THE EFFECTS OF VARIOUS DIETARY LEVELS OF POTASSIUM ON SUBSEQUENT PARTURITION AND LACTATION IN THE DAIRY COW

Thesis for the Degree of M. S.

MICHIGAN STATE UNIVERSITY

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ABSTRACT

THE EFFECTS OF VARIOUS DIETARY LEVELS OF POTASSIUM
ON SUBSEQUENT PARTURITION AND LACTATION
IN THE DAIRY COW

By

David L. Bolenbaugh

Twenty-four multiparous cows were fed four dietary levels of potassium during late gestation, parturition and early lactation. Dietary levels (as percent of dry matter) were 0.37, 0.59, 0.74 and 1.68%. Cows were individually fed their respective diets beginning 35 days before expected calving date and extended through 35 days of lactation. Cows were bled twice weekly during the prepartum and post-partum periods, and at four-hour intervals during parturition.

Signs common to potassium deficiency were observed in cows receiving the 0.37% potassium diet which included: marked depression in appetite, emaciation, incoordination, gradual dulling of hair coats, listlessness and evidence of ketosis. Cows fed the 0.37% potassium diet experienced an 84% greater ($P<.05$) weekly body weight loss, produced 10.5 kg less daily milk ($P<.05$), and experienced a greater incidence of health disorders than cows receiving the higher potassium diets. Dietary potassium caused a significant ($P<.05$) linear response in the level of both milk sodium and potassium, such that, sodium replaced potassium in milk from cows fed the 0.37% diet,

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while the inverse occurred in the 1.68% diet and potassium replaced sodium. Dietary level of potassium was linearly related ($P<.05$) to serum concentrations of magnesium, creatinine, alkaline phosphatase, glutamic pyruvic transaminase, creatine phosphokinase, glutamic oxalacetic transaminase, blood urea nitrogen and glucose. A curvilinear relationship ($P<.05$) existed between dietary potassium and serum sodium, potassium, phosphorus and cholesterol concentrations.

Regression of blood serum parameters, feed intake and milk yield upon dietary potassium indicates optimum allowances of 0.9 and 1.2% potassium during the dry period, and 1.2% of ration dry matter during lactation.

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By

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TABLE OF CONTENTS

Tab

1

2

3

4.

5.

6.

7.

8.

9.

10.

11.

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Initial interest in the area of mineral nutrition appeared as an offshoot of disclosures that certain minerals were necessary elements in life supporting mammalian systems. Early recognition for the importance of potassium was best emphasized in a statement from a letter that Leibig in 1847 sent to Hoffman:

> "I see a boundless field before me, and doubt not that for every quality of the animal body, something which can be estimated quantitatively will be discovered to which it is indebted for its properties." He then went on to say, "I have found that the fluids without the blood and lymphatic vessels contain only potash and phosphate of magnesia, whilst the blood and lymph contain merely those of phosphate of soda. If, therefore, the latter is indispensible to the formation of blood and the processes of life, it is evident that an animal on the continent, which finds in plants only potash salts, should have chloride of sodium given to it, by means of which the phosphate of potash is transformed into chloride of potash and phosphate of soda," as cited by McCollum (1957).

Following Leibig's predictions and observations on the contrasting distributions of sodium and potassium in biological fluids, a number of scientifically verified reports appeared. One such report by Ringer in 1885, as cited by McCollum (1957), noted that functional integrity of organic structures were best preserved in solutions containing a critical ratio of the salts sodium chloride, potassium chloride, and calcium chloride.

By the early 1900s, investigators realized that a complete and thorough understanding of the nutritional importance of potassium

would necessitate the induction of a state of potassium malnutrition in otherwise adequate diets. A classical study of this nature was conducted by Orent-Kieles and McCollum (1941), in which, reduced growth rates, lowered feed consumption, and delayed sexual maturity was produced in young growing rats fed diets containing 0.01% potassium. These rats were characterized as showing a roughness and thinning of fur, a striking alertness and a peculiar appetite termed pica, which involved hair licking and biting. Low potassium intakes did not effect body potassium balance in these rats, but changes were noted in magnesium and chloride body pools. In addition, a reciprocal relationship was noted in heart and kidney tissues in which sodium replaced potassium.

Since these early studies, potassium has been established as an essential mineral for animals. Similar consequences following low potassium intakes have been reported in the chick (Ben Dor, 1941; Gillis, 1948, 1950; Leach et al., 1959; Rinehart et al., 1968), growing swine (Cox et al., 1966; Leibholz, 1966), finishing lambs (Telle et al., 1964; Campbell and Roberts, 1965), finishing steers (Roberts and St. Omer, 1965; Devlin et al., 1969), growing heifers (St. Omer and Roberts, 1967), and lactating dairy cows (DuToit et al., 1934; Pradhan and Hemken, 1968; Dennis and Hemken, 1975; Dennis et al., 1976).

Although much information on the physiological function and body distribution of potassium has accumulated in the literature, there still exists a remarkable shortage of information concerning the dietary requirements for potassium in the dairy cow during late gestation and early lactation.

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LITERATURE REVIEW

Biological Activity of Potassium

The content of potassium in the body is similar to sodium, but unlike the latter, it exists primarily as a cellular constituent. Intracellularly, potassium is mainly in the ionic state and apparently serves the same general function relating to osmotic pressure regulation and acid-base balance in the cell as does sodium in extracellular fluid. According to conventional theory, potassium moves out of the cell along a chemical gradient and into the cell following an electrical gradient or as a result of expenditures of metabolic energy (Hodgkin, 1958). The concept of membrane permeability necessitated the movement of potassium coupled to a sodium-potassium pump, which promotes high intracellular potassium by the active extrusion of sodium from within the cell. Studies involving red blood cells have shown that increasing either extracellular potassium or intracellular sodium will lead to activation of membrane-situated ATP-ase causing increased influx of potassium and efflux of sodium in the cell (Whittam, 1962). The integrity of this mechanism is dependent not only on cellular adenosine triphosphate (ATP), but upon the absolute concentrations of other cations which directly or indirectly affect ATP-ase activity (Lowell and Bokin, 1963).

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Potassium ions influence many essential life supporting systems. These include the following: electrical activity of nerve and muscle cells and the process of synaptic transmission; distribution of ions and water between fluid compartments and regulation of interstitial and intracellular pH; secretive (Smith and Blaxter, 1963) and absorptive processes (Quastel, 1964) of the gastiointestinal tract; and the concentration of urine by the kidney. In addition, potassium ions are cofactors in the following enzyme systems: ATP-pyruvate phosphotransferase; myosine ATP-ase; phosphorylation of adenylic acid (Boyer et al., 1942, 1943); and the functional activity of choline acetylase (Nachmansohm and John, 1944). Moreover, evidence suggest that phosphocreatine, which serves as a reservoir of energy during rephosphorylation of adenosine diphosphate (ADP), exists in skeletal muscle as a dipotassium salt (Myers and Mangium, 1940).

Potassium is quantitatively the most prominent cation in intracellular fluid and much evidence has accumulated linking it with carbohydrate metabolism. Fenn (1939) noted that the potassium content of rat liver paralleled the glycogen content and that deposition of liver glycogen is accompanied by increased hepatic potassium as well as water content. In addition, Hasting et al. (1952) demonstrated the necessity of providing a perfusion medium high in potassium for achieving optimum glycogenesis in liver slices. Of interest are several observations (Heppel, 1939; Dodgen and Muntwyler, 1958; Spergel et al., 1967) that during moderate or short term potassium depletion in the rat, both muscle and liver glycogen content increase, but paradoxically, hepatic potassium remains unaltered although in

the presence of a sharp reduction in muscle potassium. One might assume that part of the glycogen formation may have accumulated as a result of increased glycogenesis from amino acids diverted from tissue synthesis (Spergel et al., 1967). Buell and Turner (1941) postulated two pools of potassium as an explanation for the failure of hepatic potassium to decline. One pool, "glycogen-bound," parallels liver glycogen content and would not reflect potassium change, as liver water varies in parallel with liver glycogen content. A second pool, labile, small and independent of glycogen content is responsive to body potassium. Conversely, in muscle the glycogen-bound pool is small, while the labile pool is large. Recognizing the relatively small labile potassium pool in the liver compared to the large glycogen-bound pool, any alterations of body potassium content would reflect only minor changes in liver potassium and be difficult to detect.

In 1923 Harrop and Benedict (1923) noted a fall in serum potassium upon the administration of insulin and suggested that potassium entered the liver cell along with glucose during glycogenesis. Later work demonstrated that the disappearance of potassium was in fact due to tissue potassium uptake in both the liver (Fenn, 1939; Burton and Ishida, 1967) and skeletal muscle (Hiatt et al., 1973). More recent studies, both <u>in vivo</u> and <u>in vitro</u>, have clearly shown that increasing increments of potassium within normal physiological ranges stimulate pancreatic secretions of insulin (Hiatt et al., 1972, 1973) and the converse, insulin levels too small to

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stimulate glucose uptake will induce a net increase in muscle potassium (Zierler, 1966).

Pettit and Vick (1974) postulated a two-compartment model involving potassium ion exchange and tissue uptake of potassium in extrarenal potassium homeostatis. During intravenous infusion of potassium chloride to splenectomized and nephrectomized dogs, the rate of both active and passive tissue uptake of potassium was proportional to circulating plasma potassium and suggestive of a linear transfer of potassium from the extracellular fluid (ECF) to the intracellular fluid (ICF). Earlier data indicated that both insulin and high circulating plasma potassium cause cellular hyperpolarization which leads to influx of potassium into cellular spaces. When the dogs were pancreatectomized and again infused with potassium chloride, Pettit and Vick (1974) noted movement of potassium from ICF to ECF with an accompanied rise in plasma potassium. To the authors this indicated that removal of the pancreas diminished the volume of ICF available for potassium storage and exchange, and concluded that the translocation of potassium through the insulin secretion portion of pancreas during acute potassium loading was the trigger mechanism for insulin release.

It has been clearly documented in rats (Gardner et al., 1950; Furham, 1951; Spergel et al., 1967) and humans (Sagild et al., 1961; Kaess et al., 1971; Gorden, 1973) that restricted potassium intakes affects glucose tolerance. When humans were experimentally depleted by 5-10% of total body potassium and later given either oral or intravenous glucose challenge, there occurred an impairment in the

early phase of insulin release leading to moderate glucose intolerance. The basal levels of insulin are usually normal prior to glucose challenge (Kaess et al., 1971; Gorden, 1973), but in a few cases, insulin levels had been elevated (Sagild et al., 1961). In addition, a few studies (Gorden et al., 1972) noted a delayed response of growth hormone to glucose loading, similar to the impaired growth hormone utilization associated with disease conditions such as aldosteronism, which lead to potassium depletion in humans and are usually corrected with potassium supplements (Podolsky et al., 1971). Furthermore, Sagild and Andersen (1964) demonstrated that the experimentally potassium-depleted subjects showed a normal response or sensivity to exogenous insulin or glucagon when given during the fasting state. The ability to respond to glucagon implies that hepatic function or glycogenolysis was not impaired, while normal sensitivity to insulin suggests that the reason for the glucose intolerance was decreased peripheral uptake of glucose from lack of available endogenously produced insulin.

In the rat the major sites of long term potassium depletion are in the serum and muscle, and are accompanied by either a decrease (Gardner et al., 1950; Mondon et al., 1968) or increase (Spergel et al., 1967) in liver glycogen depending on the duration of potassium deficiency.

Similarly the rat, like potassium-depleted humans, shows an impaired insulin response to glucose loading, and in addition, is also able to respond to exogenously supplied insulin. The basal levels of insulin, corticosterone and free fatty acids are normal in

the potassium-depleted rat, but basal glucose levels are usually elevated (Mondon et al., 1968).

Initially workers (Gardner, 1950; Fuhrman, 1951) associated the glucose intolerance found in potassium-depleted rats with increased adrenal cortical activity as indicated by both eosinopenia and adrenal hypertrophy in their experiments. However, more recent studies (Roseman et al., 1955; Singer and Stack-Dunne, 1955) have been unable to duplicate the adrenal hypertrophy and suggest that the eosinopenia noted earlier was related to reduced food intake and subsequent lowered amino acid absorption in the potassium-depleted rat.

Of interest is the work of Mondon et al. (1968), in which glucose tolerances were conducted in rats that were fed either (1) potassium depleted; (2) normal potassium-caloric restricted; or (3) normal potassium-normal caloric diets. He reported only rats from the potassium-depleted and normal potassium-caloric restricted diets that demonstrated complete cessation of body growth showed glucose intolerance accompanied by a suppression of insulin release. This suggests that the malnutrition associated with potassium deficiency may be the main reason for the impaired carbohydrate metabolism.

Kaess et al. (1971) studied the composition and distribution of electrolytes and insulin content of the pancreas during experimental potassium deficiency in rats. He reported a diminished content of potassium in both the intracellular and extracellular fluids of the pancreas and the body compartments, while sodium increased in the extracellular fluids of both the pancreas and skeletal muscle. The

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potassium content of the liver was normal as was the insulin content of the pancreas, but upon glucose challenge a diminished release of insulin was noted. Actually, insulin secretion is thought to be regulated by the concentration of calcium in the cytosol of the beta cells and normally the flux of calcium ions and the intracellular translocation of these ions constitute major mechanisms for insulin release. Perhaps this altered concentrations of sodium and potassium in intracellular and extracellular fluid of the pancreas during potassium depletion cause indirect modification of calcium movement in the pancreas. The resulting distribution could then be the prerequisite for impaired insulin secretion.

Body and Fluid Potassium

Potassium is distributed throughout the fat-free body and is the most abundant intracellular ion in animals. Because of the large amounts of potassium in muscle tissue compared to fat, bone, or extracellular water, whole-body potassium has been used as a quantitative index of lean body mass (Anderson, 1963). The theoretical basis for using the radioactive isotope ^{40}K to measure muscle mass was the assumption that cellular concentrations of potassium remained relatively constant in animal tissues. There is evidence, however, that potassium concentrations in muscle masses are subject to variations.

Gillett et al. (1967) compared 7 different muscles from growing beef steers and reported differences in tissue potassium concentration when expressed on a wet basis, a fat-free, moisture-free, and a protein basis. Mean tissue potassium concentration had variations

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as high as 13% in some cases. Similar observations have been noted in dairy cows (Bennink et al., 1967), in that, the potassium movement across tissue membranes tended to be greater and the concentrations more variable in nonskeletal tissues. Also, as the fat content of a tissue increased, there tended to be a decreased tissue potassium content.

To estimate the carry-over effects of diet on ^{40}K count, blood serum and muscle potassium, Johnson et al. (1972) fed three dietary levels of potassium (1.31, 1.03, and 0.29%) to Angus-Hereford steers for 2 weeks in a 3 x 3 latin square design. Although unable to show any significant carryover effects for each trait, he did report that the high potassium diet caused a significantly higher shrunken and unshrunken whole body ^{40}K count. He also noted that steers on the high potassium diet had higher blood serum and muscle potassium concentrations. Similar increases in whole body ^{40}K (Lohman et al., 1966) and blood (Lohman and Novtan, 1968) concentration of potassium were noted when steers were fed high roughage diets compared to low roughage diets. Clark et al. (1970) also observed the same trends from steers fed altered ratios of corn to corn silage compared to those fed only corn.

The literature available does not indicate that the metabolism of potassium post-absorptive or cellular potassium metabolism is any different in ruminants than in other species. Reports do suggest, however, that potassium components of blood are subject to normal changes coinciding with growth and sexual maturity. Blood serum potassium declines slightly with age in sheep (Long et al., 1965), and

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between 0.5 and 2 years of age in dairy heifers (Tumbleson et al., 1973). Furthermore, Israel et al. (1972) reported a decrease in potassium content of the red blood cell, with a concomitant increase in sodium occurring by 60 days of age in Holstein cattle. Similarly, two distinct types of red blood cells occur in Simmental cattle (Christinaz and Schatzman, 1972), one cell type having a higher potassium content and greater Na^+K^+ ATP-ase activity, while the second type a lowered potassium content and less reactive Na^+K^+ ATP-ase activity. The reported differences in blood components and shifts have not been attributed to breed or sex differences in the bovine, although breed groups have been shown to be responsible for red blood cell differences in sheep (Evans and King, 1955).

The concentration of potassium, like most minerals in milk, probably is not influenced a great deal by diet. Studies by Rook and Wood (1959) indicate healthy cows have the ability to produce milk with a constant concentration of potassium, but for any two given cows the variation in milk potassium may be as great as 50%. Significant variations between areas in the United States (Ward, 1963) and areas within California (Nickerson, 1960) have also been reported. Usually the concentration of potassium in milk is 5 to 10 times that found in blood plasma, whereas, the reverse is true for sodium. Colostrum milk has a lower content of potassium which gradually increases as milk becomes normal (Garrette and Overman, 1940), but with advancing lactation, the level of potassium normally declines (Forbes et al., 1922). Furthermore, during periods of extreme hot weather which normally cause a decrease in appetite, milk potassium

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concentration decreases (Kamal et al., 1961). Similarly, during periods of increased body osmotic pressure caused by reduced water intake, milk potassium will increase (Wheelock et al., 1965).

Sasser et al. (1966) fed cows alfalfa hay top dressed with molasses for a 1-month period. This increased potassium intake from 281 to 526 grams per day, with the level of milk potassium ranging from 1.44 and 1.64 gram per liter initially and 1.41 and 1.55 grams per liter at the end of one month. A similar trial (Sasser et al., 1966) noted no changes in milk potassium between groups of cows fed high or low roughage diets, but did observe large variations in potassium content between individual cows. While studying the consequences of low potassium intakes, Pradhan and Hemken (1968) reported an increased substitution of sodium for potassium in milk from cows fed a diet containing 0.26% potassium. Other workers (Dennis and Hemken, 1975; Dennis et al., 1976) studying restricted potassium intakes found no changes in milk potassium content in cows fed higher levels of potassium ranging from 0.45 to 0.97%. The literature reviewed suggest that under normal conditions, and even a wide range of elevated potassium intake, milk potassium content remained stable, at least for any one individual cow. However, when the cow is forced to regulate more closely the control mechanisms for electrolyte balances induced by either periods of stress, elevated temperatures, depressed appetites, lowered water intake, or marginal intakes of potassium, a reduction did occur in the amount of potassium excreted in milk and was possibly accompanied by a reduction in the total milk flow.

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Metabolism of Potassium

Potassium is not stored to any great extent in the body, thus, dietary sources are of major importance in homeostatic regulation between cellular and tissue potassium reserves. Ruminant animals, in comparison to other species, usually have an uncommonly large intake of potassium. In these species, potassium from the diet provides a large portion of the cations in rumen fluid and thus aids in maintaining a desirable medium for bacterial fermentation. Scott (1967) reported that in a range of 390-903 meq. of potassium found in daily feed intake, 35% could be accounted for by rumen potassium content. Ruminal sodium levels, by comparison, are generally 2 to 5 times greater than are attributable to dietary sources. These differences arise from the high sodium concentrations added by the animal's saliva. Reported ranges for potassium in saliva and rumen fluid were 4-70 and 24-85 meq. per liter, respectively (Bailey, 1961). Comparable values for sodium are 74-166 and 83-147 meq. per liter and under normal conditions sodium concentrations usually exceed potassium by a factor of 1.5 to 3.

Both sodium and potassium are absorbed from the rumen (Parythasarthy and Phillipson, 1953; Oyaert and Bouckaet, 1961; Warner and Stacy, 1972b) and the omasum (Oyaert and Bouckaet, 1961), with the rate of absorption greater for sodium than potassium. High rumen concentrations of potassium must be maintained for absorption to occur along either an electrical or concentration gradient across the rumen wall. The gastrointestinal fluids in sheep are consistently higher in potassium than blood plasma (Dobson and Phillipson, 1958)

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while the contents of the reticulorumen are consistently electro negative (30-40 mv.) relative to blood plasma for achieving absorption of potassium across the rumen wall. In contrast to potassium, sodium is absorbed throughout the entire gastrointestinal tract against both electrical and concentration gradients by means of active transport. While studying absorptive patterns of minerals, Brouwer (1961) reported the osmotic concentrations of sodium were lower in the abomasal area, sharply increased throughout the small intestine, gradually declined in the cecum, and reached the lowest concentration in the large intestine and fecal fluids. These differences, which serve to limit sodium lost via the feces, were attributed to increased water absorption throughout the small intestine and active absorption of sodium in the large intestine. The converse is true of potassium, where only marginal absorption occurs, causing colon potassium concentrations 7 times that found in blood plasma.

Although sodium and potassium differ in their absorption patterns, Warner and Stacy (1972a) reported an interesting situation which suggest that the fate of ingested sodium was intimately linked to that of potassium. When sheep were switched from a diet providing 200 meq. potassium and 30 meq. sodium to a diet containing 700 meq. potassium and 30 meq. sodium, the potassium content of the rumen increased while the sodium content decreased, apparently due to increased rate of sodium absorption across the rumen wall. The rate of sodium excretion in the urine increased by the same magnitude as the loss from the rumen, causing a negative sodium balance. When the potassium content of the diet was returned to its initial value,

there was an intense retention of sodium by the kidney, with signs of increased aldosterone activity and a positive sodium balance. Other observations of the synergistic absorptive patterns of potassium additions to the rumen have been noted (Stacy and Warner, 1966; Scott, 1969a; Warner and Stacy, 1972b), but in addition, reduced potassium intake has also been reported to decrease sodium absorption from the rumen wall (Dobson, 1965).

It is established (Blair-West et al., 1965; Dobson, 1965) that the ionic composition of sodium and potassium in the rumen can be regulated by the flow rate and electrolyte composition of saliva. During prolonged sodium deficiency the ratio of Na^+-K^+ in saliva decreases from 34 (170/5 meq. per liter) to as low as 0.2 (30/150 meq. per liter). This decreased ratio reflects a decrease of sodium additions to the saliva and a reciprocal increase in potassium content. Under these conditions the ruminal ratio of Na^+-K^+ also decreases, giving rise to an increased electrical potential across the rumen wall and greater potassium absorption (Sellers and Dobson, 1960; Scott, 1966). Similar consequences occur when grasses high in potassium content are fed to sheep resulting in elevated potassium and lowered sodium content in rumen fluid. In addition, a similar increase in the Na^+-K^+ ratio in rumen fluid occurs in sheep fed diets (0.1, 0.2, and 0.38%) low in potassium (Telle et al., 1964). A direct relationship exists between ruminal and salivary ratios of sodium and potassium and the electrical potential between the rumen and blood. The consequences of such change would ultimately affect sodium and potassium absorption across the rumen wall.

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During extreme variations in sodium and potassium intake, regulation of near normal body levels of these minerals is achieved through the action of the adrenal cortex hormone, aldosterone. Although the action of this hormone is primarily on the kidney, favoring reabsorption of sodium and excretion of potassium, it also plays a major role in controlling the Na^+-K^+ ratio in salivary secretions (Scott and Dobson, 1965). The stimulant for aldosterone release depends upon the blood levels of sodium and potassium. Low sodium or high potassium stimulates, whereas, high sodium inhibits aldosterone secretion. The alterations in blood levels of sodium and potassium are usually preceded by either dietary imbalances of sodium or potassium or by pathology of certain body organs controlling electrolyte balance. In addition, aldosterone-like effects occur during stress and the later stages of pregnancy (Watanabe, 1963).

It is misleading when considering urinary loss of sodium and potassium to regard the kidney solely as a waste organ, but rather, a regulatory device to conserve and maintain body levels of these minerals. Anderson and Pickering (1962) reported molar ratios (Na^+-K^+) of 0.43 (range 0.12-0.87) and 5.3 (range 1.65-20.8), respectively, for human and bovine urine. These differences in urinary composition arise primarily during the renal processes of filtration, reabsorption and secretion. Pickering (1965) later compared glomerular filtration rates (GFR) in these species and reported values of 125 and 936 ml/min., respectively, for the human and bovine. Primarily due to the higher GFR in the bovine, the filtered load of potassium (GFR x plasma potassium concentration) is 7.5 times greater

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than humans. Interestingly, only 10% of the filtered load is excreted by humans compared to 50% in the bovine. These differences are suggestive of a much larger dietary turnover for potassium in the bovine compared to the human.

There is evidence that under normal conditions a large amount of filtered potassium is capable of being reabsorbed in the proximal segments of the nephron and that most of the urinary excretion of potassium arises from secretion in the distal segments of the nephron. It is believed that the cells within the distal tubules compete with potassium and hydrogen ions in an exchange and concomitant reabsorption of sodium ions from the luminal tubule fluid (Davidson et al., 1958). With the inclusion of increasing plasma levels of potassium, induced by either infusion or incremental dietary increases, urinary potassium excretion increases (Campbell and Roberts, 1965; St. Omer and Roberts, 1967; Scott, 1969b; Cowin and Phillips, 1973). When the potassium intake of sheep ranged from 236 to 677 meq. per day, urinary potassium losses ranged from 105 to 480 meq. per day. At potassium intakes between 899 to 1,124 meq. per day, both the plasma potassium concentration and glomerular filtration rate increased leading to an increased amount of potassium filtered through the nephron. Urinary potassium excretion ranged from 585 to 778 meq. per day, suggesting that a maximum tubular reabsorptive capacity had been reached allowing a portion of the filtered potassium to augment the secretory process in eliminating potassium. During the period of high potassium intake fecal losses also reached a maximum, probably attributable to the larger amounts of potassium

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entering the large intestine. Ward (1966) estimated that urinary potassium as a percentage of total potassium excretion was 86 and 75% for nonlactating and lactating cows, respectively.

Potassium Influences on Other Minerals

High potassium intakes result in reduced apparent absorption of magnesium from the gastrointestinal tract, with accompanied increases in both fecal and urinary magnesium excretion (Fontenot et al., 1973) and a concomitant lowering of blood magnesium (Kunkel et al., 1953; Suttle and Field, 1967, 1969). Newton et al. (1972) reported a 46% depression in magnesium absorption when high levels of potassium (4.9 vs 0.6%) were fed to lambs. Similarly, a 48% increase in fecal magnesium excretion was found in sheep fed a high-protein, high-potassium (34.4 and 4.7%) diet when compared to a low-protein, low-potassium (12.8 and 1.4%) diet (Fontenot et al., 1960). A 2.5% reduction in magnesium availability occurred with the addition of 400 grams of potassium chloride to fresh cut grass diets fed dairy cows (Kemp et al., 1961). Reports also suggest that the availability of calcium may be affected by high potassium intakes (Fontenot et al., 1960; Suttle and Field, 1969; Newton et al., 1972). Urinary calcium excretion was 67% less when dietary potassium was 4.5 compared to 1.8%. There was little change in fecal calcium and increased blood calcium, suggesting increased calcium retention. This observation has been confirmed in other sheep studies (Fontenot et al., 1960; Newton et al., 1972), although Kunkel et al. (1953) reported a strong tendency toward hypocalcemia in sheep fed diets containing 5% potassium. The exact consequences of high potassium

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diets on calcium metabolism needs further clarification, but it would appear that grass tetancy or hypomagnesemia induced by high potassium diets may be further complicated by impaired calcium metabolism.

Low potassium intakes usually give rise to negative potassium balances within the animal and a subsequent reduction in circulating blood potassium (Campbell and Roberts, 1965; St. Omer and Roberts, 1967; Cowan and Phillips, 1973). The negative potassium balance is thought to be the consequence of reduced gastrointestinal absorption of potassium (Dobson, 1965) and is further complicated by an obligatory fecal potassium loss (Campbell and Roberts, 1965; St. Omer and Roberts, 1967; Cowan and Phillips, 1973). Renal handling of potassium normally parallels dietary intake and compensates for low potassium intakes by reducing urinary potassium losses. Other studies (St. Omer and Roberts, 1967; Cowan and Phillips, 1973) have reported negative potassium balances as rapid as day 4 and 5 in sheep and beef heifers, respectively, when diets contained .032 and .28% potassium. This amounted to a whole body potassium deficit in sheep ranging from 6.7 to 11.4% over a 5 day period.

Telle et al. (1964) reported a correlation of 0.72 between plasma potassium and potassium intake when comparing 5 dietary levels of potassium fed to lambs. In this study, potassium concentrations in whole blood and red blood cells paralleled potassium intakes, as did increasing hematocrit percent. In addition, these studies verified other similar observations (Campbell and Roberts, 1965) that feeding diets containing less than 0.3% potassium significantly depressed blood plasma potassium and phosphorus without apparent

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effects on plasma sodium, chloride, calcium, or magnesium. In comparison to lambs, feeding diets containing less than 0.36% potassium to beef steers (Roberts and St. Omer, 1965; St. Omer and Roberts, 1967; Devlin et al., 1969) or dairy cows (Pradhan and Hendken, 1968) caused a significant reduction in blood potassium. However, in bovine the low potassium diets promote elevation in blood phosphorus, calcium, magnesium, and chloride (St. Omer and Roberts, 1967; Devlin et al., 1969). The exact reason for these shifts are not clear and their importance unknown. The differences noted may have been due to the dietary levels of these minerals or the differences in renal responses to low potassium intakes by the bovine and ovine.

Dietary Level of Potassium and Animal Performance

The consequences of low potassium intake have not been attributed to the etiology of any one disease syndrome, but in general, are a complication of many factors which contribute to poor animal health and low productivity. Most feeds consumed by ruminants contain an abundance of potassium. Presumably for this reason very little consideration has been given to the effects of inadequate dietary potassium until the advent of all concentrate diets. The feeding of such diets may result in potassium intakes one-fourth of that consumed on high forage diets. In 1934, DuToit et al. reported that a ration containing 0.34% potassium was adequate for Friesland dairy cows with moderate milk production over two successive lactations and gestation periods. More recent evidence (Pradhan and Hemken,

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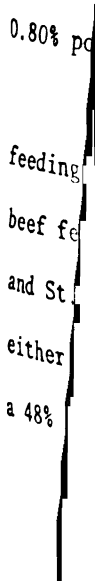

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1968; Dennis and Hemken, 1975; Dennis et al., 1976) would suggest that this concentration is inadequate for promotion of optimum feed consumption and milk production.

Dennis et al. (1976) reported a 2.4 kg increase in feed intake when cows past the peak of lactation were changed from diets containing 0.45 or 0.55 to one containing 0.66% potassium. Similar observations of improved feed intakes and milk production were noted when cows in the early part of lactation were changed from diets containing 0.46 to 0.97% potassium (Dennis and Hemken, 1975). During a change-over experiment involving 8 cows past the peak of lactation fed either low potassium (0.06 or 0.15%) or potassium-adequate diet (0.80%), symptoms of potassium deficiency were achieved in 3 to 4 weeks on the low potassium diets (Pradhan and Hemken, 1968). These included partial to complete inanition with pica characterized by hair and floor licking, while the hair coats showed a gradual loss of glossiness and finally turned dull and rough. Feed intake was reduced by 34% as was a lowered trend in milk production during the later stages of the experiment. Normal appearance, appetite and milk production was achieved when these cows were changed to the 0.80% potassium diet.

Once established that detrimental performances accompany the feeding of low potassium diets, the question of dietary adequacy of beef feedlot rations began to prompt further investigation. Roberts and St. Omer (1965) fed diets ad libitum to Hereford steers containing either 0.27, 0.51, 0.72, or 0.85% potassium for 110 days and reported a 48% reduction in feed consumption for steers fed the 0.27% diet

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compared to the 0.85% potassium diet. In a parallel study (Devlin et al., 1969) where 4 levels of potassium (0.36, 0.50, 0.67, and 0.77%) were fed ad libitum to finishing steers for 105 days, a similar reduction of 50% in feed intake occurred by day 7 for steers consuming the 0.36% diet compared to those in either the 0.67 or 0.77% potassium diet. Symptoms of potassium deficiency were manifested in steers receiving either a 0.27% (Roberts and St. Omer, 1965) or a 0.36% potassium diet (Devlin et al., 1969), which included partial to near complete inanition and pica, involving hair licking, pulling and eating of wooden fences. Hair coats had a rough appearance and body weight changes showed considerable variation among steers. Finishing steers receiving 0.5% potassium diets had intermediate growth responses, while those on diets ranging from 0.67 to 0.87% potassium performed more satisfactorily (Roberts and St. Omer, 1965; Devlin et al., 1969). In a potassium balance study (St. Omer and Roberts, 1967) Hereford heifers were estimated to need 133 meq. of potassium per 100 kg of body weight for maintenance and 278 meq. of potassium per 100 kg of body weight or 0.5% of the diet for growth. The higher potassium requirement for growth is needed to promote adequate feed consumption and body retention of potassium, whereas the maintenance diet need only support body retention. The lower potassium requirement for growing heifers compared to fattening steers might be explained by the age and weight differences when these animals were initially placed on trial. In addition, growing females have a lower whole body ^{40}K count compared to dairy bulls (Anderson et al., 1970). This

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reflects a smaller muscle mass and less capacity for body retention of potassium.

In an attempt to define the potassium requirement of growing finishing lambs, Telle et al. (1964) fed diets containing either 0.10, 0.20, 0.38, 0.42, or 0.62% potassium for 49 days. After which, diets were changed to either a 0.62 or 0.81% potassium diet and fed for 108 days. After the first 26 days, lambs on the 0.1 and 0.2% diets showed stiffness in the hind legs, which progressed to the forelegs and later to the neck and back region. The first death occurred at day 27 on the 0.1% diet, with lambs showing signs of listlessness, impaired response to sudden disturbances and convulsions preceding death. All lambs on the 0.1 and 0.2% diets and a few on the 0.38% diet were emaciated within 28 days, but lambs fed all other levels of potassium appeared normal. A graded increase in rumen papillary length was noted with increasing potassium intakes, with lambs from the 0.62% diet having papillae which were 2 times longer than those fed the 0.1% potassium diet. Kidney cortex cells from lambs fed the 0.1 and 0.2% diets were swollen and had a cloudy appearance, skeletal muscle fibers were necrotic and undergoing hyaline degeneration. When the 0.1 and 0.2% potassium fed lambs were placed on the 0.62% potassium diet they showed increased feed consumptions within 2 days and weight gains equal to that of the lambs fed the 0.42% potassium diets.

In a similar study, Campbell and Roberts (1965) reported symptoms of potassium deficiency by day 6 in lambs fed either a 0.1 or a 0.3% potassium diet. In addition to the common symptoms

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associated with potassium deficiency in lambs (Telle et al., 1964), these lambs exhibited a pica characterized by wool biting. Upon death, skeletal muscles were lower in potassium and higher in sodium when compared to lambs fed a diet containing 0.5% potassium. No lesions were noted in the heart, adrenal, or intestinal tissues, although pathological changes were present in the kidney. In a corresponding trial, Campbell and Roberts (1965) reported no dietary influences on nitrogen, energy or dry matter digestibilities when 13.7 meq. of potassium was fed to lambs, but did report a decrease in body retention of nitrogen. It is difficult to ascertain whether the lower nitrogen retention was due to derangement of protein metabolism or just lack of dietary nitrogen due to lack of appetite in the potassium depleted lambs.

Depending upon the severity and duration of potassium depletion, the following generalized signs have been noted: depressed growth, reduced feed intake, stiffness and paralysis, muscular weakness, intracellular acidosis, degeneration of vital organs and nervous disorder. The minimum dietary level of potassium necessary for normal animal health for various animal species is presented in Table 1.

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Table 1.--Minimum Dietary Levels of Potassium Required for Normal Animal Performance as Suggested by Respective Authors.[a]

Species	Minimum Level	Reference
Beef Cattle		
Finishing Steers	0.5 -0.6	Roberts and St. Omer (1965)
	0.67-0.77	Devlin et al. (1969)
Growing Heifers	0.5	St. Omer and Roberts (1967)
Sheep		
Finishing Lambs	0.6	Telle et al. (1964)
	0.3 -0.5	Campbell and Roberts (1965)
Dairy Cattle		
	0.34	DuToit et al. (1934)
	0.8	Pradhan and Hemken (1968)
	0.66	Dennis et al. (1976)
	0.69	Dennis and Hemken (1975)
Swine		
Growing Pigs	0.26	Cox et al. (1966)
Poultry		
Chicks	0.17	Ben Dor (1941)
	0.20	Gillis (1948, 1950)
	0.30	Leach et al. (1959)
Equine		
Foals	0.8	Stowe (1971)

[a]Values are percent potassium of ration dry matter.

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MATERIALS AND METHODS

Experimental Design

Studies were conducted during late gestation and early lactation on 24 Multiparous Holstein cows weighing between 604.8 and 836.4 kilograms (kg). Six cows were selected and assigned to 1 of 4 dietary levels of potassium, such that, each treatment group would be balanced for age at calving, number of previous lactations, genetic group and history of post-calving disorders. Assignment to treatments began 35 days before expected calving date, and the experimental period concluded 35 days postpartum.

Prior to placement on trial, all cows were fed individually a ration of corn silage and alfalfa hay while stanchioned with their herd contemporaries at the Michigan State University dairy barn. Water was offered ad libitum. The ingredients used to prepare the 4 treatment diets consisted of dried brewers' grain, beet pulp, shelled corn, and a vitamin-mineral supplement. Potassium chloride (Dyna-K, International Mineral and Chemical Corporation, Libertyville, Illinois) was added to adjust levels of potassium to 0.26, 0.54, 0.73, and 1.96% of the ration dry matter (Table 2). Each treatment group received 6.82 kg of assigned diet twice daily throughout the experimental period. In addition, dietary groups 0.54, 0.73, and 1.96% K^{+} also received 2.27 kg of alfalfa hay once daily. This altered the

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Table 2.--Percentage of Ingredients Used to Prepare Basal Rations.

Ingredient (%)	Dietary level potassium (Dry matter basis)			
	0.37%K	0.54%K	0.73%K	1.96%K
Brewers' grain, dried	66.0	59.3	58.8	54.3
Beet pulp	23.0	39.6	39.2	36.1
Shelled corn	9.9	--	--	--
Dicalcium phosphate	0.3	0.3	0.3	0.3
Trace Mineralized Salt	0.8	0.8	0.8	0.8
KCl	--	--	0.9	8.5
Vitamins*	+	+	+	+

*Vitamin A 341 I.U./kg, Vitamin D 170 I.U./kg, Vitamin E 0.1 I.U./kg.

level of potassium to 0.61, 0.78, and 1.83% of the ration dry matter respectively. Following parturition, additional energy demands were met by feeding 2.27 kg of shelled corn twice daily to all dietary groups. The average total dietary level of potassium fed throughout the entire experiment for the 4 groups was 0.37, 0.59, 0.74, and 1.68% K, respectively. A composite sample from each of the four basal diets, alfalfa, hay and shelled corn was taken weekly and later analyzed for mineral and nutrient components (Ohio Agriculture Research and Development Center, Wooster, Ohio). Presented in Table 3 is the average composition of the four potassium diets fed during the pre- and postpartum periods. Each value represents eight samples.

The general condition of the cows, signs of potassium deficiency, and other abnormalities were carefully observed throughout

Table

Table 3.--Average Amount of Selected Nutrients in Diets Fed During the Pre-Partum and Post-Partum Period (Dry Matter Basis).

	Dietary Potassium							
	0.37%		0.59%		0.74%		1.68%	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Crude Protein, %	22.69	19.65	19.44	17.45	20.36	17.90	18.99	17.09
Crude Fiber, %*	14.68	11.70	18.06	14.74	17.90	14.57	16.82	13.74
H_2O, %	8.74	10.00	8.31	9.58	8.65	9.84	7.99	9.28
TDN, %*	62.88	69.62	61.42	67.68	61.63	67.86	56.68	63.91
P, %	0.55	0.53	0.43	0.44	0.46	0.46	0.35	0.37
Ca., %	0.38	0.37	0.65	0.58	0.73	0.64	0.59	0.54
Mg., %	0.19	0.17	0.30	0.26	0.34	0.29	0.30	0.26
Na., %	0.15	0.15	0.14	0.15	0.11	0.12	0.13	0.13
S., %	0.23	0.18	0.24	0.19	0.25	0.19	0.24	0.19

*Estimated figure.

the experimental period. Daily feed intakes and milk production, as well as weekly body weights were recorded. During the postpartum period, a weekly one-day composite milk sample was collected from each cow by combining an aliquot from the morning (0400 hr) and afternoon (1500 hr) milking. These samples were collected and stored at -18°C in 6 ounce whirl-pak containers (Naso, Fort Arkinso, Wisconsin) until later analysis for sodium and potassium concentrations. Blood samples were collected biweekly from all cows throughout the experimental period, but daily samples were taken when parturition approached. At parturition a sample was collected and sampling continued at 4-hour intervals for the following 24 hours. A daily sample was taken for the next five days and thereafter biweekly. Blood samples were obtained by venipuncture of the tail vein using a 20 gauge 0.6 centimeter needle fitted to a 20 ml vacutainer tube (Beckman, Dickinson and Company, Ruthford, New Jersey). Caution was taken during blood collection to avoid hemolysis and immediately following was chilled in ice water for 2 hours. The cooled sample was centrifuged at 6000 xg for 10 minutes, and serum pipetted into plastic storage vials (Fisher Scientific Company, Pittsburgh, Pennsylvania) then stored at -18°C until later analysis for various mineral and blood metabolites.

Methodology

Blood Sodium and Potassium Analysis

Serum concentrations of sodium and potassium were determined by flame emission spectroscopy using a Model 453 atomic absorption/

emission spectrophotometer (Instrumentation Laboratory, Inc., Lexington, Massachusetts). Procedures used were those outlined in the operations manual. Glassware used in mineral analysis was thoroughly acid washed and rinsed in deionized distilled water. Frozen serum samples were thawed at room temperature and thoroughly mixed before duplicates of 12.5 and 50 microliters (ul) were measured with a pipette and diluted 1:200 and 1:50, respectively, for sodium and potassium, with deionized distilled water and mixed with a vortex mixer for 5 seconds.

From stock solutions (100,000 mg/liter) of sodium and potassium (Table 4), working solutions for standard curves of 2000, 3000, and 4000 mg/liter sodium, and 100, 200, and 300 mg/liter potassium were prepared (Table 5). Working solutions were processed as indicated for serum samples. Following instrumentational procedures for single channel emission mode, wave lengths of 767.6 and 580.8 nanometers (nm) were selected for sodium and potassium, respectively. Workable readouts were obtained by fine adjustments of slit width and photomultiplier voltage. A small aliquot of unknown sample was then aspirated through the flame and the intensity later compared to standards of known concentrations.

Blood Magnesium Analysis

Serum concentrations of magnesium were determined by atomic absorption spectroscopy (Instrumentation Laboratory, Inc., Lexington, Massachusetts). Following sample preparation, paired aliquots of 50 ul were diluted 1:100 with 10,000 ppm strontium (J. T. Baker Chemical Company, Phillipsburg, New Jersey) and mixed with a vortex

Table 4.--Standard Stock Solutions of Sodium, Potassium, and Magnesium.

Stock Solution (10,000 mg./liter)	Chemical[a] (gm)	Deionized Distilled Water (ml)
Sodium	NaCl[a] 25.42	974.58
Potassium	KCl 19.07	980.93
Magnesium	Mg (2.0 dissolved in HCl)	998.00

[a]J. K. Baker Chemical Company, Phillipsburg, New Jersey.

Table 5.--Proportions of Stock Solutions and Water Used to Make Working Solutions Used in Making Standard Sodium, Potassium, and Magnesium Curves.

	Working Solution (mg./liter)	Stock Solution (ml)	Deionized Distilled Water (ml)
Std. 1	2000 Na. 100 K 20 Mg.	200 100 20	680
Std. 2	3000 Na. 200 K 40 Mg.	300 200 40	460
Std. 3	4000 Na. 300 K 60 Mg.	400 300 60	240

mixer for 5 seconds. From the stock solution of magnesium working solutions for standard curves of 20, 40, and 60 mg/liter were prepared and processed as indicated for serum samples. Following outlined procedural set up for atomic absorption spectroscopy, selection of hollow cathod lamp and positioning of slit width, a peak with wave length of 250 nm was achieved. A small aliquot of sample was aspirated through the flame and the absorbency later compared to standards of known concentrations.

Blood Calcium, Phosphorus, Urea Nitrogen, Glucose, Cholesterol, Creatinine, Glutamic-Oxalacetic Transaminase, Glutamic-Pyruvic Transaminase, Alkaline Phosphatase, and Creatine Phosphokinase Analysis

The above mentioned serum components were measured on a Hycel Mark X discretionary multi-channel blood analyzer (Hycel, Inc., Houston, Texas). The principle test procedures used in this automated clinical chemistry machine is referred to in Table 6. Some modifications of tests have occurred in adaptation to automation and the reader may refer to the Hycel Chemistry Methods, Hycel, Inc., for further details.

Milk Sodium and Potassium Analysis

Concentrations of milk sodium and potassium content were determined by flame emission spectroscopy as outlined in methodology for blood sodium and potassium analysis. The sample was thawed and mixed, then 15 ml of whole milk was centrifuged at 6000 xg for 15 minutes. From the supernatant portion, 1 ml was pipetted off and

Table 6.—Serum

Table 6.--Serum Component and Test Principle Used in Hycel Mark X Discretionary Multi-Channel Blood Analyzer.[a]

Component	Principle	Reference
Total Calcium (mg/dl)	Ca^{++}-albumin + acid → Ca^{++}, complexed by cresolphthalein complexon → purple color, colorimetric technique	Kessler and Wolfman (1964), Gitelman (1967)
Inorganic Phosphorus (mg/dl)	Inorganic P + ammonium molybdate + acid → ammonium phosphomolybdate, + ferrous salt → blue color, colorimetric technique	Taussky and Schorr (1953)
Creatinine (mg/dl)	Serum proteins + heated water → Clo^{+} + creatinine, + picrate ion, alkaline solution → red complex, creatinine picrate, colorimetric technique	London, Freiberger and Marymont (1967)
Alkaline Phosphatase (U/L)[b] (SAP)	Hydrolysis of thymolphthalein monophosphate by serum alkaline phosphatase at pH 9.85, to thymolphthalein + phosphate ion. Stop Rx, thymolphthalein + alkali → blue color, colorimetric technique	Coleman (1966)

Table 6.--Continued.

Component	Principle	Reference
Glutamic-Pyruvic Transaminase (U/L) (SGPT)	Incubate + sera + α-Ketoglutarate + L-alanine in buffer pH 7.4, add 2,4-dinitrophenylhydrzine → 2,4-dinitrophenylhydrazone + brown color, + NaOH to dissolve 2,4-dinitrophenylhydrazone, colorimetric technique	Reitman and Frankel (1957)
Glutamic-Oxalacetic Transaminase (U/L) (SGOT)	α-Ketoglutarate + L-aspartate → L-glutamate + Oxalacetate, Sodium oxalacetate couples with diazoniun salt → red color, colorimetric technique	Babson et al. (1962)
Creatine Phosphokinase (U/L) (CPK)	Creatine + ATP $\underset{\text{pH 7.4}}{\overset{\text{pH 8.9}}{\rightleftharpoons}}$ creatine phosphate + ADP Creatine phosphate is hydrolyzed → inorganic P, measure with Fiske-Subbarow Rx, blue-green color, colorimetric technique	Okinaka et al. (1961)
Total Cholesterol (mg/dl)	Rx acetic anhydride with double bond between C5 and C6 on ring B of sterol → green color, colorimetric technique	Kaser (1953)

Table 6.--Continued.

Component	Principle	Reference
Blood Urea Nitrogen (mg/dl) (BUN)	Rx Urea with diacetyl monoxine in acid solution in presence of thiosemicarbazide and ferric ion, oxidation, pink color, colorimetric technique	Crocker (1967)
Glucose (mg/dl)	O-toluidine + Hexose $\rightleftharpoons$ glucosylamine + schiff base, blue-green complex, colorimetric technique	Dubowski (1962), Hyvarinen and Nikkila (1962)

[a]Hycel, Inc., Houston, Texas.

[b]μ is an international unit, where 1 μ is the amount of enzyme which will catalyse the reaction of 1 μ mole of substrate per minute. Any further detail on test principle may be obtained in Hycel Chemistry Methods, Hycel, Inc., Houston, Texas.

deproteinized in duplicate with 12.5% TCA (trichloracetic acid, J. T. Baker Chemical Company, Phillipsburg, New Jersey), diluting 1:5. The deproteinized sample was mixed for 10 seconds on a vortex mixed and allowed to stand at room temperature for 10 minutes before centrifuging at 6000 xg for 15 minutes. A 83.3 and 50 ul aliquot from each sample was transferred and diluted 1:3 and 1:50 for sodium and potassium, respectively, with deionized distilled water and vortexed for 10 seconds. The same standards and dilution rates as in procedure for blood sodium and potassium analysis were used, but corrected for dilution differences when determining concentrations of sodium and potassium in milk.

Statistical Analysis

Statistical analyses of the data were by analysis of variance of a split plot repeat measure design, Scheffe's least significant difference and regression analysis (Steel and Torrie, 1960).

RESULTS

Performance

Level of dietary potassium exerted a very marked effect upon appetite, body weight loss and milk yield. Cows fed the 0.37% diet consumed an average of 9.9 kg grain daily over the entire experimental period. This is about 14% less ($P<.05$) than that of cows fed the three higher K diets (Table 7). Similar trends in grain consumption were observed for all treatment diets during the prepartum period. However, beginning the second week postpartum cows receiving the 0.37% diet experienced a marked reduction of appetite leading to near complete inanition in some individual cows. Total feed consumption (including hay) was not analyzed statistically because the 0.37% diet which received no hay, while the cows in the 0.59, 0.74 and 1.68% diets were offered hay. When grain intake from all diets was regressed against dietary level of K a significant ($P<.05$) curvilinear response was noted during both the prepartum and postpartum periods (Table 8). These formulations indicate that maximum grain consumption occurs at a dietary level of 0.9 and 1.1% K during the prepartum and postpartum periods, respectively (Table 9).

Cows receiving the 0.37% diet experienced an average weekly body weight loss of 17.5 kg, which was 84% greater than the average of cows fed other potassium diets (Table 10). The greatest weight

Table 7.--Average Daily Grain Intake (kg) for Cows Fed Four Dietary Levels of Potassium During Late Gestation and Early Lactation.

Weeks Relative Parturition	Dietary Potassium (Dry Matter Basis)			
	0.37%	0.59%	0.74%	1.68%
-4	8.2	11.1	9.4	8.5
-3	8.9	10.2	9.5	8.9
-2	9.9	9.6	9.3	9.0
-1	8.3	8.5	7.1	8.3
1	11.3	13.1	12.1	11.4
2	10.2	12.7	13.7	13.0
3	9.4	13.8	14.1	13.8
4	10.0	14.3	14.3	13.4
5	13.0	14.3	12.8	13.7
Overall means ± SE	9.9 ± 0.2[a]	11.9 ± 0.2[b]	11.4 ± 0.2[b]	11.1 ± 0.2[b]

[a,b]Means in rows with unlike superscripts are different at $P \leq .05$.

Table 8.--Significant[a] Linear or Curvilinear Regression Effects of Grain Intake, Milk Weight, Milk Sodium and Potassium Content (Y) and Dietary Level of Potassium (X).

Component	Prepartum (days)	Postpartum (days)
	28 through 1	1 through 35
grain intake	$8.7 + 1.2(X) - 0.7(X^2)$	$5.9 + 1.6(X) - 7(X^2)$
milk weight		$4.5 + 59.4(X) - 25.2(X^2)$
milk sodium		$262.4 - 15.0(X)$
milk potassium		$71.2 + 5.1(X)$

[a]Regression significant at $P \leq .05$.

Table 9.--Optimum Dietary Level of Potassium as Calculated from Significant ($P \leq .05$) Curvilinear Regression Equations Generated During Selected Time Periods.

Component[a]	Prepartum (days)		Parturition (hours)	Postpartum (days)	
	28 through 14	5 through 1	0 through 20	1 through 5	14 through 25
Serum K	1.2 mx	1.3 mx	1.4 mx	1.2 mx	1.3 mx
Serum Na	0.9 mx	0.8 mx	0.8 mx	0.9 mx	0.9 mx
Serum P		1.2 mn[b]	1.2 mn	1.2 mn	1.2 mn
Serum cholesterol			1.1 mn		1.1 mx
Grain intake	-------0.9 mx--------			-------1.1 mx--------	
Milk weight				-------1.2 mx--------	

[a]Value denotes dietary potassium level to achieve maximum component response.

[b]Value denotes dietary potassium level to achieve minimum component response.

Table 10.--Average Weekly Body Weight (kg) Change of Cows Fed Four Dietary Levels of Potassium During Late Gestation and Early Lactation.

Weeks Relative Parturition	Dietary Potassium (Dry Matter Basis)			
	0.37%	0.59%	0.74%	1.68%
-3	6.2	11.2	10.5	7.6
-2	5.1	8.5	10.5	-3.9
-1	20.8	17.5	21.2	9.0
1	-112.4^{a}	-72.6^{b}	-96.7^{b}	-59.4^{b}
2	-17.9	-22.5	-7.8	-5.1
3	-40.1^{a}	-21.1^{ac}	3.6^{b}	-14.8^{bc}
4	-7.4	4.1	-12.6	-0.4
5	2.3	4.5	2.6	-8.5
Overall means ± SE	-17.5 ± 6.5^{a}	-9.9 ± 4.6^{b}	-9.3 ± 5.9^{b}	-9.4 ± 4.3^{b}

^{a,b,c}Means in rows with unlike superscripts are different at $P \leq .05$.

loss occurred for cows receiving the 0.37% diet during the first and third weeks postpartum, causing this diet group to accumulate a 72% greater postpartum weight deficit than other potassium diets.

Cows fed the 0.37% diet produced 10.5 kg ($P<.05$) less daily milk than the average of cows receiving the higher K diets (Table 11) during the experimental period. Regressing milk yield against dietary level of K revealed a significant ($P<.05$) curvilinear response during the postpartum period (Table 8). This indicates that maximum milk yield (Table 9) occurred when a 1.2% K diet is fed.

Concentrations of sodium and potassium in milk are presented in Table 12. Dietary potassium exerted a significant ($P<.05$) linear response in concentrations of both milk sodium and potassium during the postpartum period (Table 8). An important reciprocal relationship existed in which sodium replaced potassium during restricted potassium intakes, while potassium replaced sodium during elevated potassium intakes. Concentration of milk potassium increased with increasing dietary K, causing milk from cows receiving the 1.68% diet to have a 10% higher milk K concentration than cows receiving the 0.37% diet.

Health

Signs commonly associated with potassium deficiency (Telle et al., 1964; Campbell and Roberts, 1965; Pradhan and Hemken, 1968; Delvin et al., 1969) were observed in cows receiving the 0.37% diet. Following an average of eight days on the postpartum diet containing 0.26% K two cows showed a marked depression in appetite and appeared emaciated. By day ten these individuals demonstrated incoordination and wobbling of hind quarters when forced to move, and had developed

Table 11.--Average Daily Milk (kg) Yield for Cows Fed Four Dietary Levels of Potassium During Late Gestation and Early Lactation.

Weeks Postpartum	Dietary Potassium (Dry Matter Basis)			
	0.37%	0.59%	0.74%	1.68%
2	25.1	32.4	33.0	33.4
3	23.2	32.9	34.3	34.2
4	21.9	34.8	36.5	35.7
5	24.5^{a}	33.3^{b}	34.1^{b}	35.7^{b}
Overall Mean ± SE	23.7 ± 0.9^{a}	33.3 ± 0.9^{b}	34.5 ± 1.0^{b}	34.8 ± 0.9^{b}

^{a,b}Means in rows with unlike superscripts are different at $P \leq .05$.

clinical ketosis based on concentration of urinary ketones. Because those cows were unable to continue satisfactorily and for fear of probable death, they were changed to a recovery diet of corn silage and alfalfa hay. After five days on the recovery diet both cows had regained normal health and mobility. Two additional cows were added to the 0.37% diet, and the data from these problem cows were not included in the data analysis. No apparent health effects were noted for cows receiving the 0.59, 0.74 and 1.68% K diets or the other cows on the 0.37% diet during the prepartum period.

Following parturition all cows receiving the 0.37% diet showed signs of emaciation, gradual dulling of hair coat, variable feed intakes and were generally rough in appearance. Cows receiving

the 0.37% diet had normal calves and calvings, and were not complicated postpartum with retained placentas, metritis, mastitis or milk fever. After an average of 17 days postcalving three of six cows receiving the 0.37% diet demonstrated complete inanition, severe emaciation, listlessness and became ketotic. Upon treatment these cows did not respond to normal therapy of 50% dextrose (IV) and oral sirlene (propyleneglycol) and were subsequently changed to the recovery diet. Following seven days on the recovery diet these cows had regained normal appetite and were more alert.

On cow each in the 0.59 and 1.68% K diets developed an uncompleted spontaneous clinical case of ketosis and responded to therapy of 50% dextrose and oral sirlene. Also, one cow in the 0.74% diet developed a clinical case of metritis and mastitis, and a secondary case of ketosis. This cow also responded to therapy.

Regression of Serum Metabolites During Periparturient Period Upon Dietary Potassium

The biological significance of statistical differences is difficult to assess because most values were within normal ranges quoted by others (Table 13). Most treatment diets provided nutrients equal to or greater than National Research Council (1971) recommendations, an exception being the 0.37% diet, which contained a lower calcium and fiber content (Table 2). Serum samples were analyzed for metabolites and data were pooled into periods for presentation of means, and for regression analysis upon dietary potassium. Five comparisons were among diet treatments for each metabolite; prepartum (28 through 14 days, 5 through 1 days), parturition

Table 12.--Average Sodium and Potassium Levels in Milk Obtained Once Weekly From Cows Fed Four Dietary Levels of Potassium During Late Gestation and Early Lactation.

Weeks Postpartum	Dietary Potassium (Dry Matter Basis)							
	0.37%		0.59%		0.74%		1.68%	
	Na mg/100 ml	K mg/100 ml	Na mg/100 ml	K mg/100 ml	Na mg/100 ml	K mg/100 ml	Na mg/100 ml	K mg/100 ml
1	47.7	97.4	50.9	92.2	48.3	103.9	45.8	98.7
2	44.4	94.0	49.1	91.0	41.6	96.9	41.4	98.1
3	43.6	92.1	46.9	89.8	46.3	106.9	40.4	98.5
4	39.5	78.9	45.2	87.0	39.9	91.9	40.8	100.8
5	42.9	87.3	47.6	90.2	43.3	96.0	40.8	97.9
Overall mean ± SE	43.6 ± 1.3	89.9 ± 2.6	47.9 ± 0.8	90.0 ± 1.2	43.9 ± 1.5	99.2 ± 3.4	41.8 ± 1.1	98.8 ± 1.5

(0 through 20 hours) and postpartum (1 through 5 days, 14 through 35 days). Treatment means (Table 14) and regression data (Table 15) are more relevant for these serum components than overall mean differences. During most of the period comparisons level of potassium exerted a significant ($P<.05$) linear or curvilinear response upon the serum concentrations of the 13 metabolites measured.

Dietary level of potassium exerted a significant ($P<.005$) curvilinear response on serum concentrations of sodium and potassium during the prepartum, parturition and postpartum periods (Table 15). Cows receiving the 0.37% K diet experienced a marked and lasting depression in serum potassium, averaging 13.9 mg/100 ml throughout experimental periods. Similar concentrations of 16.4, 15.6 and 15.6 mg/100 ml were observed for cows receiving the 0.59, 0.74 and 1.68% K diets, respectively (Table 14). Cows fed the 1.68% K diet experienced a marked reduction in serum sodium, which averaged 271.4 mg/100 ml compared to 301.7, 305.0 and 319.5 mg/100 ml for the 0.37, 0.59 and 1.68% K diet groups. Calculations from regression equations during the prepartum and postpartum periods indicate a dietary level of 0.9 and 1.3% K (Table 12) are necessary for maximum serum concentrations of sodium and potassium, respectively.

Beginning five days prepartum, during parturition and throughout the postpartum period dietary level of potassium exerted a significant ($P<.005$) curvilinear response in serum phosphorus (Table 15). Cows receiving the 0.37% diet experienced a usually marked and persistent elevation in serum phosphorus. The 0.37% K diet group averaged 5.4 mg/100 ml serum phosphorus, while diet groups 0.59, 0.74

Table 13.--Summary of Means or Ranges of Blood Components from Various Sources.

Blood Parameter	Pierce and Laird (1972)	Teer (1974)	Payne et al. (1970)	Modwax (1969)
Potassium (mg/100 ml)			18.5	18.7
Sodium (mg/100 ml)			320.0	327.0
Calcium		9.4-12.2	9.2	10.8
Phosphorus (mg/100 ml)	6.6	4.0- 7.0	5.4	6.0
Magnesium (mg/100 ml)			2.5	2.8
Creatinine (mg/100 ml)		1.0- 2.0		
SAP (μ/L)	25.4	<50.0		
SGPT (μ/L)				
Cholesterol (mg/100 ml)	125.0	50-230		
CPK (μ/L)	35.5			
SGOT (μ/L)	172.0			
Urea Nitrogen (mg/100 ml)	18.0	6-27	14.9	
Glucose (mg/100 ml)	54.0	36-60		

Table 14.--Effects of Potassium Treatments Upon Blood Serum Metabolites During Periparturient Periods.

Serum Metabolite	Diet Group	Prepartum (days)		Parturition (hours)	Postpartum (days)		Overall Means
		28 through 14	5 through 1	0 through 20	1 through 5	14 through 35	
Potassium mg/100 ml	D1	14.3[a]	13.5[a]	14.6[a]	13.7[a]	13.7[a]	13.9
	D2	16.8	16.5	16.5	16.6	15.9	16.4
	D3	16.2	15.0	15.4	15.8	16.0	15.6
	D4	16.9	16.3	16.7	16.8	16.7	15.6
Sodium mg/100 ml	D1	295.4[a]	304.2[a]	310.2[a]	301.6[a]	290.6[a]	301.7
	D2	291.9	301.8	311.1	314.8	297.6	305.0
	D3	322.4	319.3	324.8	321.2	307.4	319.5
	D4	266.8	262.3	278.0	270.9	276.7	271.4
Calcium mg/100 ml	D1	7.0	7.3	7.4	7.5	7.2	7.3
	D2	7.5	7.5	7.1	7.6	7.5	7.4
	D3	8.7	8.1	6.8	7.1	7.6	7.6
	D4	8.1	8.2	7.4	7.6	7.5	7.7
Phosphorus mg/100 ml	D1	5.5	5.5[a]	5.4[a]	5.3[a]	5.5	5.4
	D2	5.4	4.9	3.4	4.4	5.5	4.6
	D3	5.3	5.0	3.8	3.8	4.6	4.4
	D4	5.3	4.7	3.5	4.2	4.9	4.4
Magnesium mg/100 ml	D1	1.5	1.5[b]	1.5	1.5	1.6	1.5
	D2	2.3	2.3	2.4	2.2	2.4	2.3
	D3	1.4	1.2	1.4	1.3	1.4	1.3
	D4	1.6	1.5	1.7	1.6	1.9	1.6

Table 14.--Continued.

Serum Metabolite	Diet Group	Prepartum (days)		Parturition (hours)	Postpartum (days)		Overall Means
		28 through 14	5 through 1	0 through 20	1 through 5	14 through 35	
Creatinine	D1	1.1	1.3	1.6[b]	1.4	1.0	1.3
ng/100 ml	D2	1.0	1.4	1.5	1.1	0.8	1.2
	D3	1.2	1.5	1.5	1.4	1.0	1.3
	D4	1.2	1.2	1.3	1.2	1.0	1.2
SAP[c]	D1	5.9[b]	8.0[b]	11.1[b]	8.8[b]	6.0[b]	8.4
(μ/L)	D2	8.0	9.5	11.2	9.9	7.0	9.4
	D3	7.8	10.5	12.1	9.9	6.2	9.8
	D4	8.8	11.9	15.8	11.4	7.5	11.6
SGPT[d]	D1	28.4	27.3	33.1[b]	27.4[b]	26.2	28.8
(μ/L)	D2	33.5	30.2	32.4	30.4	30.2	31.3
	D3	32.8	28.2	30.3	27.0	26.3	28.8
	D4	29.4	27.9	28.6	25.7	27.3	27.7
Cholesterol	D1	103.4	99.1	94.8[a]	88.2	119.5[a]	99.7
mg/100 ml	D2	100.9	86.9	76.1	84.4	169.3	99.7
	D3	114.9	90.7	77.6	79.3	154.8	99.1
	D4	91.7	89.3	83.0	81.6	155.6	97.8
CPK[e]	D1	206.8[b]	211.3[b]	206.3[b]	194.2[b]	197.4[b]	203.3
(μ/L)	D2	224.7	214.1	173.1	189.8	270.3	209.3
	D3	215.5	197.6	156.0	157.8	190.5	179.2
	D4	170.2	165.2	128.2	148.3	173.2	153.9

Table 14.--Continued.

Serum Metabolite	Diet Group	Prepartum (days)		Parturition (hours)	Postpartum (days)		Overall Means
		28 through 14	5 through 1	0 through 20	1 through 5	14 through 35	
SGOT[f] (μ/L)	D1	96.5	96.5	119.6[b]	111.0	84.8	99.0
	D2	88.9	88.9	108.9	102.0	72.2	92.3
	D3	109.6	109.6	115.4	110.7	67.8	98.6
	D4	77.3	77.3	91.4	92.4	81.5	82.9
Urea Nitrogen mg/100 ml	D1	16.0[b]	16.6[b]	17.8[b]	12.0[b]	10.6[b]	14.9
	D2	17.7	16.4	14.4	11.7	12.7	14.2
	D3	17.5	14.9	15.8	12.2	12.3	14.5
	D4	14.5	13.2	12.6	9.2	9.4	11.7
Glucose (mg/100 ml	D1	62.1	60.8	72.1	57.4	51.0[b]	61.5
	D2	59.8	64.4	75.6	62.1	59.8	65.4
	D3	64.3	64.8	69.6	55.9	48.3	61.1
	D4	62.1	63.4	69.3	53.5	46.4	59.7
Obs/Mean		18	30	36	30	24	138

[a]Significant curvilinear relationship to dietary potassium ($P \leq 0.05$).

[b]Significant linear relationship to dietary potassium ($P \leq 0.05$). D1 = 0.37%K; D2 = 0.59%K; D3 = 0.74%K; D4 = 1.68%K.

[c]SAP = serum alkaline phosphatase. [d]SGPT = serum glutamic pyruvic transaminase.

[e]CPK = creatine phosphokinase. [f]SGOT = serum glutamic oxalacetic transaminase.

Table 15.--Significant Linear or Curvilinear Regression Effects of Serum Metabolite (Y) and Dietary Level of Potassium (X).

Metabolite	Prepartum (days)		Parturition (hours)	Postpartum (days)	
	28 through 14	5 through 1	0 through 20	1 through 5	14 through 35
Potassium	$11.0 + 11.2(X) - 4.6(X^2)^a$	$10.9 + 9.6(X) - 3.8(X^2)^a$	$13.4 + 4.8(X) - 1.7(X^2)^a$	$10.1 + 12.2(X) - 4.9(X^2)^a$	$10.0 + 12.2(X) - 4.8(X^2)^a$
Sodium	$242.9 + 157.5(X) - 85.1(X^2)^a$	$269.2 + 109.9(X) - 67.8(X^2)^a$	$277.5 + 103.7(X) - 61.5(X^2)^a$	$257.6 + 146.7(X) - 82.5(X^2)^a$	$256.8 + 108.1(X) - 57.2(X^2)^a$
Magnesium		$1.8 - 0.2(X)^b$			
Calcium	$5.2 + 6.4(X) - 2.8(X^2)^c$				
Phosphorus		$7.4 - 5.0(X) + 2.1(X^2)^a$	$8.3 - 9.7(X) + 4.1(X^2)^a$	$7.8 - 7.8(X) + 3.3(X^2)^a$	$7.1 - 4.5(X) + 1.9(X^2)^c$
Creatinine		$1.5 - 0.1(X)^c$	$1.6 - 0.2(X)^b$		
SAP[d]	$6.2 + 1.7(X)^b$	$7.7 + 2.7(X)^a$	$9.3 + 3.8(X)^a$	$8.5 + 1.8(X)^b$	$5.8 + 1.0(X)^b$
SGPT[e]			$34.0 - 3.3(X)^a$	$29.5 - 2.2(X)^a$	
Cholesterol			$126.1 - 104.5(X) + 46.9(X^2)^a$		$55.3 + 221.4(X) - 96.4(X^2)^b$
CPK[f]	$235.1 - 36.4(X)^b$	$229.3 - 38.0(X)^a$	$209.6 - 51.6(X)^a$	$200.9 - 33.5(X)^a$	$245.7 - 42.3(X)^b$
SGOT[g]		$106.3 - 15.6(X)^c$	$126.2 - 20.5(X)^b$	$115.3 - 13.3(X)^c$	
Urea Nitrogen	$18.0 - 1.8(X)^b$	$17.5 - 2.6(X)^a$	$18.0 - 3.3(X)^a$	$13.3 - 2.3(X)^a$	$12.7 - 1.7(X)^b$
Glucose				$60.9 - 4.3(X)^c$	$56.3 - 5.9(X)^b$

[a]Regression significant at $P \leq .005$.

[b]Regression significant at $P \leq .05$.

[c]Regression significant at $P \leq .10$.

[d]SAP = serum alkaline phosphatase.

[e]SGPT = serum glutamic pyruvic transaminase.

[f]CPK = creatine phosphokinase.

[g]SGOT = serum glutamic oxalacetic transaminase.

and 1.68% K averaged 4.6, 4.4 and 4.4 mg/ml phosphorus, respectively (Table 14). Calculations from regression equations suggest that a diet containing at least 1.2% K is necessary for a more normal concentration of serum phosphorus during both the prepartum and postpartum periods (Table 12).

During the day of calving and again beginning two weeks postpartum, dietary level of potassium exerted a significant ($P<.05$) curvilinear response in serum cholesterol (Table 15). Cows fed the 0.37% diet maintained the highest cholesterol concentrations during calving, and the lowest levels beginning two weeks postpartum. Similar concentrations and trends in cholesterol were observed for cows on the higher K diets (Table 14). Calculations from regression equations suggest that a diet containing at least 1.1% K is necessary to achieve a reduced or more normal cholesterol at the time of calving, while a 1.1% K diet is needed for promoting elevated cholesterol levels during early lactation (Table 12).

Beginning four weeks prepartum and continuing through five weeks postpartum concentration of dietary potassium exerted a significant ($P<.05$) linear response (Table 15) on the serum components alkaline phosphatase (SAP), creatine phosphokinase (CPK) and urea nitrogen (BUN). Serum concentrations of SAP increased, while CPK levels decreased with increasing dietary potassium. BUN concentrations were the lowest in the 1.68% K diet group and averaged 11.7 mg/100 ml throughout experimental periods. Similar BUN concentrations of 14.9, 14.2 and 14.5 mg/100 ml were observed for the 0.37, 0.59 and 0.74% K diets (Table 14).

During the day of calving dietary potassium exerted a significant ($P<.05$) linear response (Table 15) toward the serum components creatinine, glutamic pyruvic transaminase (SGPT) and glutamic oxalacetic transaminase (SGOT). Cows receiving the 0.37% diet maintained the highest level of creatinine, SGPT and SGOT during the day of calving (Table 14).

General Trends for Serum Components

Serum constituents had similar responses among treatments. Data were pooled and overall means were plotted against sample days (Figure 1 to 8) to illustrate general responses during late gestation, parturition and early lactation.

Potassium (Figure 1) decreased five days preceding parturition, peaked the day of parturition and declined during the first week of lactation. Sodium (Figure 1) increased previous to parturition, peaked the day of parturition, and declined parallel to that of potassium during the first week of lactation. Both sodium and potassium gradually increased beginning the second week of lactation. Calcium (Figure 2) remained stable prepartum, declined to a low at parturition, increased to prepartum levels by the first week of lactation and remained constant thereafter. Phosphorus (Figure 2) fluctuated prepartum, declined rapidly beginning three days prior to calving, reaching a low at parturition and increased thereafter. Magnesium (Figure 2) peaked at parturition, declined the first week postpartum and remained constant thereafter.

Glucose (Figure 3) declined prepartum, gradually increased beginning three days prior to calving, had a two-fold spike at the

Fig. 1.--Mean blood serum sodium and potassium values and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.

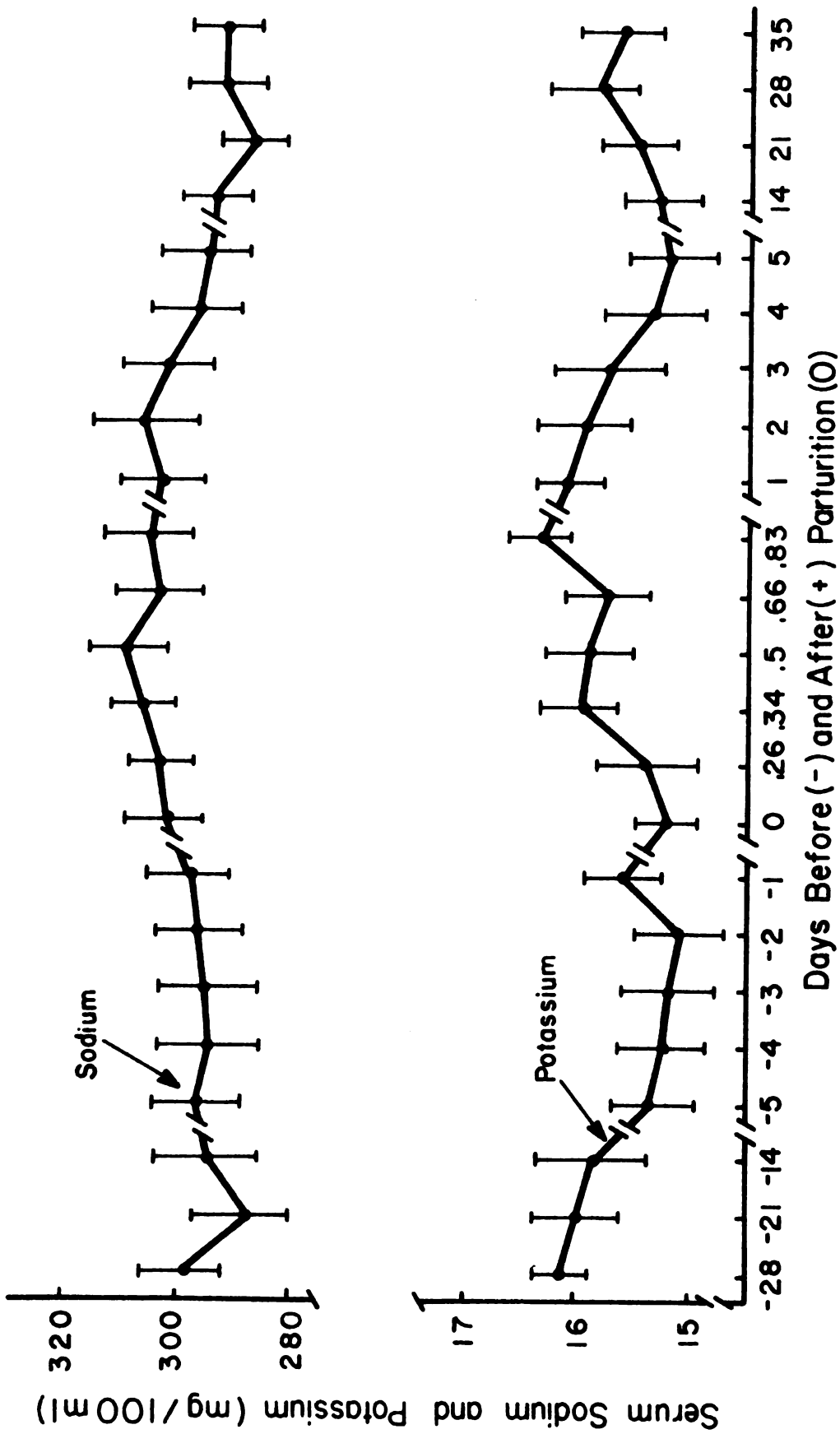

Serum Sodium and Potassium (mg/100 ml)
320
300
280
Sodium
17
16
15
Potassium
-28 -21 -14 -5 -4 -3 -2 -1 0 .26 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before(-) and After(+) Parturition(0)

Fig. 2.--Mean blood serum calcium, phosphorus and magnesium values and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.

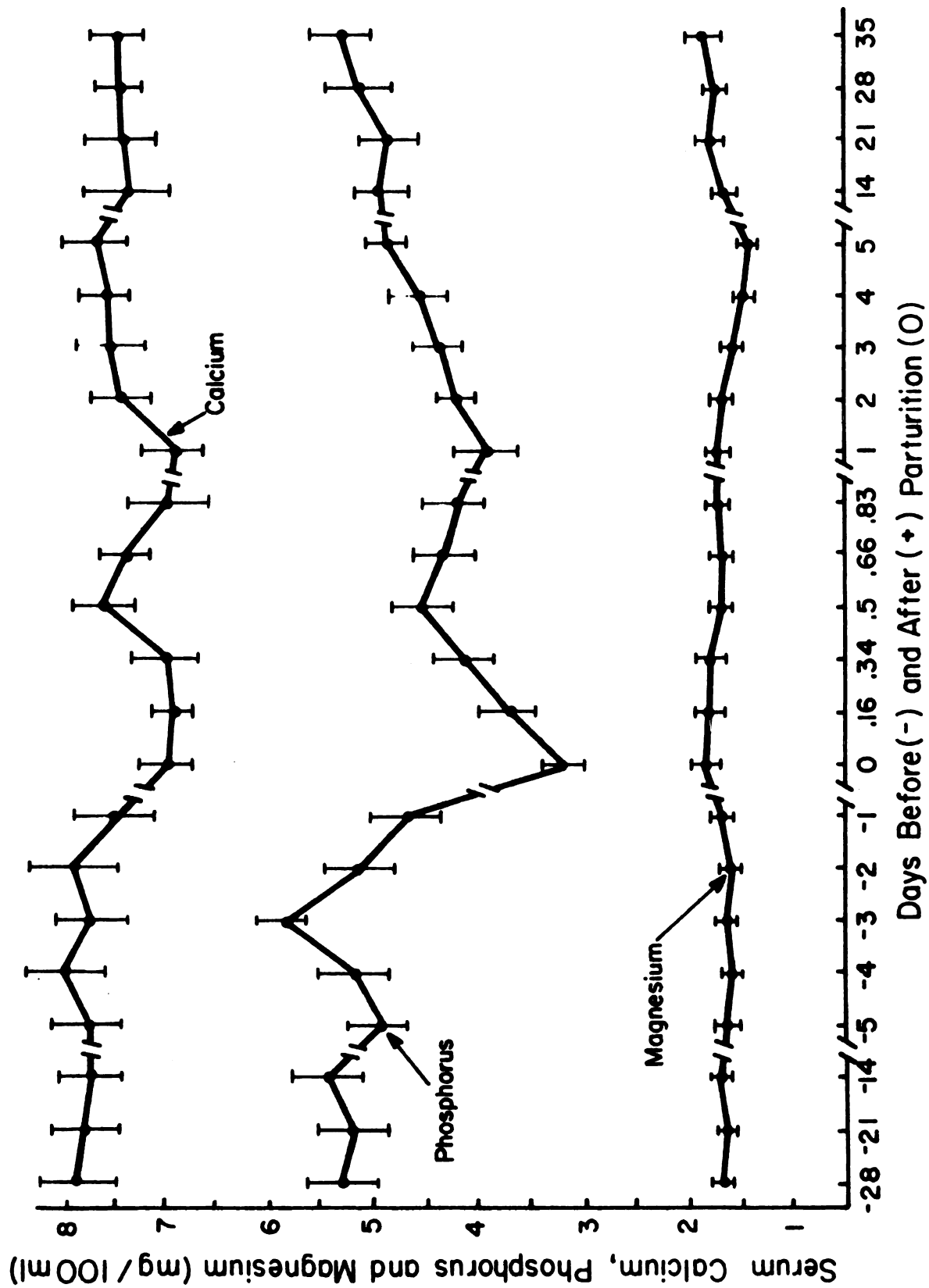
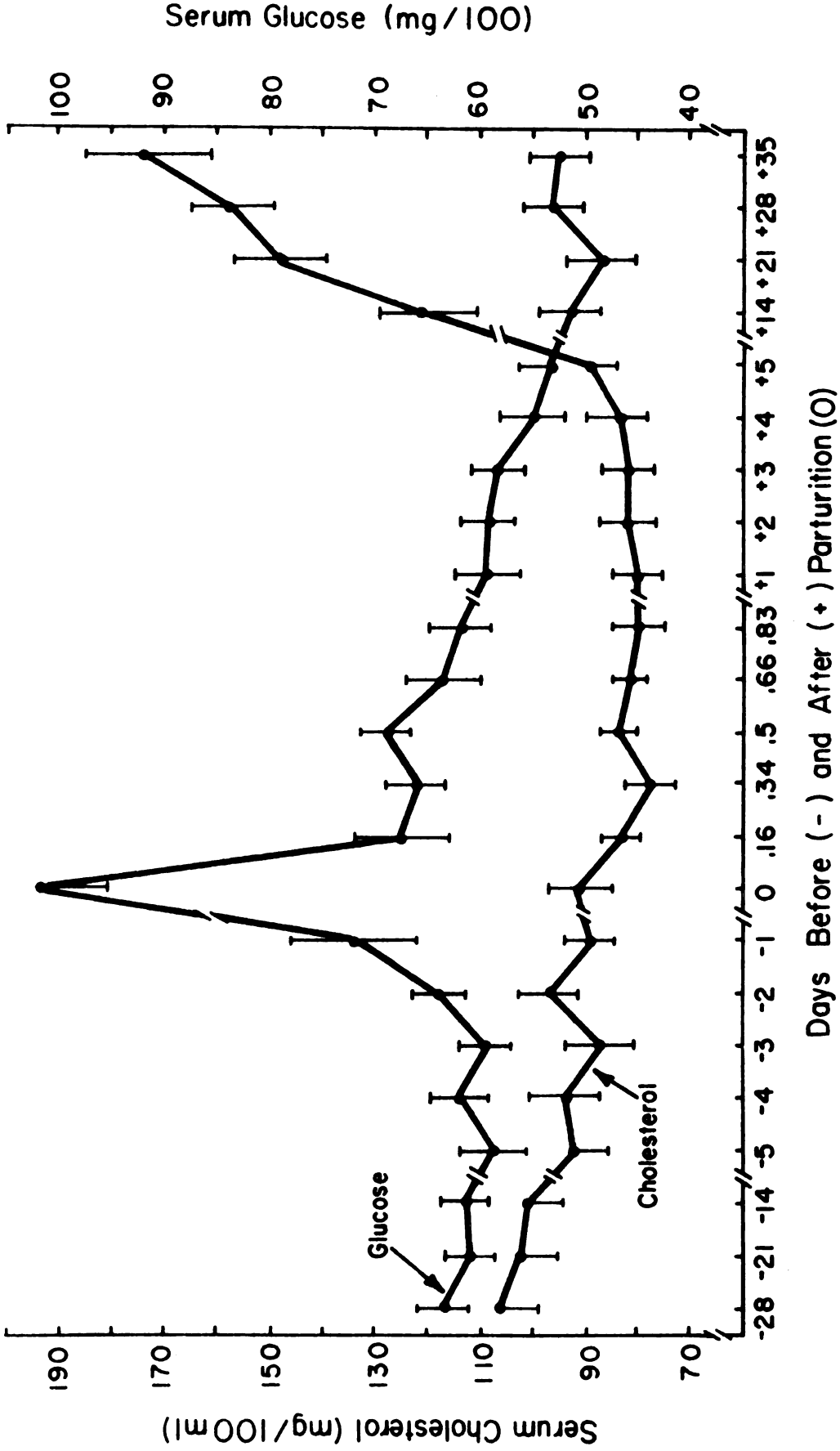

Serum Calcium, Phosphorus and Magnesium (mg/100 ml)
8
7
6
5
4
3
2
1
Calcium
Phosphorus
Magnesium
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before(-) and After (+) Parturition (0)

Fig. 3.--Mean blood serum cholesterol and glucose values and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.


Serum Glucose (mg/100)
100
90
80
70
60
50
40
Glucose
Cholesterol
Serum Cholesterol (mg/100 ml)
190
170
150
130
110
90
70
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 +1 +2 +3 +4 +5 +14 +21 +28 +35
Days Before (-) and After (+) Parturition (0)

time of calving, decreased to below prepartum levels during the first week of lactation and fluctuated thereafter. Cholesterol (Figure 3) declined prepartum reaching a low on the day of calving, and gradually increased during lactation to levels twice that observed prepartum.

Alkaline phosphatase (Figure 4) increased prepartum, peaked on the day of calving and declined to below prepartum levels during lactation.

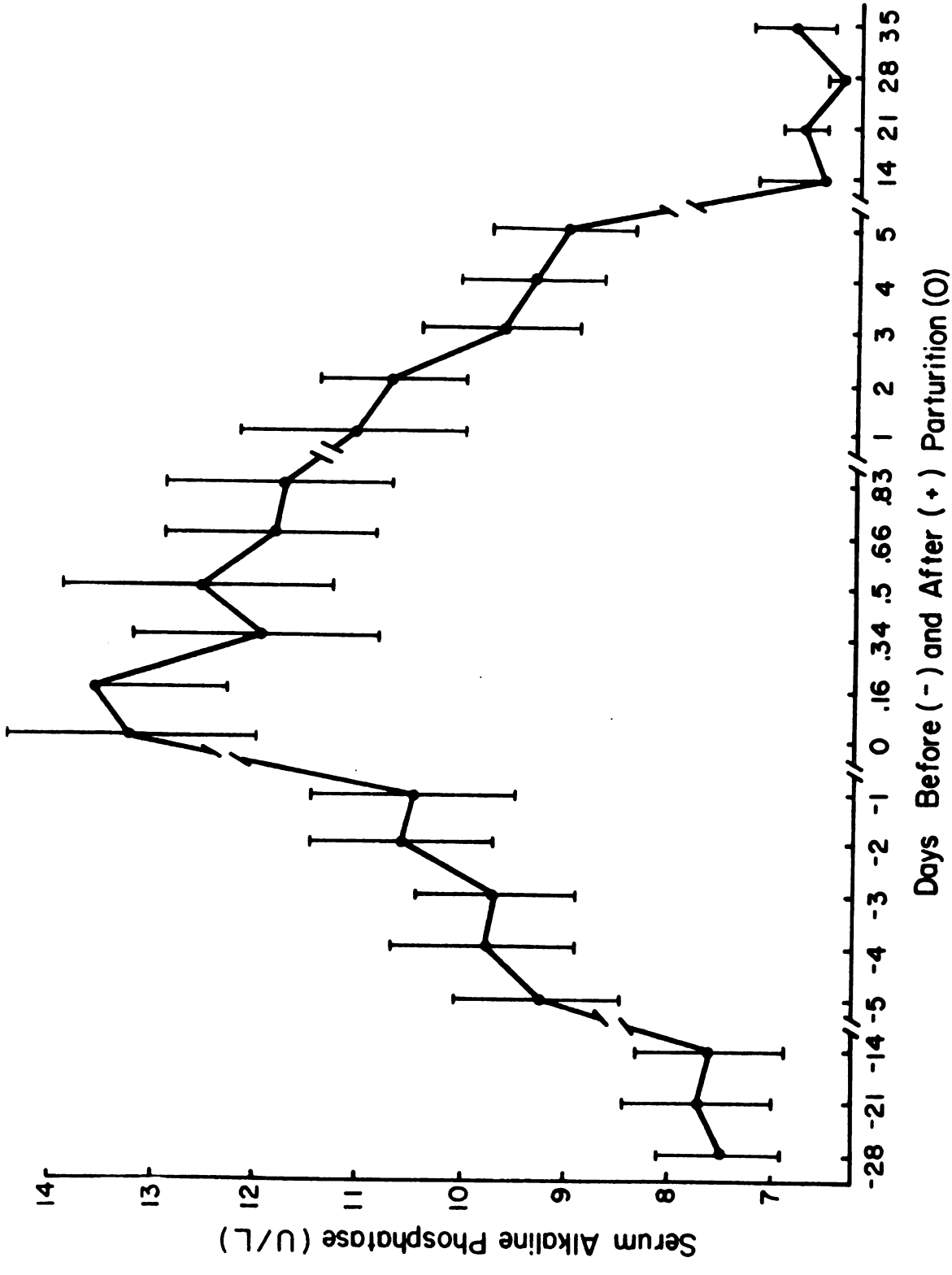
Urea nitrogen (Figure 5) began declining one week prepartum, peaked the day of calving, and declined to below prepartum levels during lactation.

Creatine phosphokinase (Figure 6) fluctuated prepartum, dropped sharply on calving day, and increased to above prepartum levels during lactation.

Glutamic oxalacetic transaminase (Figure 7) increased prepartum, peaked on the day of calving, and decreased to slightly agove prepartum levels during lactation.

Glutamic pyruvic transaminase (Figure 8) declined prepartum, peaked the day of calving, declining to below prepartum levels during the first week of lactation, and increased thereafter.

Fig. 4.--Mean blood serum alkaline phosphatase value (units/liter) and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.


Serum Alkaline Phosphatase (U/L)
14
13
12
11
10
9
8
7
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before (-) and After (+) Parturition (0)

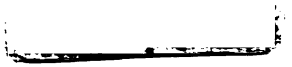


Fig. 5.--Mean blood serum urea nitrogen value and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.

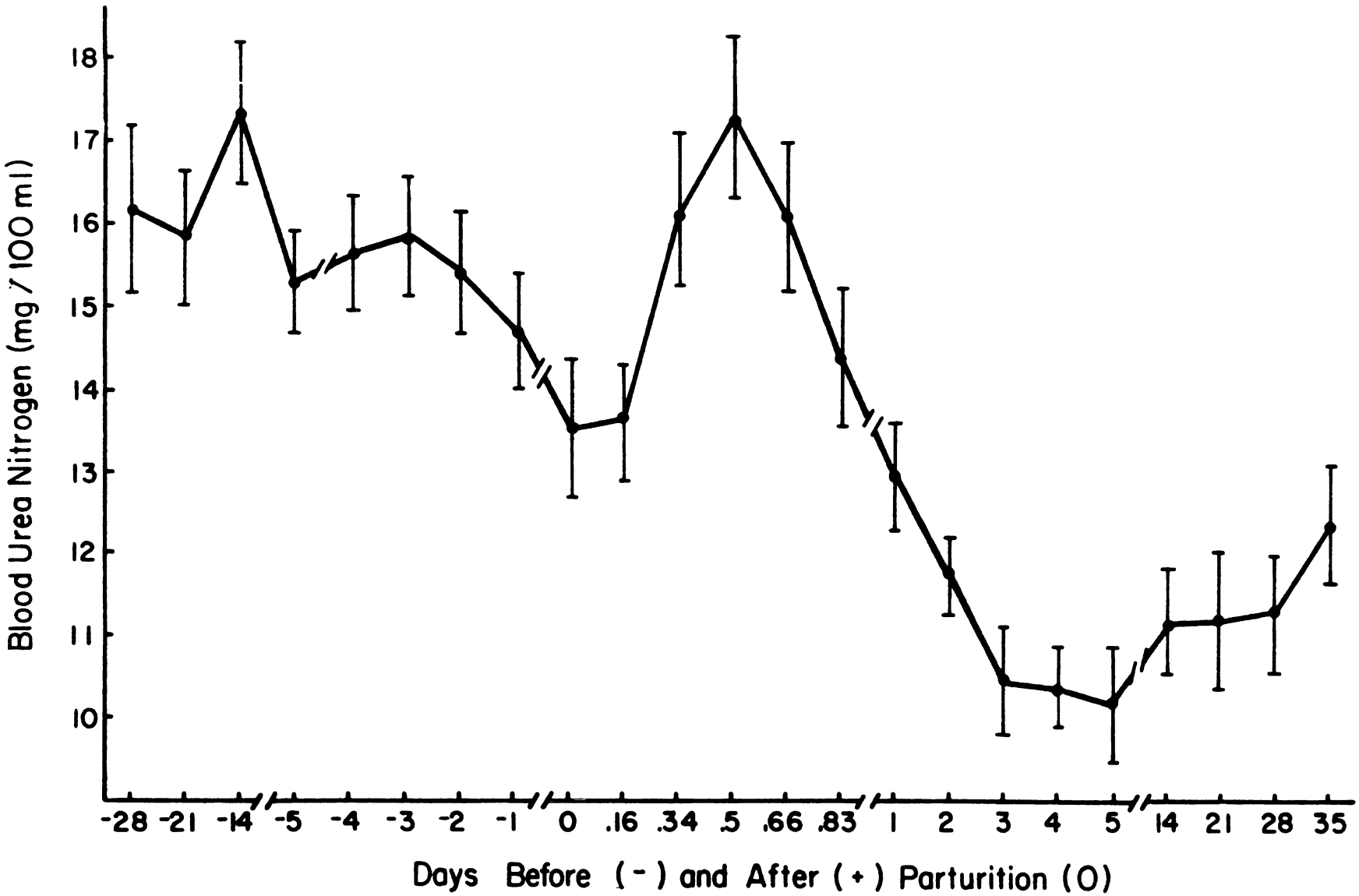

Blood Urea Nitrogen (mg % 100 ml)
18
17
16
15
14
13
12
11
10
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before (-) and After (+) Parturition (0)

Fig. 6.--Mean blood serum creatine phosphokinase value (units/liter) and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.

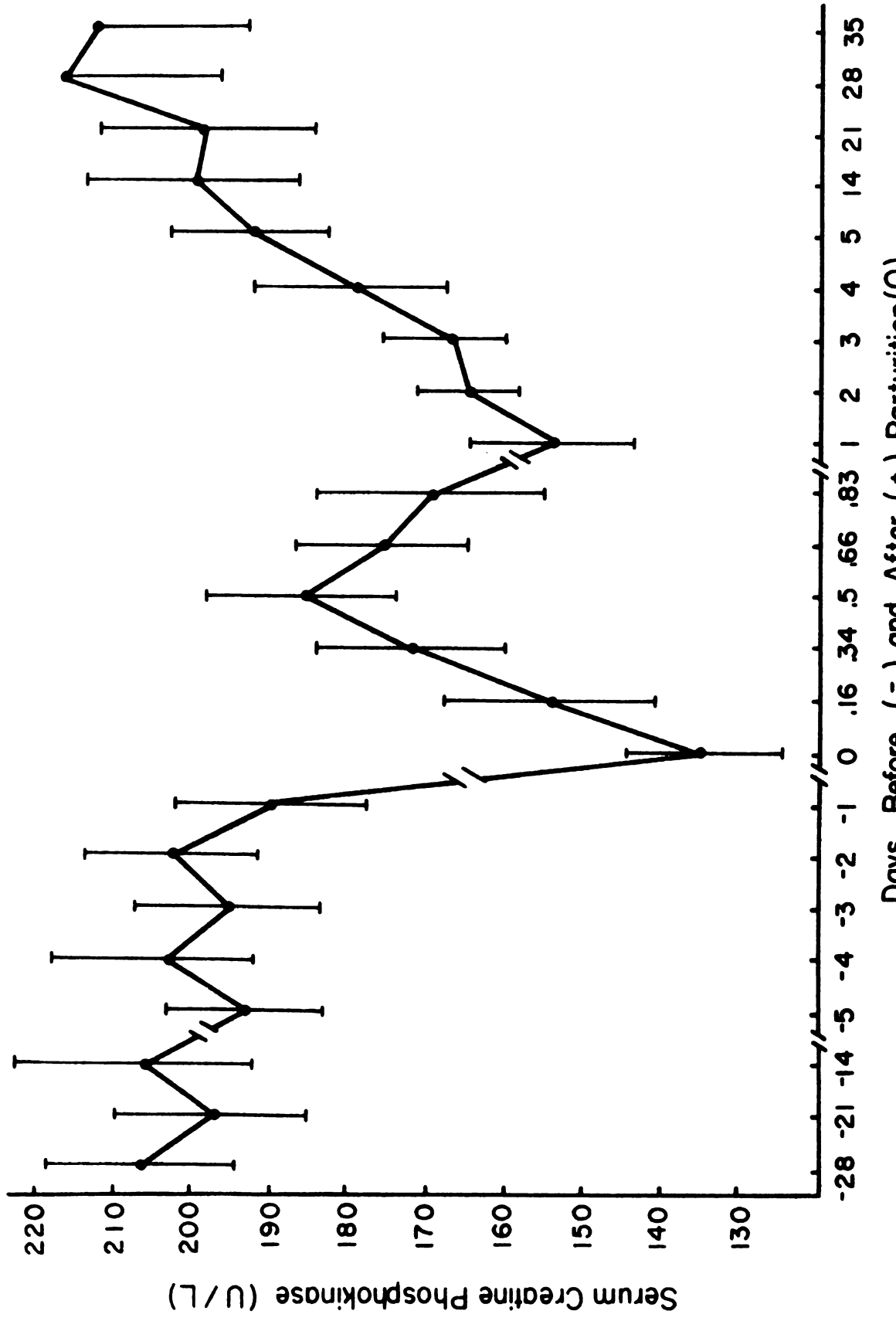

Serum Creatine Phosphokinase (U / L)
220
210
200
190
180
170
160
150
140
130
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before (-) and After (+) Parturition(0)

Fig. 7.--Mean blood serum glutamic oxalacetic transaminase value (units/liter) and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.

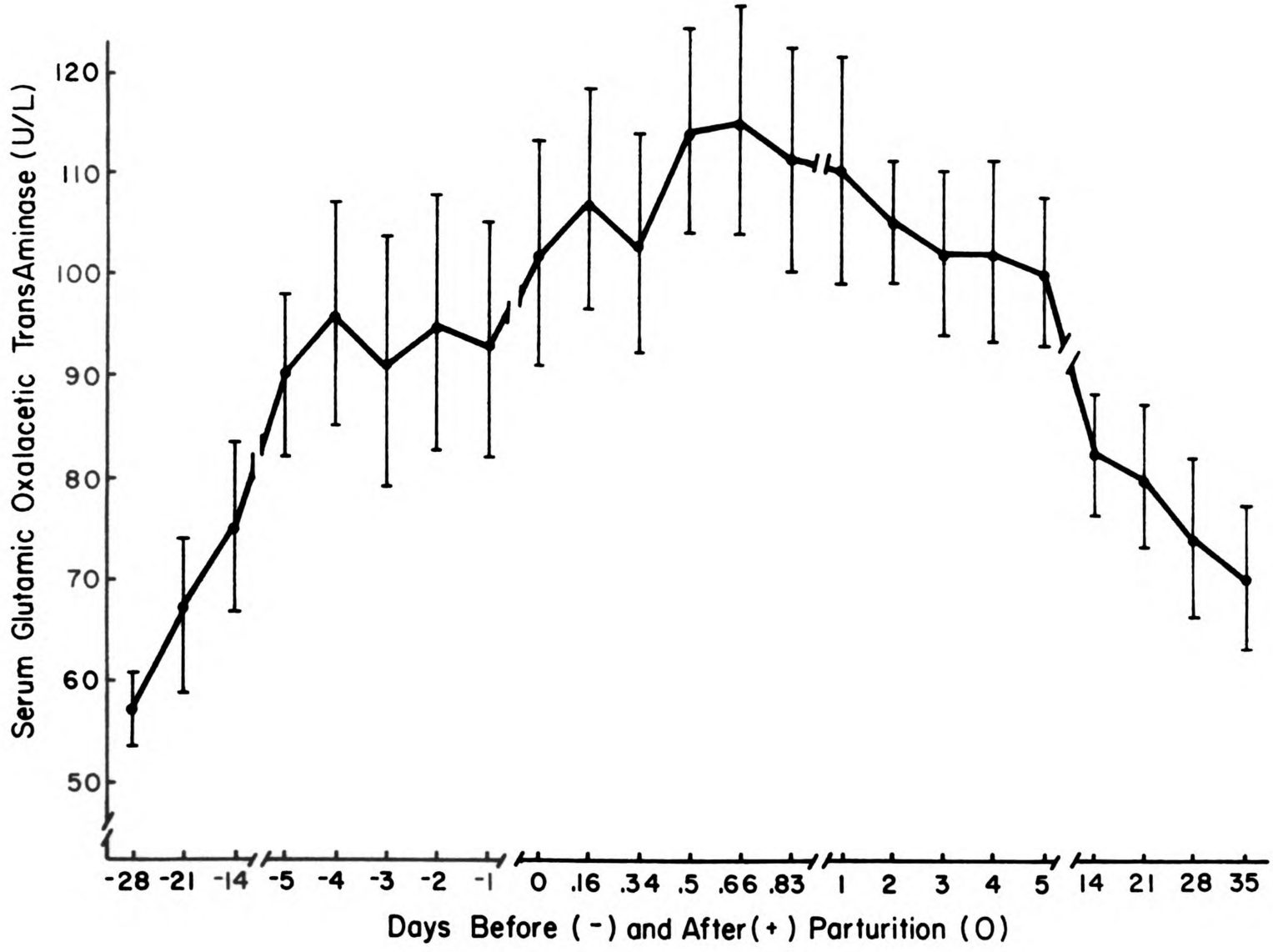
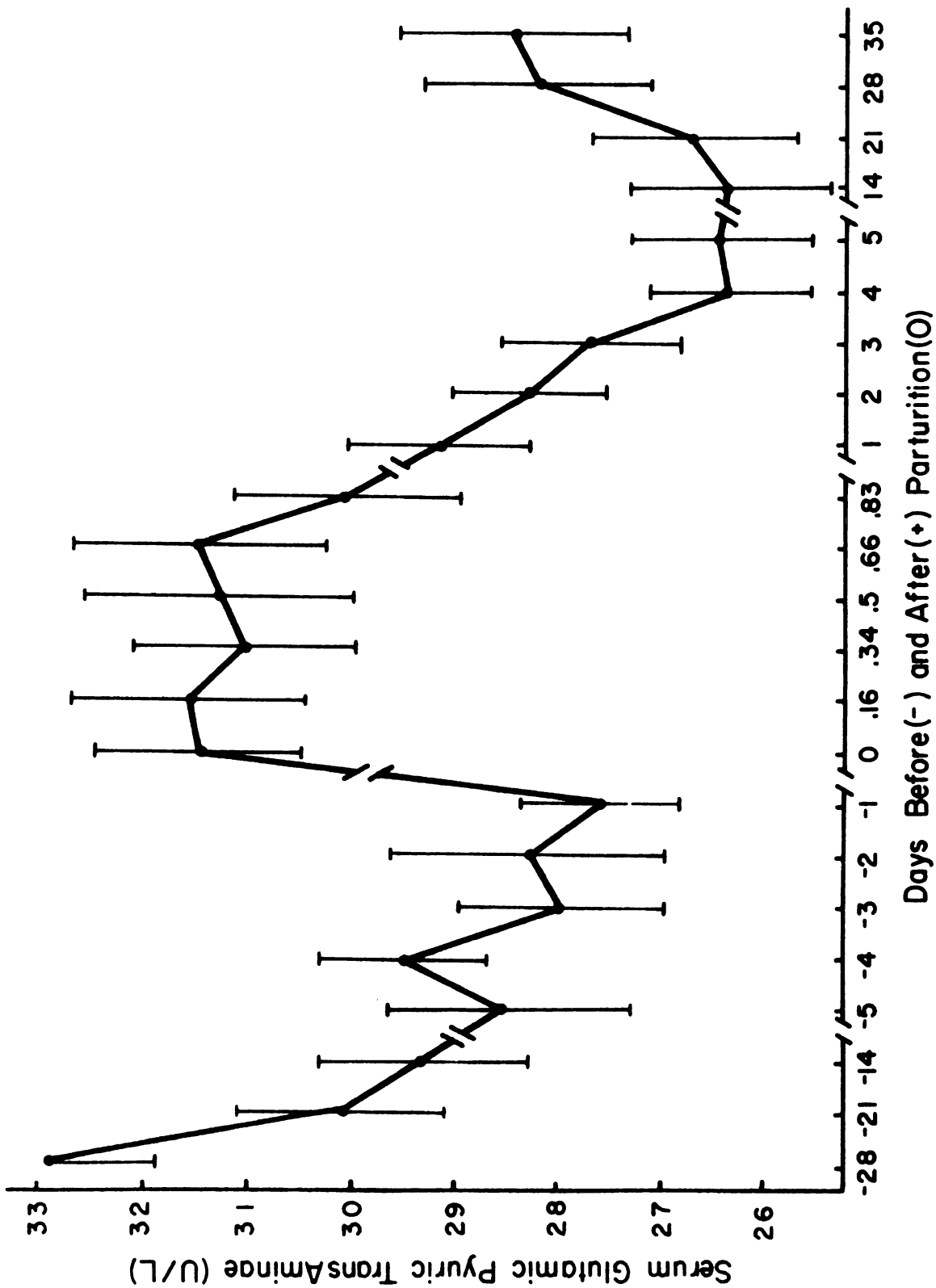

Serum Glutamic Oxalacetic TransAminase (U/L)
120
110
100
90
80
70
60
50
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before (-) and After (+) Parturition (0)

Fig. 8.--Mean blood serum glutamic pyruvic transaminase value (units/liter) and standard errors for 24 multiparous cows during late gestation, parturition and early lactation.


Serum Glutamic Pyuric TransAminase (U/L)
33
32
31
30
29
28
27
26
-28 -21 -14 -5 -4 -3 -2 -1 0 .16 .34 .5 .66 .83 1 2 3 4 5 14 21 28 35
Days Before(-) and After(+) Parturition(0)

DISCUSSION

A ration containing 0.37% potassium has been shown to be too low to promote maximum feed consumption, weight gains and milk yields, and the data suggest that the potassium requirement was greater than 0.37% K but not higher than 1.68% K of ration dry matter. Interpretation of two mathematical models of this data indicate that a dietary level of K ranging from 0.9 to 1.2% is suitable during gestation; while a 1.2% is more desirable during lactation.

The potassium requirement for lactating dairy cows in this study was considerably higher than the requirement (0.34%) observed for Friesland cows by DuToit (1934). The difference might be explained on the basis of level of milk production. In the present study the suggested K requirement was that which promoted near optimum feed intake and milk yields. In DuToit's (1934) study lactational performance was relatively low when compared to high producing dairy cows, and it is possible that feed consumption was insufficient to support higher production yields. Low amounts of dietary potassium can adversely effect appetite. The results of this study are in agreement with those of Dennis et al. (1975, 1976). Those authors reported that dairy cows' feed consumption increased by 1.5 kg when changing from 0.46% to a 0.97% K diet, and by 2.4 kg when changing from 0.45% or 0.55% to a 0.66% K diet. Similar

increased intake was observed in finishing beef steers fed diets containing 0.65 or 0.72% K, compared to those receiving either 0.27, 0.51 or 0.56% K diets (Roberts and St. Omer, 1965). Only slight intake differences were reported in sheep fed diets ranging from 0.3% to 0.6% K (Telle et al., 1964). The dramatic effects of low K intakes on appetite were well demonstrated in this study, when two of six prepartum cows showed complete feed refusal after eight days on the diet containing 0.26% K, and again when three of six lactating cows receiving the 0.37% K diet experienced complete feed refusal 17 days postpartum. Similar subtle effects on appetite resulting from low K diets have been observed in beef steers following 7 days on a diet containing less than 0.51% K (Delvin et al., 1969) and in lambs containing less than 0.3% K (Telle et al., 1964; Campbell and Roberts, 1965).

During this study the low K (0.37%) diet adversely influenced weight gains and resulted in a 84% greater weekly body weight loss. Weight losses are a consistent consequence of low K intakes and have both reported with fattening steers (Roberts and St. Omer, 1965; Delvin et al., 1969), lambs (Telle et al., 1964) and dairy cows (Dennis et al., 1976). Part of the weight loss may be the result of reduced feed intake, but there also exists a possibility that utilization of certain nutrients, particularly glucose, may be impaired by inadequate dietary potassium. The most pronounced weight losses occurred during the first and third weeks postpartum, coinciding with normal stress periods arising in-part from parturition and approaching lactational demands.

The lower concentrations of potassium in milk of cows receiving the low K diet (0.37%) suggest a sparing action on part of the cow to conserve tissue levels of K at the expense of altered milk K levels. An important reciprocal relationship existed in milk where sodium replaced potassium during periods of restricted K intakes, and potassium replaced sodium during elevated K intakes. Other workers (Pradhan and Hemken, 1968) have observed similar alteration in milk sodium and potassium during restricted potassium intakes. This observation may be observed only in cows fed extremely deficient diets and in early lactation, as Dennis et al. (1975, 1976) reported no changes in milk composition during the later part of lactation from cows fed diets greater than 0.5% K. This suggests that maintenance of tissue K levels and alterations in milk K are more pronounced when low K diets are augmented by high milk yields.

Effects of dietary K upon serum components are noteworthy. The depression in serum potassium experienced in cows receiving the K deficient diet (0.37%) can be explained by the fact that obligatory fecal K loss exists (Campbell and Roberts, 1965), and dietary K levels were not sufficient to meet the body's physiological demands. Data corroborating the effects of dietary K on serum K levels have been reported in beef steers (Roberts and St. Omer, 1965; Delvin et al., 1969) and heifers (St. Omer and Roberts, 1967), lambs (Telle et al., 1964; Campbell and Roberts, 1965) and to a lesser degree in dairy cows (Pradhan and Hemken, 1968). Possible explanation for the lack of consistent effects of dietary K upon serum K levels in other studies with dairy cows are probably a reflection of level and

duration of K fed, individual variability, number of animals per treatment and the stage of lactation during the experiment. Pradhan and Hemken (1968) used four pairs of cows past the peak of lactation averaging 12.4 kg milk. From their study, however, a general lowering from 21 to 19 mg/100 ml was observed in plasma K when a 0.26% K was fed for 10 weeks. Similarly, Dennis et al. (1976) fed four cows past the peak of lactation, that were averaging 26 kg milk, a diet containing 0.47% K for 12 weeks and reported no changes in blood K levels. In both of the above studies the cows were on the downward slope of the lactation curve with no physiological stresses associated with higher plains of nutrition needed to support maximum milk yields. In comparison, cows in this study were fed low potassium diets during late gestation and early lactation where stresses and nutritional demands are the greatest. At the conclusion of this experiment the cows receiving the higher K diets were averaging 34 kg of milk, while those receiving the 0.37% diet were averaging only 25 kg.

Remains difficult to ascertain from this study whether the increased gains observed from cows fed the higher K diets were actually due to a desirable influence of K or a stimulatory response from the added fiber content of these diets. There does occur an increased Na^{+}-K^{+} ratio in rumen fluid on low K diets and a reduction in rumen papillea length, although rumination remains normal (Telle et al., 1964; Delvin et al., 1969). Hubbert et al. (1958) reported that <u>in vitro</u> cellulose digestion was decreased with a high Na^{+}-K^{+} ratio. Therefore, if the Na^{+}-K^{+} ratio was altered in the 0.37% diet there

would occur a decreased action and/or number of rumen organisms available for digestion, while the altered Na^+-K^+ ratio would affect buffering capacity of the rumen. Is unknown whether the degressed appetite observed in the 0.37% diet was a direct effect of altered rumen function or a secondary response following a systemic depletion of tissue K reserves.

No additional benefits were observed from the 1.68% K in terms of serum K levels beyond those obtained in the 0.59 and 0.74% K diets. A study (Brink, 1961) with sheep indicated that serum K increased with increasing dietary K up to a level of 0.5%, but decreased at levels of 0.9% K. St. Omer and Roberts (1967) demonstrated in beef heifers that urinary K excretion reflected dietary K and observed a 3-fold increase in urinary K levels in heifers receiving 1086 meq. K compared to those receiving 440 meq. daily. The lack of any long term body storage of K places a large regulatory demand on the kidney to maintain and conserve tissue levels of K. Elevated intakes of K would cause an increase in the glomerular filtration rate, thus more K would be filtered at the kidney and excreted into the urine. Similarly, large amounts of excess dietary K would also be passed to the large intestine and excreted in the feces. Feeding of diets containing greater than 1.68% K to the dairy cow results in no observable benefits.

Reasons for other serum electrolyte changes are rather obscure. Serum phosphorus increased in the 0.37% diet, while sodium was depressed in the 1.68% K diet. Elevated phosphorus was a consequence of low K intakes in beef cattle (Roberts and St. Omer, 1965; Delvin

et al., 1969), but not in lambs (Telle et al., 1964; Campbell and Roberts, 1965). The reason for this observation in the bovine species remains unclear, but deserves further clarification. The depression in sodium observed in the 1.68% diet is most unusual when considering dietary sodium intake was similar for all treatments diets (Table 2). Delvin et al. (1969) observed a general lowering of serum sodium with increasing dietary K, but offered no explanation for this trend. High dietary K levels have been shown (Warner and Stacy, 1972a) to cause a net accumulation of ruminal K concentrations and force an accelerated absorption of sodium across the rumen wall. This additional circulatory sodium would cause a greater amount of sodium filtered at the kidney and excreted into the urine. Eventually, if dietary sodium intake remained the same, body stores of sodium would be depleted and a negative sodium balance would arise leading to a lowering of blood sodium concentration.

The reason for the higher blood magnesium concentrations in the 0.59% diet is not clear, as dietary intake of magnesium was similar between treatment diets (Table 2). Serum calcium was only slightly influenced ($P<.10$) by dietary potassium during the 28th through the 14th day prepartum, with the lowest calcium levels on the 0.37% diet. The importance of this observation is not known.

Serum enzymes were influenced by levels of dietary K. Serum alkaline phosphatase was lower and creatine phosphokinase higher on the low (0.37%) diet. Contributions to serum for SAP arise from bone, liver, intestine and kidney, whereas CPK activity represents skeletal muscle disorders. The lower SAP noted in the 0.37% diet may be

associated with lowered bone resorption, as also indicated by the elevation in serum phosphorus. Hibbs et al. (1946) found that the blood levels of alkaline phosphatase fall in conjunction with parturition, with the biggest drop found in paretic cows. Decrease in SAP activity is also observed in cattle as age increases. The higher CPK levels in the 0.37% diet probably represents skeletal tissue injury, which is a consistent consequence of potassium deficiency.

During the day of calving levels of SGOT and SGPT were influenced by level of dietary K, with the highest levels occurring in the 0.37% diet. SGOT serves as an indicator of organ damage, with the greatest concentrations being found in heart and liver. SGPT is present mainly in the liver and to a lesser extent in the kidney. Elevations of both SGOT and SGPT probably reflect some degree of liver damage occurring in the 0.37% diet during the day of calving. No dietary influence on these enzymes was observed after the fifth day postpartum.

Blood urea nitrogen was lowest in the 1.68% diet, but probably reflected a lowered dietary protein content in this diet compared to other treatment diets (Table 2). The reason for the elevation in serum cholesterol during the day of calving for the 0.37% diet is unknown. Similarly the depression in cholesterol toward the end of the experiment in all diet groups is not clear. Cholesterol is involved in lipid transport and a positive association usually exists with milk yield. Cows receiving the low K diet (0.37%) failed to achieve expected maximum milk yields. A general lowering trend for serum glucose occurred in all diet groups as lactation advanced.

However, cows receiving the 1.68% K diet demonstrated the lowest serum concentration of glucose. The reason for this trend is unclear.

CONCLUSIONS

A feeding trial was conducted to study the effects of feeding various levels of dietary potassium to the dairy cow during late gestation, parturition and early lactation. Twenty-four Holstein cows were used in the trial which consisted of four dietary treatments of 0.37, 0.59, 0.74 and 1.68% potassium, with six cows each.

Potassium deficiency, as indicated by poor appetite, loss in body weight, lowered milk yields and low serum K, was observed in cows receiving the 0.37% potassium diet. Consequences of low K intakes were most apparent beginning the week of calving when serum metabolite changes reflected varying degrees of alterations in phosphorus homostatis, lipid metabolism and liver dysfunction. An important inverse relationship was observed in milk, where in general, an increased substitution of potassium for sodium occurred as dietary potassium concentration increased.

A diet containing 0.37% potassium was inadequate for support of health and optimum lactational performance of dairy cows, while a diet containing 0.59 to 1.68% potassium had similar responses in all criteria used. From regression analysis the optimum level of dietary K for optimum feed intake, milk yields and normal blood serum parameters ranged from 0.9 to 1.2% K during gestation and 1.2% K during lactation.

APPENDIX

APPENDIX

Table A-1.--Blood Serum Metabolites From Cows Fed Four Dietary Levels of Potassium During Late Gestation, Parturition and Early Lactation.

Serum Metabolite	Diet Group	Prepartum (days)							
		28	21	14	5	4	3	2	1
Potassium	D1	15.1	13.8	13.9	13.4	13.4	13.0	13.9	13.8
mg/100 ml	D2	16.4	17.1	16.8	16.8	16.4	16.4	16.2	16.6
	D3	16.6	16.2	15.8	15.0	15.3	15.2	14.5	15.1
	D4	16.6	17.1	16.9	16.2	16.0	16.1	15.9	16.9
Overall period mean ± S.E.M.		16.1±0.3	16.1±0.4	15.9±0.5	15.3±0.4	15.3±0.5	15.2±0.5	15.1±0.4	15.6±0.4
Sodium	D1	296.6	292.0	297.4	310.7	301.8	299.8	304.7	304.1
mg/100 ml	D2	297.3	280.9	297.7	295.6	304.8	301.9	305.7	300.9
	D3	325.8	316.2	325.1	320.6	315.9	321.0	315.4	323.5
	D4	274.8	264.4	261.2	262.4	259.2	259.9	263.0	267.2
Overall period mean ± S.E.M.		298.6±7.5	288.4±8.5	295.3±9.3	297.3±8.0	295.5±9.3	295.6±8.6	297.2±7.9	298.9±7.8
Magnesium	D1	1.5	1.5	1.6	1.6	1.5	1.4	1.6	1.6
mg/100 ml	D2	2.3	2.1	2.3	2.3	2.3	2.3	2.2	2.2
	D3	1.5	1.3	1.3	1.2	1.2	1.3	1.1	1.4
	D4	1.6	1.6	1.5	1.4	1.4	1.5	1.5	1.6
Overall period mean ± S.E.M.		1.7±0.1	1.6±0.1	1.7±0.1	1.6±0.1	1.6±0.1	1.6±0.1	1.6±0.1	1.7±0.1
Calcium	D1	5.6	7.7	7.7	6.9	7.7	7.5	7.2	7.2
mg/100 ml	D2	8.5	6.9	7.1	7.8	8.0	6.7	8.0	7.2
	D3	9.1	8.9	8.1	8.3	8.5	8.0	9.8	8.1
	D4	8.2	7.9	8.1	7.9	7.8	8.7	8.5	7.8
Overall period mean ± S.E.M.		7.9±0.5	7.8±0.4	7.7±0.3	7.8±0.4	8.0±0.4	7.7±0.3	7.9±0.4	7.6±0.4
Phosphorus	D1	3.9	6.0	6.5	4.9	5.8	5.2	5.5	5.9
mg/100 ml	D2	6.4	4.9	4.0	5.0	5.5	4.6	5.4	4.1
	D3	5.3	5.4	5.2	5.3	4.9	5.2	4.7	4.7
	D4	5.6	4.8	5.5	4.6	4.7	4.9	5.1	4.2
Overall period mean ± S.E.M.		5.3±0.4	5.3±0.4	5.5±0.4	5.0±0.3	5.2±0.4	5.0±0.3	5.2±0.3	4.7±0.4

Table A-1.--Continued.

Serum Metabolite	Diet Group	Prepartum (days)							
		28	21	14	5	4	3	2	1
Creatinine	D1	0.9	1.0	1.3	1.3	1.3	1.5	1.2	1.4
mg/100 ml	D2	0.9	1.1	1.0	1.3	1.1	1.4	1.6	1.6
	D3	1.2	1.1	1.3	1.4	1.6	1.4	1.7	1.4
	D4	1.0	1.2	1.3	1.1	1.2	1.2	1.3	1.1
Overall period mean ± S.E.M.		1.0±.05	1.1±.05	1.2±.06	1.3±.08	1.3±.08	1.4±.09	1.5±0.1	1.4±.12
Alkaline	D1	5.6	6.0	6.0	7.1	7.7	7.9	8.6	8.5
Phosphatase	D2	8.2	7.9	8.0	9.5	10.2	8.3	10.2	9.2
(μ/L)	D3	8.2	7.9	7.2	9.5	10.3	10.3	11.1	11.6
	D4	8.0	9.1	9.1	11.0	11.1	12.3	12.4	12.8
Overall period mean ± S.E.M.		7.5±0.6	7.7±0.7	7.6±0.7	9.3±0.8	9.8±0.9	9.7±0.8	10.6±0.9	10.5±1.0
Glutamic-Pyruvic	D1	29.2	28.8	27.3	27.5	29.0	26.8	24.7	28.3
transaminase	D2	38.8	30.2	31.5	29.2	32.3	28.3	32.8	28.5
(μ/L)	D3	37.0	32.7	28.8	29.5	29.7	27.8	27.0	27.8
	D4	30.2	28.8	29.3	28.3	27.2	29.7	28.8	25.7
Overall period mean ± S.E.M.		33.8±1.4	30.1±1.0	29.3±1.0	28.6±1.2	29.5±0.8	28.0±1.0	28.3±1.3	27.6±0.8
Cholesterol	D1	114.0	93.3	102.8	99.8	109.8	91.8	100.3	97.2
mg/100 ml	D2	108.5	97.5	96.7	83.7	98.3	78.0	94.8	79.5
	D3	118.0	119.2	107.5	94.0	90.7	84.8	90.0	94.0
	D4	86.3	91.0	97.8	88.0	82.5	94.5	95.2	85.5
Overall period mean ± S.E.M.		106.7±7.0	100.3±6.9	101.2±7.2	91.6±6.3	94.3±7.1	87.3±6.9	95.2±6.6	89.0±5.1
Creatine	D1	166.6	214.0	239.7	191.2	224.5	185.5	223.5	231.7
Phosphokinase	D2	263.7	205.7	204.7	216.5	234.7	200.3	238.2	180.7
(μ/L)	D3	225.5	217.5	203.5	211.5	194.7	209.0	187.2	185.8
	D4	176.8	152.8	181.0	152.0	158.2	191.5	164.3	160.0
Overall period mean ± S.E.M.		208.2±12.8	197.5±12.2	207.2±15.1	192.8±10.6	203.0±14.3	196.5±11.6	203.3±11.8	189.5±11.8

Table A-1.--Continued.

Serum Metabolite	Diet Group	Prepartum (days)							
		28	21	14	5	4	3	2	1
Glutamic-Oxalacetic transaminase (u/L)	D1	44.3	60.8	77.8	97.8	102.7	86.0	97.2	98.7
	D2	58.0	82.0	85.5	89.5	94.2	78.3	95.8	86.7
	D3	67.7	67.5	67.5	94.8	115.3	116.3	108.0	113.5
	D4	57.7	59.0	67.8	78.5	73.8	82.8	79.5	72.0
Overall period mean ± S.E.M.		56.9±4.1	67.3±8.0	74.6±7.6	90.2±7.8	96.5±11.4	90.9±11.6	95.1±11.6	92.7±11.6
Urea Nitrogen mg/100 ml	D1	15.2	15.0	17.8	17.5	16.4	16.2	16.1	16.7
	D2	17.6	16.5	18.8	15.2	17.4	15.9	17.7	16.0
	D3	17.6	17.3	17.7	14.4	14.9	16.5	14.4	14.4
	D4	13.9	14.8	14.9	13.7	13.2	14.3	13.2	11.8
Overall period mean ± S.E.M.		16.1±1.0	15.9±0.8	17.3±0.8	15.2±0.6	15.5±0.7	15.7±0.7	15.4±0.7	14.7±0.7
Glucose mg/100 ml	D1	62.0	62.0	62.1	58.3	59.2	53.2	67.3	66.2
	D2	66.5	53.8	59.0	54.5	66.3	53.2	63.0	79.8
	D3	66.0	65.3	61.5	60.5	63.5	61.7	62.0	76.2
	D4	60.0	63.3	63.0	61.8	59.8	55.8	64.0	65.5
Overall period mean ± S.E.M.		63.6±2.2	61.1±2.6	61.4±2.5	58.8±2.8	62.2±2.7	59.7±2.5	64.1±2.5	71.9±5.9

Serum Metabolism	Diet Group	Parturition (hours)					
		0	4	8	12	16	20
Potassium mg/100 ml	D1	13.9	13.7	14.6	14.6	15.1	15.8
	D2	16.2	15.7	16.6	16.5	17.0	17.0
	D3	14.8	15.2	16.1	15.4	14.9	15.8
	D4	16.1	16.9	17.1	17.1	16.0	16.8
Overall period mean ± S.E.M.		15.3±0.3	15.4±0.5	16.1±0.3	15.9±0.4	15.8±0.4	16.3±0.3

Table A-1.--Continued.

Serum Metabolite	Diet Group	Parturition (hours)					
		0	4	8	12	16	20
Sodium	D1	301.3	307.2	304.3	318.7	307.8	322.1
mg/100 ml	D2	316.0	311.3	310.6	318.9	299.2	310.5
	D3	320.5	319.5	323.6	330.4	335.7	319.0
	D4	277.5	279.5	289.9	272.7	274.7	273.6
Overall period mean ± S.E.M.		303.8±6.6	304.4±6.4	307.1±6.2	310.2±7.1	304.4±8.4	306.3±8.4
Magnesium	D1	1.6	1.5	1.5	1.5	1.5	1.6
mg/100 ml	D2	2.5	2.5	2.4	2.4	2.3	2.5
	D3	1.4	1.3	1.5	1.3	1.3	1.4
	D4	1.7	1.8	1.8	1.7	1.7	1.6
Overall period mean ± S.E.M.		1.8±0.1	1.8±0.1	1.8±0.1	1.7±0.1	1.7±0.1	1.8±0.1
Calcium	D1	7.3	6.7	7.6	7.5	7.8	7.5
mg/100 ml	D2	6.5	7.5	7.0	7.7	7.2	6.6
	D3	6.9	6.7	6.2	7.5	7.0	6.6
	D4	7.5	6.9	7.3	7.7	7.6	7.4
Overall period mean ± S.E.M.		7.0±0.3	6.9±0.3	7.0±0.4	7.6±0.3	7.4±0.3	7.0±0.4
Phosphorus	D1	4.4	4.8	5.4	5.7	6.1	6.2
mg/100 ml	D2	2.3	3.5	4.1	4.1	3.5	2.9
	D3	3.0	3.8	3.7	4.1	4.0	4.0
	D4	3.0	2.7	3.5	4.4	3.8	3.9
Overall period mean ± S.E.M.		3.2±0.2	3.7±0.3	4.2±0.3	4.6±0.3	4.4±0.3	4.3±0.4
Creatinine	D1	1.5	2.0	1.4	1.7	1.5	1.5
mg/100 ml	D2	1.7	1.9	1.5	1.3	1.2	1.1
	D3	1.4	1.7	1.6	1.6	1.3	1.2
	D4	1.5	1.1	1.2	1.2	1.3	1.2
Overall period mean ± S.E.M.		1.5±	1.7±.17	1.4±.09	1.4±.12	1.3±.07	1.3±.08

Table A-1.--Continued.

Serum Metabolite	Diet Group	Parturition (hours)					
		0	4	8	12	16	20
Alkaline Phosphatase (μ/L)	D1	11.8	12.3	10.7	10.6	10.4	10.8
	D2	11.3	13.1	10.9	11.5	10.4	10.2
	D3	13.2	12.7	11.3	11.8	12.0	11.3
	D4	16.6	16.5	15.2	16.7	14.9	14.8
Overall period mean ± S.E.M.		13.2±1.2	13.6±1.3	12.0±1.2	12.6±1.3	11.9±1.0	11.8±1.1
Glutamic-Pyruvic transaminase (μ/L)	D1	32.2	32.8	32.5	34.2	33.5	34.0
	D2	31.2	33.8	32.7	33.7	34.0	29.2
	D3	32.5	32.8	30.2	28.5	30.0	28.3
	D4	30.0	27.0	29.2	28.7	28.3	28.8
Overall period mean ± S.E.M.		31.5±1.0	31.6±1.3	31.1±1.1	31.3±1.3	31.5±1.2	30.1±1.1
Cholesterol mg/100 ml	D1	104.2	94.5	85.8	97.5	92.5	94.2
	D2	81.0	76.5	79.3	79.2	73.1	67.7
	D3	87.2	78.2	70.0	78.3	77.7	74.3
	D4	92.8	80.8	77.8	81.3	82.0	83.3
Overall period mean ± S.E.M.		91.3±5.8	82.5±3.9	78.3±5.5	84.0±4.0	81.3±4.5	79.9±4.6
Creatine Phosphokinase (μ/L)	D1	176.2	178.7	207.7	210.0	222.3	242.8
	D2	118.8	179.0	199.8	217.0	182.2	141.8
	D3	131.5	158.7	156.3	172.0	162.2	155.3
	D4	108.5	102.0	128.8	150.0	137.8	141.7
Overall period mean ± S.E.M.		133.8±9.6	154.6±13.1	173.2±11.6	187.3±12.2	176.1±10.8	170.4±14.3
Glutamic-Oxalacetic transaminase (μ/L)	D1	112.2	113.7	104.5	127.3	129.8	130.2
	D2	99.2	115.2	106.8	111.3	116.0	105.2
	D3	112.5	112.5	111.3	119.0	120.5	116.3
	D4	84.0	87.5	91.0	97.5	94.2	94.0
Overall period mean ± S.E.M.		101.9±10.8	107.2±11.0	103.4±11.0	113.8±10.2	115.1±10.8	111.4±10.6

Table A-1.--Continued.

Serum Metabolite	Diet Group	Parturition (hours)					
		0	4	8	12	16	20
Urea Nitrogen mg/100 ml	D1	17.1	16.1	19.3	18.4	18.3	17.6
	D2	13.4	13.4	14.8	16.5	15.7	13.3
	D3	13.3	13.9	16.9	19.6	16.7	14.5
	D4	10.2	11.3	14.0	14.6	13.5	12.3
Overall period mean ± S.E.M.		13.5±0.8	13.6±0.7	16.2±0.9	17.3±1.0	16.1±0.9	14.4±0.8
Glucose mg/100 ml	D1	101.0	61.7	62.2	72.3	66.2	69.1
	D2	121.2	77.5	75.0	69.0	59.7	54.0
	D3	100.7	66.0	63.5	68.0	63.0	56.4
	D4	84.8	65.0	62.8	66.7	67.5	68.8
Overall period mean ± S.E.M.		101.9±6.3	67.5±4.3	65.9±3.3	69.0±2.7	63.3±3.3	62.1±3.3

Serum Metabolite	Diet Group	Postpartum (days)								
		1	2	3	4	5	14	21	28	35
K	D1	14.7	13.6	13.8	13.2	13.2	13.6	13.7	13.6	14.0
	D2	17.3	16.9	16.8	16.2	15.6	15.9	15.7	16.0	16.0
	D3	15.2	16.6	15.9	15.6	15.5	15.1	16.6	16.5	15.8
	D4	17.3	16.6	16.8	16.4	16.6	16.7	16.2	17.3	16.8
Overall period mean ± S.E.M.		16.1±0.3	15.9±0.4	15.8±0.5	15.4±0.5	15.2±0.4	15.3±0.4	15.5±0.4	15.9±0.5	15.7±0.4
Na	D1	305.9	299.4	304.1	298.2	300.1	303.2	288.5	284.8	285.9
	D2	316.3	324.0	305.8	316.7	311.1	303.5	291.6	298.6	296.1
	D3	321.8	333.4	331.6	310.7	308.8	308.2	311.4	302.6	307.5
	D4	275.6	272.6	270.7	269.8	265.9	268.5	263.6	289.9	284.9
Overall period mean ± S.E.M.		304.9±7.6	307.3±9.2	303.1±7.7	298.8±8.3	296.5±8.0	295.8±6.4	288.8±6.4	293.9±6.7	293.8±6.3

Table A-1.--Continued.

Serum Metabolite	Diet Group	Postpartum (days)								
		1	2	3	4	5	14	21	28	35
Mg	D1	1.5	1.6	1.4	1.4	1.4	1.6	1.6	1.5	1.7
	D2	2.4	2.3	2.2	2.0	2.0	2.3	2.4	2.4	2.4
	D3	1.3	1.3	1.3	1.2	1.1	1.3	1.4	1.6	1.4
	D4	1.7	1.7	1.5	1.5	1.4	1.7	1.9	1.7	2.1
Overall period mean ± S.E.M.		1.8±0.1	1.7±0.1	1.6±0.1	1.5±0.1	1.5±0.1	1.7±0.1	1.8±0.1	1.8±0.1	1.9±0.1
Ca	D1	7.2	7.4	8.2	7.4	7.1	6.8	7.2	7.7	7.1
	D2	7.3	7.9	7.5	7.3	8.2	7.7	7.9	7.4	7.1
	D3	6.3	6.9	7.0	7.5	7.8	7.4	7.6	7.3	8.1
	D4	7.0	7.9	7.4	8.1	7.6	7.7	7.1	7.6	7.7
Overall period mean ± S.E.M.		6.9±0.3	7.5±0.3	7.5±0.3	7.6±0.3	7.7±0.3	7.4±0.4	7.4±0.4	7.5±0.2	7.5±0.3
P	D1	5.5	5.1	5.6	4.9	5.1	4.8	5.4	6.3	5.6
	D2	3.6	4.2	4.4	4.7	5.3	5.4	5.7	5.4	5.5
	D3	3.5	3.7	3.7	3.8	4.3	4.5	4.7	4.0	4.9
	D4	3.4	4.0	3.9	5.0	4.9	5.1	4.0	5.0	5.4
Overall period mean ± S.E.M.		3.9±0.3	4.3±0.2	4.4±0.3	4.6±0.3	4.9±0.2	5.0±0.3	5.0±0.3	5.2±0.4	5.4±0.3
Cr	D1	1.6	1.2	1.3	1.5	1.2	1.2	1.0	0.9	1.0
	D2	1.2	1.1	1.0	1.2	0.9	0.8	0.8	0.8	0.8
	D3	1.5	1.3	1.5	1.5	1.3	1.1	0.9	0.9	1.0
	D4	1.3	1.2	1.1	1.3	1.2	1.1	1.0	0.8	1.0
Overall period mean ± S.E.M.		1.4±.11	1.2±.09	1.2±.09	1.4±.13	1.1±.08	1.1±.08	0.9±.05	0.8±.05	0.9±.04
A.P.	D1	8.7	9.0	8.7	9.1	8.4	5.6	6.5	6.1	6.0
	D2	10.9	9.9	9.6	9.3	9.7	6.9	8.0	6.8	6.2
	D3	11.8	11.0	9.2	8.7	9.0	5.8	5.9	5.3	7.6
	D4	12.9	13.2	11.1	10.7	9.3	8.2	6.7	7.3	7.9
Overall period mean ± S.E.M.		11.1±1.1	10.8±0.7	9.7±0.8	9.4±0.7	9.1±0.7	6.6±0.6	6.8±0.6	6.4±0.4	6.9±0.4

Table A-1.--Continued.

Serum Metabolite	Diet Group	Postpartum (days)								
		1	2	3	4	5	14	21	28	35
SGPT	D1	29.0	29.1	28.5	26.0	24.3	25.5	28.5	26.0	24.8
	D2	31.8	31.0	30.5	28.8	30.2	30.2	29.3	30.7	30.5
	D3	29.2	27.2	27.2	25.0	26.5	23.3	25.7	27.7	28.7
	D4	26.8	26.2	25.0	25.7	24.8	26.7	23.7	28.8	30.2
Overall period mean ± S.E.M.		29.2±0.9	28.4±0.8	27.8±0.9	26.4±0.8	26.5±0.9	26.4±1.0	26.8±1.0	28.3±1.1	28.5±1.1
Cholesterol	D1	88.3	86.3	95.7	86.5	84.2	98.0	123.8	121.8	134.5
	D2	80.0	78.8	80.0	81.8	101.5	143.0	170.0	170.0	193.7
	D3	79.5	74.6	76.8	79.2	86.6	119.5	159.7	165.5	174.7
	D4	72.0	87.0	76.2	87.0	86.0	119.7	137.0	171.3	194.3
Overall period mean ± S.E.M.		80.0±4.6	81.7±5.6	82.2±4.8	83.6±5.6	89.5±5.3	120.1±9.4	147.7±9.2	157.2±8.6	174.3±11.8
CPK	D1	197.8	182.0	194.6	197.8	198.7	182.0	211.3	229.1	167.0
	D2	160.5	187.0	184.5	196.1	220.7	250.7	250.0	285.0	295.5
	D3	143.5	152.0	154.8	160.0	178.7	186.3	191.8	180.7	203.3
	D4	125.0	139.3	135.8	169.3	172.0	181.7	143.2	180.7	187.2
Overall period mean ± S.E.M.		156.7±10.8	165.1±7.8	167.5±8.4	180.8±12.0	192.5±10.4	200.2±13.9	199.0±14.1	218.9±20.2	213.3±19.8
GOT	D1	107.7	111.0	115.7	117.0	103.7	81.7	94.8	98.5	64.0
	D2	122.3	111.3	95.7	86.8	94.0	90.7	75.1	63.0	60.0
	D3	124.8	103.3	106.0	110.1	112.3	67.5	64.0	58.3	81.5
	D4	90.3	95.5	89.0	96.0	91.0	87.7	84.5	76.3	77.7
Overall period mean ± S.E.M.		110.5±10.8	105.3±6.1	101.6±8.2	102.5±8.8	100.3±7.1	81.9±5.9	79.3±6.9	74.0±8.2	70.8±7.1
UN	D1	15.4	10.8	9.5	11.5	12.7	10.8	11.4	9.8	10.3
	D2	12.5	11.6	11.6	11.6	11.4	11.7	12.1	13.8	13.3
	D3	13.5	13.8	12.7	10.6	10.6	9.7	12.5	13.0	14.1
	D4	10.5	11.1	8.2	8.1	8.0	8.6	8.9	8.5	11.8
Overall period mean ± S.E.M.		13.0±0.6	11.8±0.5	10.5±0.6	10.4±0.5	10.2±0.7	11.2±0.6	11.2±0.8	11.3±0.7	12.4±0.7
Glucose	D1	57.8	62.1	63.0	53.7	50.2	51.2	46.3	57.2	49.5
	D2	61.3	66.0	61.3	59.8	61.8	58.2	58.7	61.3	60.8
	D3	63.7	53.7	58.3	52.7	51.2	47.5	48.0	46.7	50.8
	D4	55.0	55.3	51.8	54.3	51.0	48.3	40.8	47.0	49.5
Overall period mean ± S.E.M.		59.5±2.9	59.3±2.4	58.6±2.3	55.1±2.8	53.5±3.2	51.3±3.2	48.5±3.3	53.0±3.0	52.7±2.8

Table A-1.--Continued.

Serum Metabolite	Diet Group	Overall treatment means ± S.E.M.
K	D1	13.9±0.2
	D2	16.4±0.1
	D3	15.6±0.1
	D4	16.6±0.1
Overall period means ± S.E.M.		15.7±.08
Na	D1	301.7±2.9
	D2	305.0±2.8
	D3	319.5±1.9
	D4	271.4±3.5
Overall period mean ± S.E.M.		299.4±1.6
Mg	D1	1.5±.04
	D2	2.3±.04
	D3	1.3±.03
	D4	1.6±.02
Overall period mean ± S.E.M.		1.7±.02
Ca	D1	7.3±0.2
	D2	7.4±0.1
	D3	7.6±0.1
	D4	7.7±0.1
Overall period mean ± S.E.M.		7.5±.07
P	D1	5.4±0.2
	D2	4.6±0.1
	D3	4.4±0.1
	D4	4.4±0.1
Overall period mean ± S.E.M.		4.7±.07
Cr	D1	1.3±.05
	D2	1.2±.05
	D3	1.3±.06
	D4	1.2±.01
Overall period mean ± S.E.M.		1.3±.02
A.P.	D1	8.4±0.3
	D2	9.4±0.3
	D3	9.8±0.4
	D4	11.6±0.5
Overall period mean ± S.E.M.		9.8±0.2

Table A-1.--Continued.

Serum Metabolite	Diet Group	Overall treatment mean ± S.E.M.
SGPT	D1	28.8±0.5
	D2	31.3±0.4
	D3	28.8±0.5
	D4	27.7±0.4
Overall period mean ± S.E.M.		29.2±0.2
Cholesterol	D1	99.7±2.8
	D2	99.7±3.9
	D3	99.1±3.4
	D4	97.8±3.8
Overall period mean ± S.E.M.		99.1±.17
CPK	D1	203.3±5.6
	D2	209.3±6.5
	D3	179.2±4.5
	D4	153.9±4.1
Overall period mean ± S.E.M.	186.4	186.4±2.8
GOT	D1	99.0±3.7
	D2	92.3±3.4
	D3	98.6±5.9
	D4	82.9±1.9
Overall period mean ± S.E.M.	93.2 2.0	93.2±2.0
UN	D1	14.9±0.4
	D2	14.2±0.3
	D3	14.5±0.4
	D4	11.7±0.3
Overall period mean ± S.E.M.	13.8 0.2	13.8±0.2
Glucose	D1	61.5±1.5
	D2	65.4±1.9
	D3	61.1±1.6
	D4	59.7±1.4
Overall period mean ± S.E.M.		61.9±0.8

D1 = 0.37% K
D2 = 0.59% K
D3 = 0.74% K
D4 = 1.68% K

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