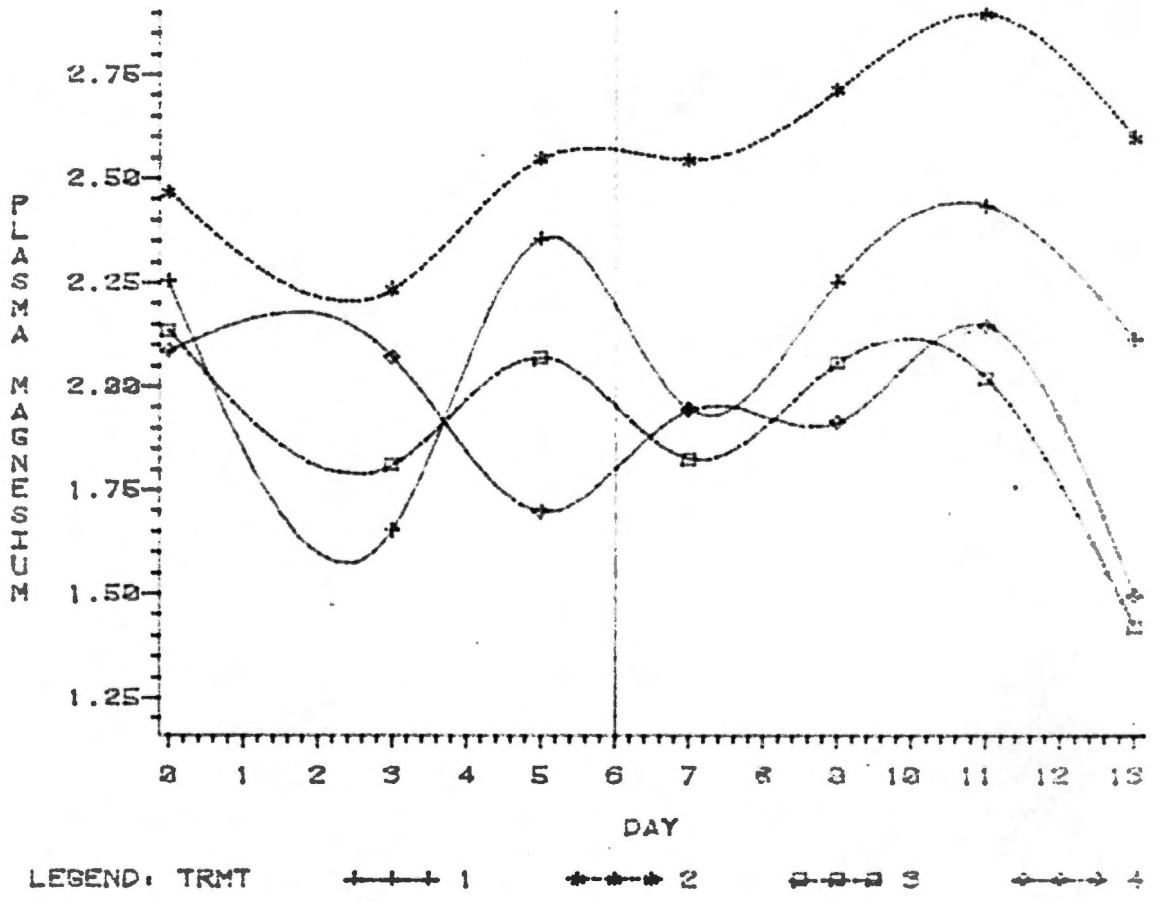

Figure 3. Changes in Plasma Mg: Trial I, 1983.

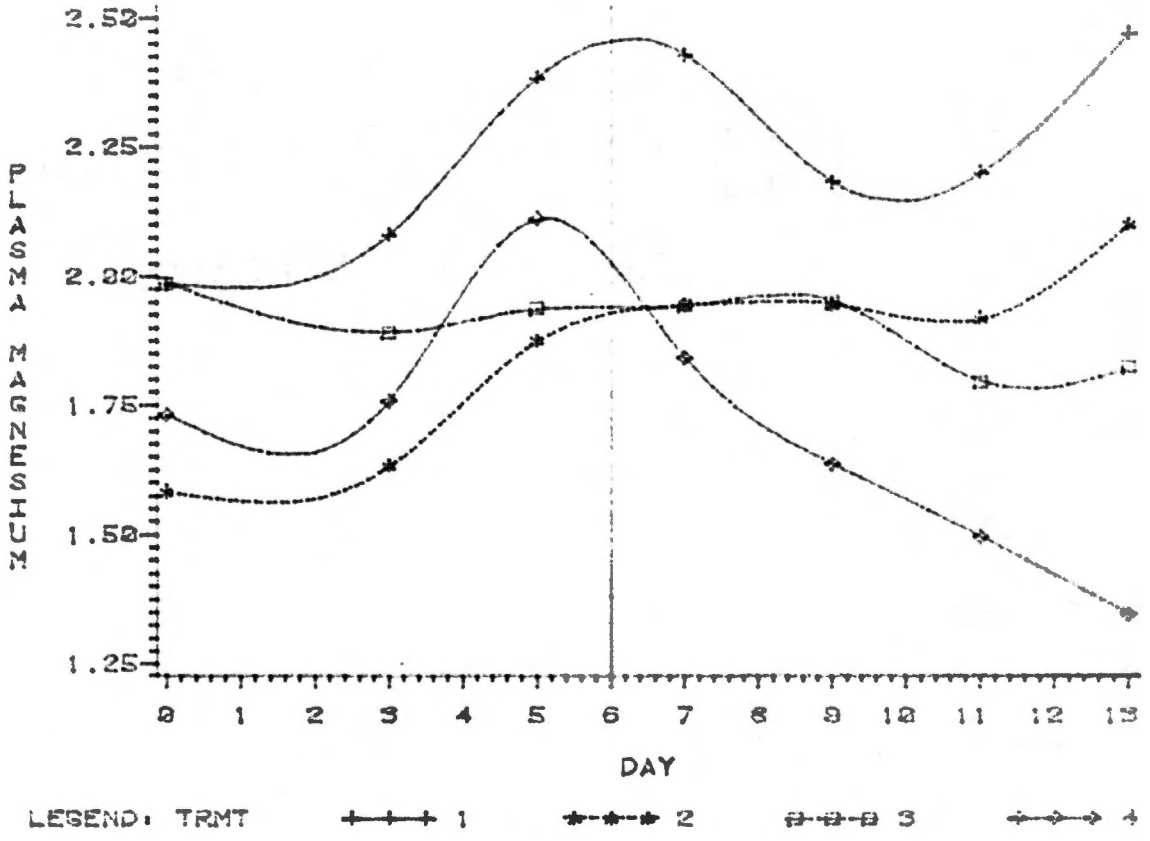

Figure 4. Changes in Plasma Mg: Trial II, 1983.

Table 20. Effect of Treatment on Plasma Mg (mg/dl) in Trial I and II in 1982

Treatment	Trial 1	Trial 2
1	1.977[a]	2.355[c]
2	1.782[a]	2.056[c]
3	1.358[b]	1.457[d]
4	1.305[b]	1.537[d]

[a,b]Means with different superscripts within columns are different ($P < .05$).

[c,d]Means with different superscripts within columns are different ($P < .01$).

Table 21. Effect of Treatment on Plasma Mg (mg/dl) in Trial I and II in 1983

Treatment	Trial 1	Trial 2
1	2.185[b]	2.319[a]
2	2.687[a]	1.975[b]
3	1.83[c]	1.877[b]
4	1.874[b,c]	1.577[c]

[a,b,c]Means with different superscripts within columns are different ($P < .05$).

these animals decreased through day 3, then increased throughout the remainder of the trial.

Table 22 presents a summary of the incidences of hypomagnesemia (< 1.5 mg of Mg/dl of plasma). There were 68 observations of hypomagnesemia which represented 21% of the total number of observations. Of these 68 observations, 54 (79%) occurred in cows on the K fertilized pasture. Of the animals on this pasture, 67% (16 out of 24) were hypomagnesemic at one time. Plasma Mg values of several cows were below 1.0 mg/dl, but at no time were any signs of tetany observed.

Table 22. Summary of the Incidences of Hypomagnesemia

Treatment	# of Observations below 1.5 mg of Mg/dl	% of Total	# of Animals	Minimum Value
1	4	6	3	.98
2	10	15	2	1.03
3	25	37	7	.34
4	29	42	9	.56
Total	68	21[a]	21	

[a]68 represents 21% of the total number of observations.

Plasma Mg levels were within the range reported by Rook and Storry (1962). It appears that the depression in plasma Mg of the animals in treatments 3 and 4 was due to a factor or factors in the

compositional make-up of the forage. Ramsey et al. (1979) showed a decrease in plasma Mg when cows were switched from a wintering ration to a lush spring pasture.

The excess K fertilization did increase the K content of the forage. It appears that the excess K interferes with Mg absorption in the gut, or Mg itself is tied up in the forage and is unavailable. Forage samples of the K fertilized pasture were notably greener in color when compared to the control pasture samples. From this observation, it appears that the K fertilization increased the chlorophyll content of the forage. Since chlorophyll is highly indigestible (Reid, 1962) and Mg is the center of the chlorophyll molecule (Lehninger, 1975), the increase in chlorophyll may have caused a decrease in Mg availability.

In Table 23 treatment effects on mean plasma Ca levels for 1982 and 1983 are presented. Changes in plasma Ca over time for 1982 and 1983 are presented in Figures 5 and 6. In both years, plasma Ca was variable and inconsistent. In 1982, plasma Ca was higher for the animals in treatment 1 ($P < .05$) than for those animals in treatments 2 and 3. Plasma Ca levels for animals in treatment 4 did not differ significantly from any other treatment. Animals in treatment 1 in 1983 had higher plasma Ca levels ($P < .05$) than did animals in treatment 3. Animals on treatments 2 and 4 did not differ from those on treatments 1 or 3.

Changes in plasma K over time are presented in Figures 7, 8, 9 and 10. Tables 24 and 25 contain plasma K means for trials 1 and 2 in

Table 23. Effect of Treatment on Plasma Ca (mg/dl)

Treatment	Year	
	1982	1983
1	10.24[a]	10.506[a]
2	9.89[b]	10.282[ab]
3	9.807[b]	10.166[b]
4	10.157[ab]	10.362[ab]

[a,b]Means with different superscripts within columns are different ($P < .05$).

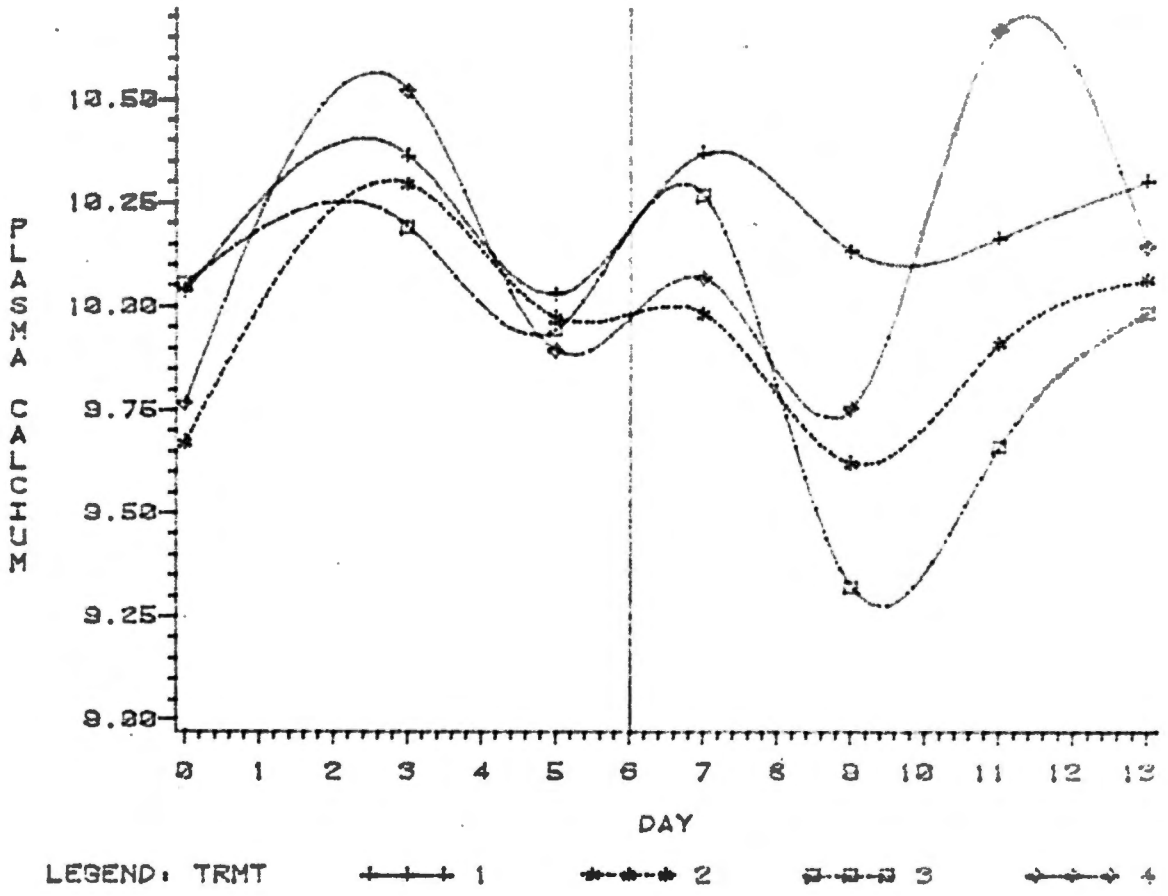

Figure 5. Changes in Plasma Ca in 1982.

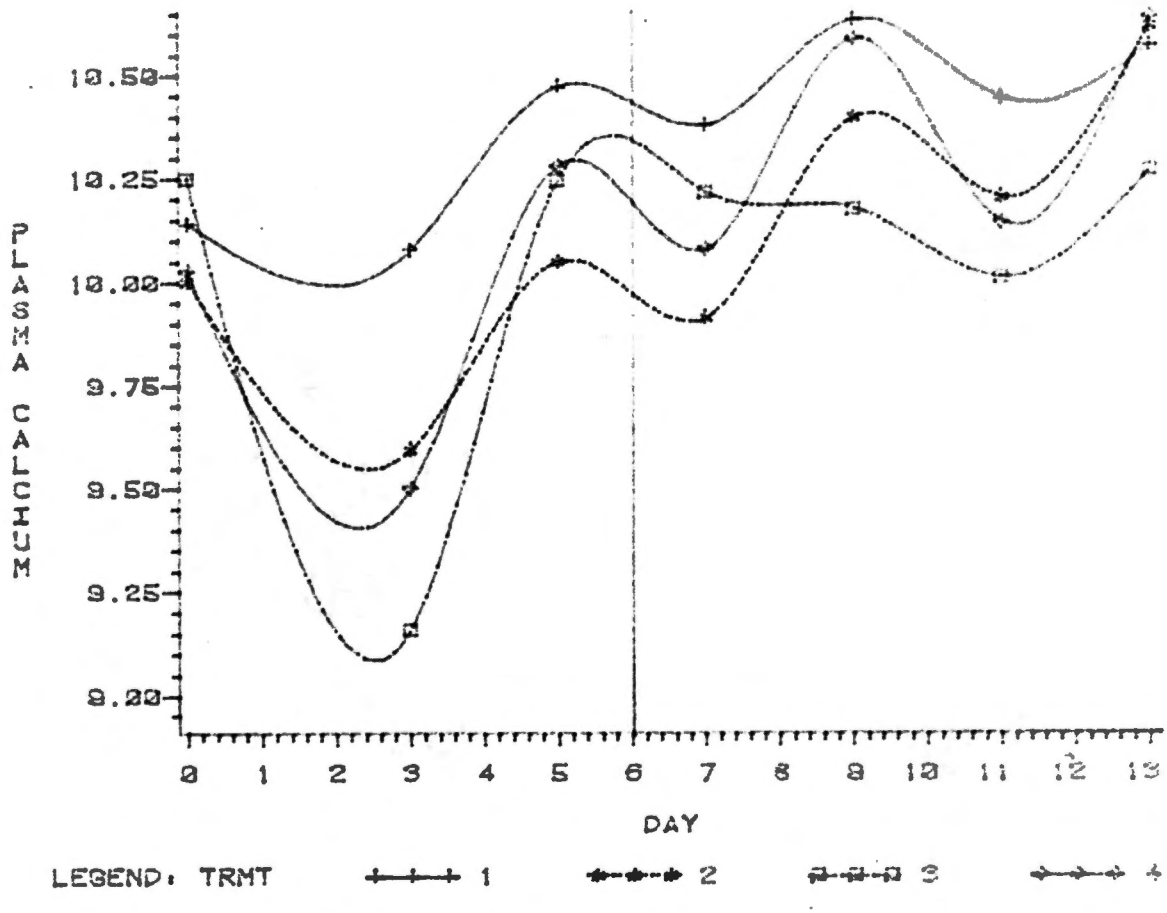

Figure 6. Changes in Plasma Ca in 1983.

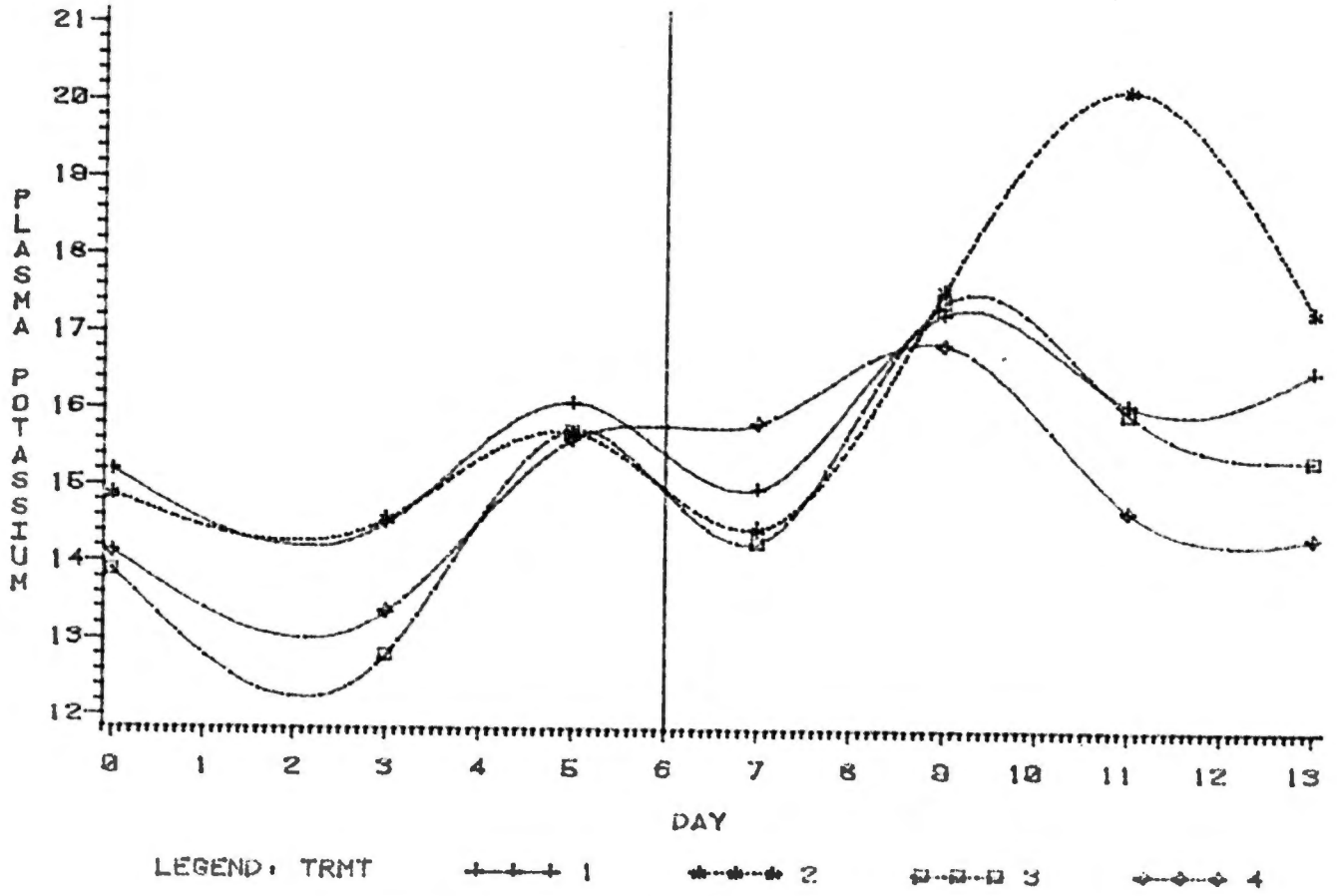

Figure 7. Changes in Plasma K: Trial 1, 1982.

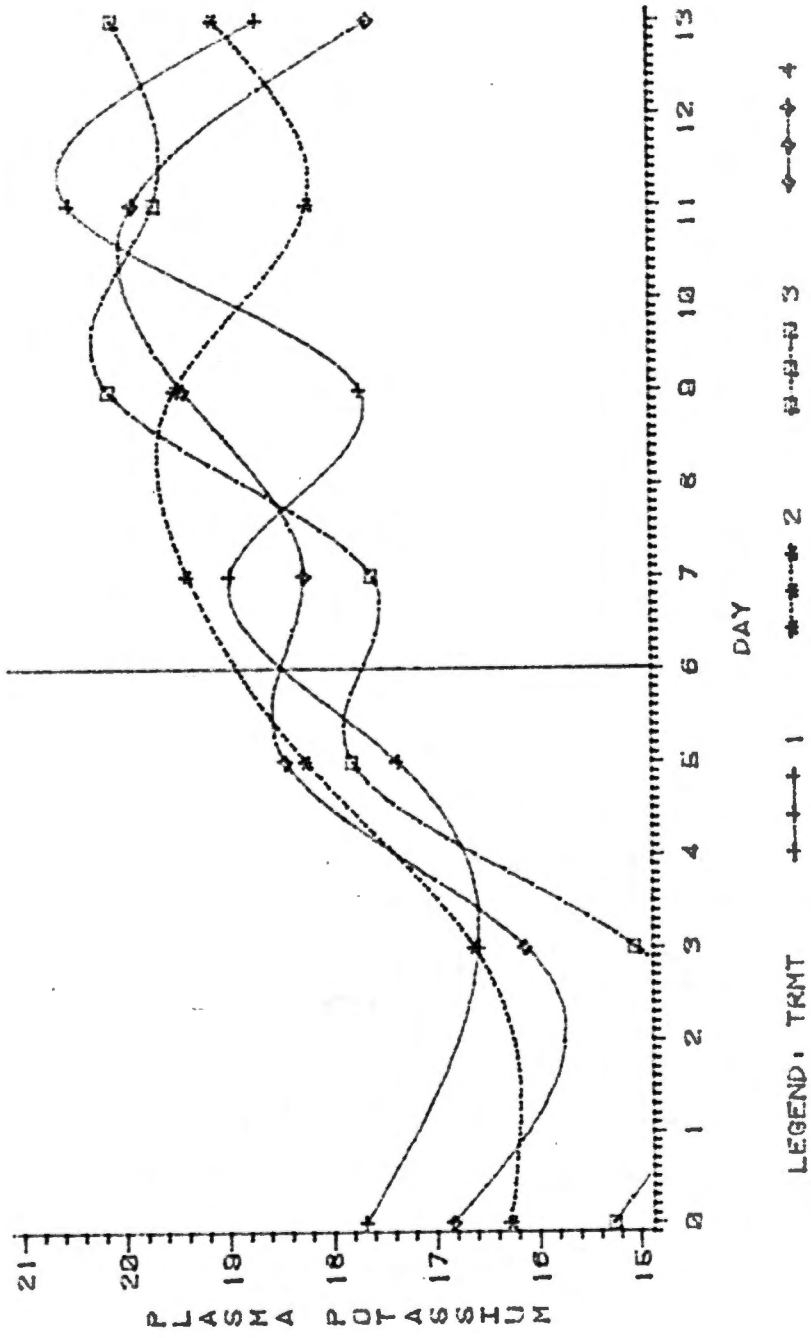

Figure 8. Changes in Plasma K: Trial 2, 1982.

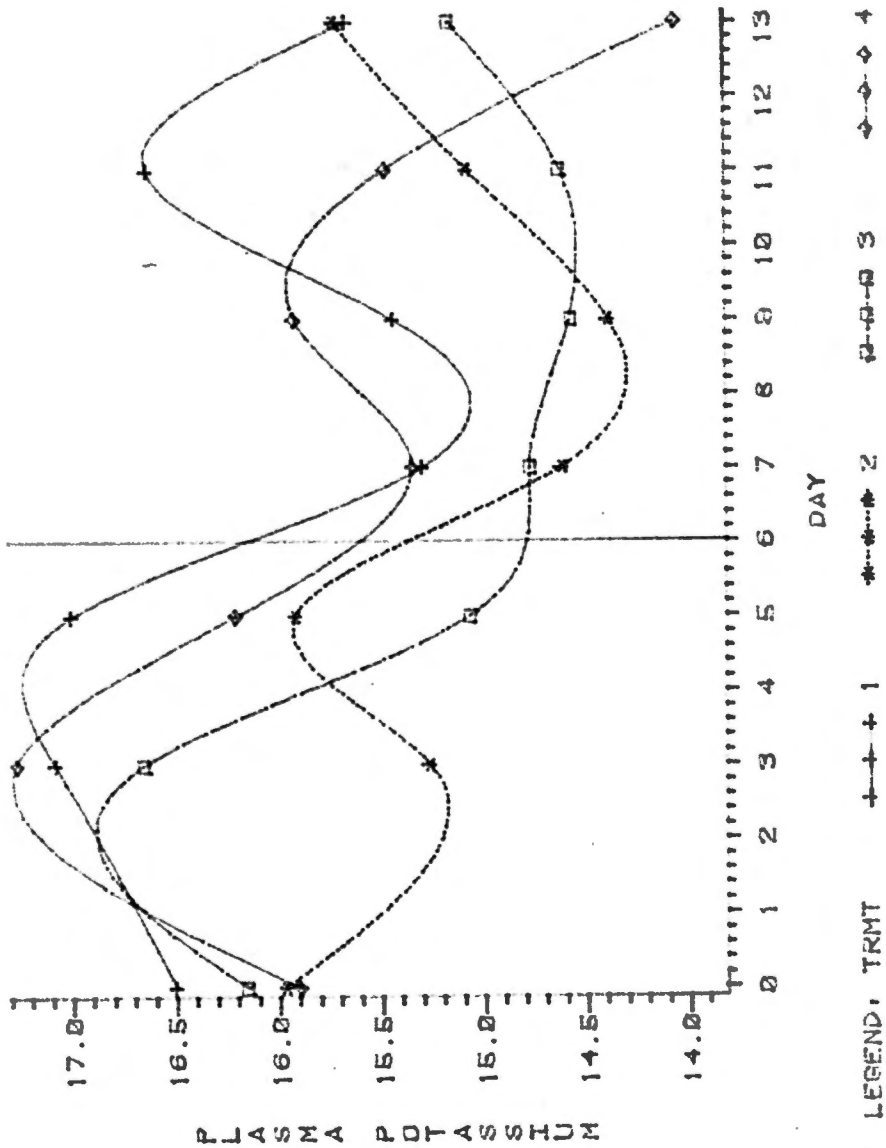

Figure 9. Changes in Plasma K: Trial 1, 1983.

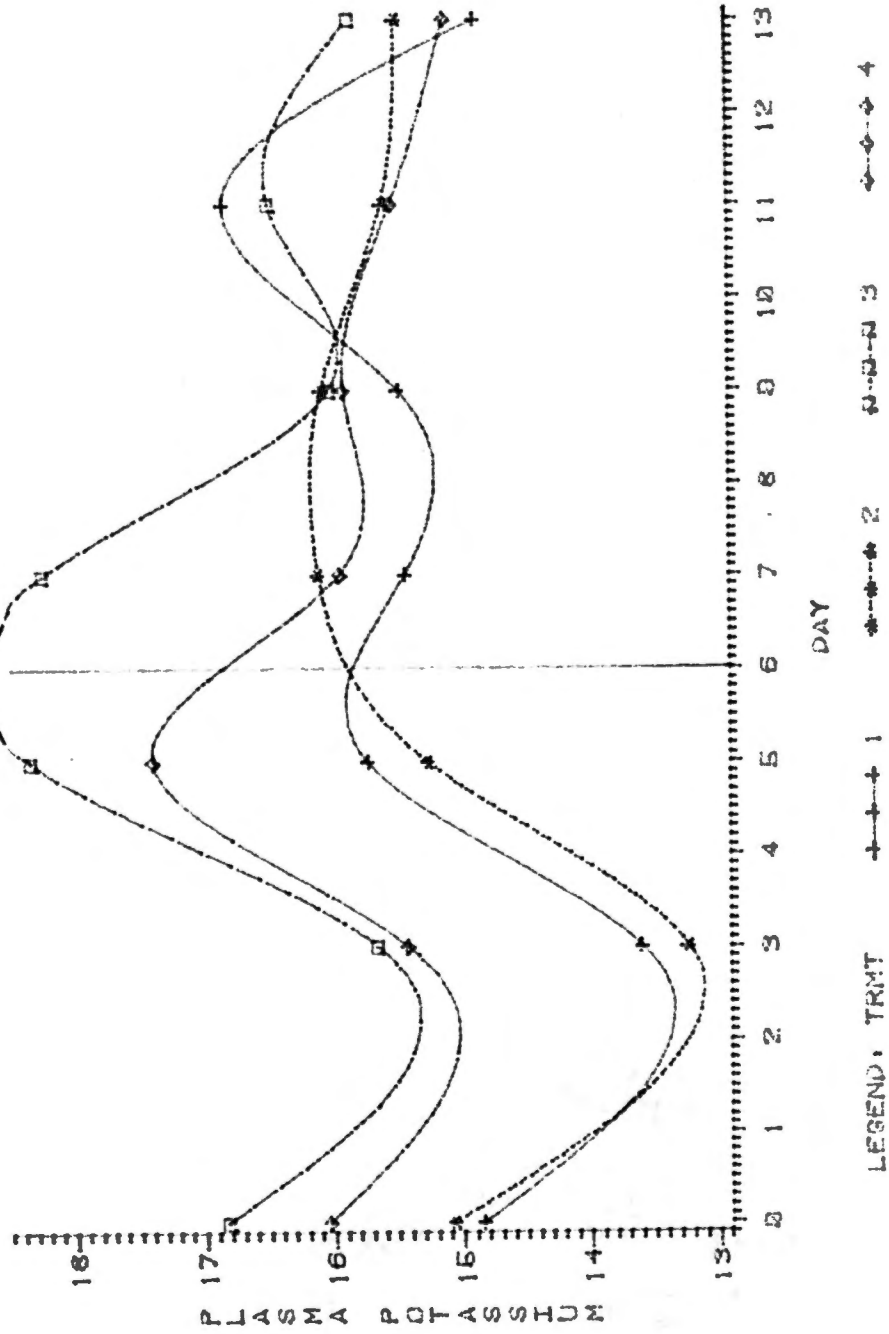

Figure 10. Changes in Plasma K: Trial 2, 1983.

Table 24. Effect of Treatment on Plasma K (mg/dl) in Trial I and II in 1982

Treatment	Trial 1	Trial 2
1	16.216[a]	18.961[a]
2	17.371[a]	19.041[a]
3	15.759[a]	19.375[a]
4	15.444[a]	18.787[a]

[a]Means within columns with different superscripts differ ($P < .05$).

Table 25. Effect of Treatment on Plasma K (mg/dl) in Trial I and II in 1983

Treatment	Trial 1	Trial 2
1	15.752[a]	15.647[a]
2	14.947[a]	15.809[a]
3	14.780[a]	16.645[a]
4	15.195[a]	15.613[a]

[a]Means within columns with different superscripts differ ($P < .05$).

1982 and 1983, respectively. There were no significant differences between treatments in all trials. It is hypothesized that if blood samples were drawn one to two hours post KCl bolusing, an increase in plasma K may have been observed. Reynolds (unpublished data) reported an increase in plasma K of calves when KCl was infused into the jugular vein. This same author also reported a rapid disappearance of K from the blood upon cessation of KCl infusion. These data indicate the importance of correlating time of blood sampling with the disappearance or appearance of the blood constituent of interest. As stated previously, there were no significant differences between treatments in either year. However, in both years, animals in trial 2 had higher plasma K levels (P .01). This is evidently a reflection of the higher K content of the forage which the animals were consuming as shown in Tables 3 and 4, page 25. However, plasma K was not affected by KCl bolusing. Here again, form of K may affect plasma K. It appears that the K in the forage is in some way different from the K in KCl. The unidentified factor or form of K may increase the availability or absorption of K.

In both trial 1 and trial 2 of 1982, plasma Mg decreased while plasma K increased. The increase in plasma K may be a reflection of increased K intake, while the decrease in plasma Mg is a result of a decrease in Mg intake. In trial 1 of 1983, plasma K was opposite of the effect seen in 1982. Plasma K decreased throughout the trial. No explanation is readily available for this trend, however the extremely dry winter of 1983 may have had an effect on the forage K and consequently on plasma K.

III. GENERAL DISCUSSION

Environmental factors have been implicated as a major cause of grass tetany. It is evident from the plasma data that the large differences seen in plasma minerals between years and trials were likely due to environmental factors. Kemp and t'Hart (1957) stated that grass tetany was more prevalent in cool weather. In both of the trials conducted in 1982, ambient temperatures were 5.0° and 8.7° above normal for trials 1 and 2 respectively. Kemp and t'Hart (1957) also stated that the incidence of grass tetany is greater in years that are normal in precipitation than in years that are exceptionally dry or wet. The winter and early spring of 1983 were among the driest on record. For the 8-week period from February 1 until March 31, 1983, only 4.9 inches of precipitation was recorded. The normal for this period is 9.75 inches. It is possible that the warmer than normal temperatures in 1982 and lack of precipitation in 1983 could have decreased the active agent or agents involved in grass tetany.

Fecal Mg excretion values were higher than data reported by Ritter (1979) and Hodge (1981). Crampton and Harris (1969) emphasized the importance of an adaptation period when using Cr_2O_3 as a marker. Their recommendations were to feed Cr_2O_3 for 10 days prior to the sampling period and a minimum of 50 grab samples per animal be collected to reduce the error to below 5%. In this study only 14 samples per animal were collected. These 14 samples were composited into 7 samples. This procedure may have caused the estimation of fecal dry

matter to be inflated which in turn increased the values of fecal Mg, Ca and K.

Bolusing of animals with KCl was initiated on day 5 of each trial. It is conceivable that an effect due to KCl bolusing on plasma constituents may have been seen if KCl treatment was started on day 0 of the trial. Also, due to the small number of animals per treatment per trial the statistical significance of the treatments was greatly influenced. It was anticipated at the beginning of the study that data could be pooled across trials or at least by year. This would have increased the number of animals per treatment and may have provided a clearer picture to some of the variability seen in the results.

It appears from these results and results reported by Hodge (1981) that increased K fertilization may increase the incidence of hypomagnesemia, but excess K fertilization does not seem to be the major causative agent of grass tetany. It appears that environmental factors such as ambient and soil temperature, amount of rainfall and sunlight or some other as of yet unidentified factor control the triggering of grass tetany.

CHAPTER V

SUMMARY

Four pasture balance trials were conducted in February and March of 1982 and 1983. The purpose of this study was to examine the effects of K fertilization of fescue pasture and dietary K on Mg, Ca and K metabolism. Plasma, fecal, urine and milk samples were collected.

Potassium fertilization decreased forage Mg and Ca concentrations. Potassium content of the forage was increased by K fertilization. In 1982, fecal Mg was increased ($P < .05$) by K fertilization. Effect of K fertilization on fecal Mg was variable in 1983. Potassium chloride bolusing had no effect on fecal Mg. Urinary Mg was unaffected by pasture fertilization or KCl treatment.

Plasma Mg was depressed by pasture fertilization in both years. Potassium chloride bolusing had no effect on plasma Mg. Sixteen of 24 animals on the K fertilized pasture became hypomagnesemic while only 5 of 24 controls became hypomagnesemic. Plasma Ca was variable and inconsistent. Plasma K was not effected by treatment. However, plasma K of animals in trial 2 of both years had significantly higher plasma K. This may be a reflection of the higher K content of the forage.

LITERATURE CITED

LITERATURE CITED

Allcroft, R. 1954. Hypomagnesemia in cattle. Vet. Rec. 66:517.

Allcroft, W. M. and H. H. Green. 1938. Seasonal hypomagnesemia of the bovine. New Zealand Vet. J. 16:109.

Albin, R. C. and D. C. Clanton. 1966. Factors contributing to the variation in urinary creatinine and creatine-nitrogen ratios in beef cattle. J. Anim. Sci. 25:107.

Allen, V. 1979. Aluminum and manganese in the etiology of grass tetany. Ph.D. dissertation, Louisiana State University, Baton Rouge, Louisiana.

Allsop, T. F. and J. A. F. Rook. 1972. Fecal magnesium output and plasma magnesium concentration in sheep. Proc. Nutr. Soc. 31:65A.

A.O.A.C. 1975. Official methods of analysis (12th Ed.). Association of Official Analytical Chemists, Washington, D.C.

Bartlett, S., B. B. Brown, A. S. Foot, S. J. Rowland, R. Allcroft and W. R. Parr. 1954. The influence of fertilizer treatment of grassland on the incidence of hypomagnesaemia in milking cows. Brit. Vet. J. 110:3.

Bell, M. C. 1980. Prevention of grass tetany. Proc. of the John Lee Pratt International Symp. on Role of Magnesium in Animal Nutrition, Blacksburg, VA.

Bell, M. C., J. A. Oluokun, N. Ramsey and G. E. Beckmann. 1978. Magnesium treatment of cows for grass tetany. Feedstuffs 50:24.

Ben-Ghedalia, D., H. Tagart, S. Zamwel and A. Bondi. 1975. Solubility and net exchange of calcium and phosphorus in digesta flowing along the gut of the sheep. Brit. J. Nutr. 33:87.

Bohman, V. R., A. L. Lespearance, G. D. Harding and D. L. Grunes. 1969. Induction of experimental tetany in cattle. J. Anim. Sci. 29:99.

Brown, R. C., A. D. Care and D. W. Pichard. 1978. Magnesium absorption from the rumen of the sheep. J. Physiol. 276:62.

Burau, R. and P. R. Stout. 1965. Transaconitic acid in range grasses in early spring. Science 150:766.

Burt, A. W. A. and D. C. Thomas. 1961. Dietary citrate and hypomagnesemia in the ruminant. Nature 192:1193.

Burts, K. N. and R. Allcroft. 1967. Hypomagnesemic tetany in cattle II. Preventable measures. Brit. Vet. J. 123:383.

Butler, E. J. 1963. The mineral element content of spring pasture in relation to the occurrence of grass tetany and hypomagnesaemia in dairy cows. J. Agric. Sci. 60:329.

Camp, B. J., J. W. Dollahite and W. L. Schwarz. 1968. Biochemical changes in sheep given transaconitic acid. Am. J. Vet. Res. 29:2009.

Care, A. D. 1967. Magnesium homeostasis in ruminants. World Res. Nutr. Diet 8:127.

Care, A. D. and A. T. Van't Klooster. 1965. In Vivo transport of magnesium and other cations across the wall of the gastrointestinal tract of sheep. J. Physiol. 177:174.

Chicco, C. F., C. B. Ammerman and P. E. Loggins. 1973. Effect of age and dietary magnesium on voluntary feed intake and plasma magnesium in ruminants. J. Dairy Sci. 56:822.

Crampton, E. W. and L. E. Harris. 1969. Applied Animal Nutrition (2nd Ed.). W. H. Freeman and Co., San Franciso, CA.

DeGroot, T. 1967. The grazing dairy cow in relation to its environment under intensive farming conditions. In Rep. Proc. 9th int. congress on animal production (Edinburgh, Scotland, 1966). Oliver and Boyd, London.

Dryerre, H. 1932. Lactation tetany. Vet. Rec. 12:1163.

Ellis, R. 1979. Influence of soil, liming, magnesium, potassium and nitrogen on magnesium composition of plants. Amer. Soc. Agron. Spec. Publ. 35:1.

Field, A. C. 1962. Studies on magnesium in ruminant nutrition IV. Balance trials on sheep given different amounts of grass nuts. Brit. J. Nutr. 16:99.

Field, A. C. 1964. Studies on magnesium in ruminant nutrition. Brit. J. Nutr. 18:357.

Field, A. C. and C. S. Munro. 1977. The effect of site and quantity on the extent of absorption of magnesium infused into gastrointestinal tract of sheep. J. Agric. Sci. 89:365.

Field, A. C. and N. F. Suttle. 1979. Effect of high potassium and low magnesium intakes on the mineral metabolism of monozygotic twin cows. J. Comp. Path. 89:431.

Fisher, D. D., L. L. Wilson, P. C. Lootens and R. W. Scholz. 1978. Grass tetany in beef cows on low magnesium pastures. Sci. in Agric. 25:10.

Fontenot, J. P., R. W. Miller, C. K. Whitehair and R. MacVicar. 1960. Effect of high-protein high-potassium ration on mineral metabo lism of lambs. J. Anim. Sci. 19:127.

Fontenot, J. P., H. J. Gerken, S. L. Kalison, D. F. Watson and R. W. Engel. 1965. Value of magnesium in controlling grass-tetany in cattle. Feedstuffs 37(12):66.

Fontenot, J. P. 1979. Animal nutrition aspects of grass tetany. Amer. Soc. Agron. Spec. Publ. 35:51.

Foot, A. S., J. Connell, R. A. Allcroft and M. K. Lloyd. 1969. Magnesium bullets. Vet. Rec. 84:467.

Frye, T. M., J. P. Fontenot and K. E. Webb, Jr. 1975. Effects of dietary potassium level on utilization of magnesium by sheep fed different magnesium levels. V.P.I. and S.U. Res. Div. Rep. 163:80.

Garner, W. W., J. E. McMurtrey, C. W. Bacon and E. G. Moss. 1923. Sand drown, a chlorosis of tobacco, due to magnesium deficiency and the relationship of sulfate and chloride of potassium to the disease. J. Agric. Res. 40:27.

Grace, N. D., M. J. Wyatt and J. C. MacRae. 1974. Quantitative movement of fresh herbage by sheep. II. The movement of magnesium, calcium, phosphorus, potassium and sodium in the digestive tract. J. Agri. Sci. 82:321.

Greene, L. W., J. P. Fontenot and K. E. Webb, Jr. 1983. Effect of dietary K on absorption of magnesium and other macroelements in sheep fed different levels of magnesium. J. Anim. Sci. 56:1208.

Grunes, D. L., P. R. Stout and J. R. Brownell. 1970. Grass tetany of ruminants. Adv. Agron. 22:331.

Hall, R. F. and R. A. Reynolds. 1972. Concentrations of magnesium and calcium in plasma of Hereford cows during and after hypomagnesemic tetany. Am. J. Vet. Res. 33:1711.

Hemingway, R. G., N. S. Ritchie, A. R. Rutherford and G. M. Jolly. 1963. Effect of potassium fertilizers, age of ewe and small magnesium supplementation on blood magnesium and calcium levels of lactating ewes. J. Agri. Sci. 60:307.

Henry, P. R., W. H. Smith and M. D. Cunningham. 1977. Effect of histamine and ammonia on hypomagnesemia in ruminants. J. Anim. Sci. 44:276.

Herd, R. P. 1966. Fasting in relation to hypocalcemia and hypomagnesaemia in lactating cows and ewes. Aust. Vet. J. 42:269.

Hodge, D. L. 1981. Effects of potassium fertilization of fescue pastures on metabolism of magnesium, calcium and potassium in lactating beef cows. Master's thesis, The University of Tennessee, Knoxville.

Horvath, D. J., L. Dazea, H. E. Kedder, J. E. Warren, Jr., B. Bhatt and W. Croleshore. 1967. Magnesium supplement for cattle on spring pasture. J. Anim. Sci. 26:875 (Abstr.).

Horvath, D. J. and J. R. Todd. 1968. Magnesium supplements for cattle. P. 96-104. In 23rd annual Texas nutrition conference proceedings. Texas A&M University, College Station, TX.

House, W. A. and H. F. Mayland. 1976. Magnesium and calcium utilization in sheep treated with magnesium alloy rumen bullets or fed magnesium sulfate. J. Anim. Sci. 42:506.

House, W. A. and D. VanCampen. 1971. Magnesium metabolism of sheep fed different levels of potassium and citric acid. J. Nutr. 101:1483.

Jesse, B. W., J. W. Thomas and R. S. Emery. 1981. Availability of magnesium from magnesium oxide particles of differing sizes and surfaces. J. Dairy Sci. 64:197.

Kemp, A. and J. L. t'Hart. 1957. Grass tetany in grazing milking cows. Neth. J. Agri. Sci. 5:4-17.

Kemp, A. 1958. Influence of fertilizer treatment of grassland on the incidence of hypomagnesemic tetany (grass tetany) in milking cows. Neth. J. Agri. Sci. 6:281.

Kemp, A. 1960. Hypomagnesemia in milking cows; the response of serum magnesium to alterations in herbage composition resulting from potash and nitrogen dressings on pasture. Neth. J. Agri. Sci. 8:281.

Kemp, A., W. B. Deegs, O. J. Hemkes and A. J. H. VanEs. 1961. Hypomagnesaemia in milking cows: Intake and utilization of magnesium from herbage by lactating cows. Neth. J. Agri. Sci. 9:134.

Kemp, A. and J. R. Todd. 1970. Prevention of hypomagnesaemia in cows: The use of magnesium alloy bullets. Vet. Rec. 86:463.

Kennedy, G. S. 1968. Trans-aconitate utilization by sheep. Aust. J. Biol. Sci. 21:529.

Kunkel, H. O., K. H. Burns and B. J. Camp. 1953. A study of sheep fed high levels of potassium bicarbonate with particular reference to indirect hypomagnesemia. J. Anim. Sci. 12:451.

Lentz, D. E., F. C. Madsen, J. K. Miller and S. L. Hansard. 1976. Effect of K insulin in the bovine. J. Anim. Sci. 43:1082.

Lidgate, H. J. 1976. Effects of timing of potassium fertilization of grass on yields and herbage magnesium levels. J. Brit. Grassl. Soc. 31:43 (Abstr.).

Line, C., M. J. Head, J. A. F. Rook, A. S. Foot and S. J. Rowland. 1958. Investigation into the use of supplements for the control of hypomagnesemia in dairy cows during the spring grazing period. J. Agri. Sci. 51:353.

Linkous, W. N., W. A. Hardison, G. C. Graf and E. W. Crampton. 1955. Fecal chromic oxide concentration in twelve dairy cows as related to time and frequency of administration and to feeding schedule. J. Anim. Sci. 13:1009 (Abstr.).

Lehninger, A. L. 1975. Biochemistry (2nd Ed.). Worth Publishers, Inc., New York, NY.

Metson, A. J. 1974. Magnesium in New Zealand soils. I. Some factors governing the availability of soil magnesium. N.Z.J. Exp. Agric. 2:277.

Martens, H., J. Harmeyer and M. Michael. 1978. Magnesium transport by isolated rumen epithelium of sheep. Res. Vet. Sci. 24:111.

Mayland, H. F. and D. L. Grunes. 1979. Soil-climate-plant relationships in the etiology of grass tetany. P. 123-175. In V. V. Rendug and D. L. Grunes (eds.), Grass tetany. Am. Soc. of Agron., Madison, Wis.

McConaghy, S., J. S. V. McAllester, J. R. Todd, J. E. F. Rankin and J. Kerr. 1963. The effects of magnesium compounds and of fertilizers on the incidence of hypomagnesaemia on dairy cows. J. Agri. Sci. 60:313.

McLaren, J. B., D. W. Saylor, S. L. Hansard and L. M. Safley. 1975. Effects of supplemental magnesium on the incidence of grass tetany in beef cows. Tenn. Farm Home Sci. 94:21.

Merck. 1979. The Merck Veterinary Manual. (5th Ed.). O. H. Siegmund (ed.). Merck and Co., Inc., Rahway, NJ.

Metson, A. J., W. M. H. Saunders, T. W. Callie and V. W. Graham. 1966. Chemical composition of pastures in relation to grass tetany in beef breeding cows. N.Z.J. Agri. Res. 9:410.

Meyer, H. 1976. The pathogenesis and prophylaxis of hypomagnesemic tetany in ruminants. P. 66-76. In U. Tavan (ed.), Magnesium in ruminant nutrition. Israel Chem. Ltd., Tel Aviv, Israel.

Meyer, H. and D. W. Busse. 1975. Zur Rekstalen absorption von magnesium bien wiederkaur. Dtsch. tierarstl. wschr. 82:137.

Moore, W. F., J. P. Fontenot and K. E. Webb. 1972a. Effect of form and level of nitrogen on magnesium utilization. J. Anim. Sci. 35:1046.

Moore, W. F., J. P. Fontenot and K. E. Webb. 1972b. Effect of level of nitrogen and potassium on magnesium utilization. J. Anim. Sci. 35:271.

N.R.C. 1975. Nutrient requirements of domestic animals. No. 5. Nutrient requirements of sheep. National Research Council, Washington, D.C.

N.R.C. 1976. Nutrient requirement of domestic animals. No. 4. Nutrient requirements of beef cattle. National Research Council, Washington, D.C.

N.R.C. 1978. Nutrient requirements of domestic animals. No. 4. Nutrient requirements of dairy cattle. National Research Council, Washington, D.C.

Newton, G. L., J. P. Fontenot, R. E. Tucker and C. E. Polan. 1972. Effects of high dietary potassium intake on the metabolism of magnesium by sheep. J. Anim. Sci. 35:440.

Nicholson, M. A. and G. D. Shearer. 1938. The occurrence of lactation tetany in cattle. Vet. J. 94:388.

O'Kelley, R. E. and J. P. Fontenot. 1969. Effects of feeding different magnesium levels to drylot-fed lactating beef cows. J. Anim. Sci. 29:959.

Parr, W. H. and R. Allcroft. 1957. The application of magnesium compounds to pasture for the control of hypomagnesemia in grazing cattle: A comparison between magnesium limestone and calcined magnesite. Vet. Rec. 69:1041.

Powley, K., R. L. Reid and J. A. Balska. 1978. Performance of lambs on perennial ryegrass smooth brome, orchardgrass and tall fescue pastures. II. Mineral utilization in vitro digestibility and chemical composition. J. Anim. Sci. 46:1503.

Ramsey, N., J. K. Miller and M. C. Bell. 1979. Hypomagnesemia and insulin in cows over-wintered on stockpiled fescue pasture or changed abruptly from stored feed to lush spring pasture. Tenn. Farm Home Sci. 112:16.

Rayssiguier, Y. and C. Poncet. 1980. Effect of lactose supplement on digestion of lucerne hay by sheep. II. Absorption of magnesium and calcium in the stomach. J. Anim. Sci. 51:186.

Reid, J. T. 1962. In: Pasture and Range Research Techniques. Am. Soc. Agron., Comstock Publ. Assoc., Cornell Univ. Press, Ithaca, NY.

Reynolds, J. H. 1980. Slurry applications to increase magnesium in fescue forage. Tenn. Farm Home Sci. 113:9.

Ritchie, N. S. and G. Fishwick. 1977. Magnesium phosphate as a dietary supplement for lactating cows at spring pasture. J. Agri. Sci. 88:71.

Ritchie, N. S. and R. G. Hemingway. 1968. Magnesium alloy bullets for dairy cattle. Vet. Rec. 82:87.

Ritchie, N. S., R. G. Hemingway, J. S. S. Inglis and R. M. Peacock. 1962. Experimental production of hypomagnesaemia in ewes; its control by small magnesium supplements. J. Agri. Sci. 58:339.

Ritter, R. S. 1979. Hypomagnesemia effects on magnesium, calcium and potassium balance in lactating beef cows. Master's Thesis, The University of Tennessee, Knoxville.

Rogers, P. A. M. and D. B. R. Poole. 1971. Control of hypomagnesaemia in cows: A comparison of foliar dusting with free access to calcined magnesite-molasses mix. Irish Vet. J. 25:197.

Rogers, P. A. M. and D. B. R. Poole. 1976. Control of hypomagnesaemia in cows: A comparison of magnesium acetate in the water supply with magnesium oxide in the feed. Irish Vet. J. 30:129.

Rogers, G., R. Porter, L. C. Jolley and D. D. Learer. 1977. Management factors and grass tetany in dairy cattle. Australian Vet. J. 53:523.

Rook, J. A. F. and C. C. Balch. 1958. Magnesium metabolism in the dairy cow. II. Metabolism during the spring grazing season. J. Agri. Sci. 51:199.

Rook, J. A. F. and J. E. Storry. 1962. Magnesium in the nutrition of farm animals. Nutr. Abstr. Rev. 32:1055.

Ross, J. G. and W. G. Halliday. 1975. Surveys of bovine blood chemistry in Scotland. I. Serum magnesium. Br. Vet. J. 131:309.

S.A.S. 1982. SAS Users' Guide. Statistical Analysis System Institute, Cary, NC.

Scotto, K. C., V. R. Bohman and A. L. Lesperance. 1971. Effects of oral potassium and sodium chloride on plasma composition of cattle: A grass tetany related study. J. Anim. Sci. 32:354.

Sims, F. H. and H. R. Crookshank. 1956. Wheat pasture poisoning. Texas Agri. Expt. Sta. Bull. 842.

Sjollema, B. 1932. Nutritional and metabolic disorders in cattle. Nutr. Abstr. Rev. 1:621.

Smith, R. H. 1959. Calcium and magnesium metabolism in calves. 3. Endogenous fecal excretion and absorption of magnesium. Biochem. J. 71:306.

Smyth, A. J., A. Conway and M. J. Walsh. 1958. The influence of different fertilizer treatments on the hypomagnesaemia proneness of a ryegrass sward. Vet. Rec. 70:846-849.

Sokal, R. R. and F. J. Rohlf. 1969. Biometry. W. H. Freeman and Co. San Francisco, CA.

Stuedemann, J. A., S. R. Wilkinsen, W. A. Jackson and J. B. Jones, Jr. 1973. Foliar dusting with magnesium oxide to prevent hypomagnesemia and/or grass tetany. J. Anim. Sci. 36(1):227. (Abstr.).

Stewart, B. A., D. L. Grunes, A. C. Mathes and F. P. Hern. 1981. Chemical composition of winter wheat forage grown where grass tetany and bloat occur. Agron. J. 73:337.

Stewart, J. and E. W. Moodie. 1956. The absorption of magnesium from the alimentary tract of sheep. J. Comp. Path. Therap. 66:10.

Stillings, B. R., J. W. Bratzler, L. F. Mariot and R. C. Miller. 1964. Utilization of magnesium and other minerals by ruminants consuming low and high nitrogen containing forages and vitamin D. J. Anim. Sci. 23:1148.

Stout, P. R., J. Brownell and R. G. Burau. 1967. Occurrences of trans-aconitate in range forage species. Agron. J. 59:21.

Suttle, N. F. and A. C. Field. 1969. Studies on magnesium in ruminant nutrition. IX. Effect of potassium and magnesium intakes on level-aponent of hypomagnesaemia in sheep. Brit. J. Nutr. 23:81.

Todd, J. R. and N. E. Morrison. 1964. Control of hypomagnesaemic tetany by foliar application of calcined magnesite. J. Brit. Grassl. Soc. 19:179.

Todd, J. R., W. C. P. Seally and J. M. Ingram. 1966. Studies of the effectiveness of "free access" feeding of a magnesia-molasses mixture for the prevention of hypomagnesemia in dairy cows at pasture. Vet. Rec. 78:888.

Tomas, F. M. and B. J. Potter. 1976a. The site of magnesium absorption from the ruminant stomach. Brit. J. Nutr. 36:37.

Tomas, F. M. and B. J. Potter. 1976b. Interaction between sites of magnesium absorption in the digestive tract of the sheep. Austr. J. Agr. Res. 27:437.

Van Soest, P. J. and R. H. Vine. 1967. Use of detergent in the analysis of fibrous feed. IV. Determination of plant cell wall constituents. J. Assoc. Agri. Chem. 50:50.

Wilcox, G. E. and J. E. Huff. 1974. Grass tetany: An hypothesis concerning its relationship with ammonium nutrition of spring grasses. J. Dairy Sci. 57:1085.

Wilkinson, S. R. and J. A. Stuedemann. 1979. Tetany hazard of grass as affected by fertilization with nitrogen, potassium or poultry litter and methods of grass tetany prevention. In, Grass Tetany, A.S.A. Sp. Publ 35:93.

Wilkinson, S. R., J. A. Stuedemann, J. B. Jones, Jr., W. A. Jackson and I. W. Dobsin. 1972. Environmental factors affecting magnesium concentrations and tetanigenicity of pastures. Chapter 6. Magnesium in the environment. Fort Valley State College, Fort Valley, GA, p. 163.

Williams, C. H., C. J. David and O. Ismaa. 1962. The determination of chromic oxide in faeces samples by atomic absorption spectrophotometry. J. Agri. Sci. 59:381.

Wilson, A. A. 1964. Hypomagnesemia and magnesium metabolism. Vet. Rec. 76:1382.

Young, P. W. 1975. Magnesium supplementation for the prevention of grass staggers in cattle. Raukuara Farmers Conference Proc., New Zealand, p. 39-45.

VITA

Stephen Richard Banet, son of Richard and Elizabeth Banet, was born in Princeton, Indiana, on November 7, 1958. He attended elementary and secondary schools in Princeton. Upon graduation from Princeton Community High School, he enrolled at Purdue University and received the Bachelor of Science degree in Agriculture in May of 1981. He immediately entered graduate school at The University of Tennessee, Knoxville, and received the Master of Science degree in Animal Science in December 1983.