POTASSIUM METABOLISM IN THE OVINE

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Lloyd Darwin Campbell

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ABSTRACT

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LLOYD DARWIN CAMPBELL

Two 30-day balance trials, a 56-day feeding trial, a 56-day paired feeding trial, and an appetite trial were conducted to study the role of potassium in ovine nutrition. Western range wether lambs were used in the experiments. A semi-purified ration consisting of brewers grains, corn starch, solka floc and animal tallow was used in the balance study. A similar ration was used in the feeding trials. Levels of potassium fed were 0.7, ("low"), 2.2 ("medium") and 3.7 ("high") g per day in the balance study and 0.1, 0.3, 0.5 and 0.7% of the ration in the feeding trials.

The data suggest that the optimal ration level of potassium for fattening lambs is between 0.3 and 0.5% of the air dry ration. The 0.5% treatment exhibited an average daily gain of 0.37 lb per day in contrast to 0.06 lb per day for the 0.3% lambs. The 0.1% level resulted in a marked decrease in feed consumption, a loss of weight, listlessness, pica, damage to the kidney and a decrease in non-scoured wool potassium concentrations. A paired feeding trial revealed an effect of potassium on growth as lambs consuming a 0.7% potassium ration

gained significantly ($P < 0.05$) more weight than lambs consuming the same quantity of a 0.3% potassium ration.

Serum potassium and phosphorus concentrations of the 0.1 and 0.3% potassium lambs were significantly lower than those of the other groups. On the other hand, there were no significant differences among treatments in serum levels of sodium, calcium, magnesium or chloride. However, when lambs were pair fed the 0.3 and 0.7% potassium rations no differences in serum phosphorus levels were observed but the serum calcium concentrations of the 0.3% lambs was significantly higher than those of the 0.7% lambs.

Potassium depletion resulted in decreased potassium concentrations in skeletal and heart muscle and an increase in liver potassium. An increase in skeletal muscle sodium levels was observed while the heart muscle sodium concentration decreased.

Apparent potassium balance was significantly lower for the "low" potassium lambs than for the other treatment groups. The balance data indicate that the potassium maintenance requirement of wether lambs is less than 2.2 g per day. Level of potassium in the ration had an effect on sodium retention causing an increased urinary excretion when 0.7 g potassium per day was fed and a lower net absorption when 3.7 g potassium per day was fed.

Nitrogen balance was less positive for the "low" potassium lambs; however, level of potassium intake had no effect on apparent nitrogen digestibility. Similarly, apparent digestibilities of dry matter and energy were not affected by treatment.

The marked appetite effect observed in the lambs receiving low potassium rations did not appear to be mediated via the microorganisms since microbial activity, as measured by an *in vitro* technique, was not affected by the treatment levels of potassium.

TABLE OF CONTENTS

LIST OF TABLES

Table Page

LIST OF FIGURES

INTRODUCTION

Potassium is the major intra-cellular cation in animals and as such plays a prominant role in metabolism. It is intimately involved in enzyme systems which function in carbohydrate and protein metabolism. In addition, potassium functions in body electrolyte balance, osmosis and muscle contraction. Potassium is not stored to any great extent in the body thus a certain quantity must be fed to prevent body depletion and subsequent cellular disfunction. In fact Muntwyler *et al.* (35) have stated that:

"As long as there is a flow of urine the body apparently will continue to lose potassium unless the element is replenished by the diet."

Most natural feeds contain an abundance of potassium. However, because of the use in recent years of experimental purified rations and also all concentrate rations for beef cattle and sheep, a definition of their potassium requirement is imperative. Considerable work has been done with rats, mice, chickens and turkeys in regard to dietary potassium requirement. In contrast to this the potassium requirement of fattening lambs has not been definitely defined and there is a paucity of information concerning the metabolic role of potassium in ruminants. Consequently, it was the objective of this study to ascertain the role of potassium in ovine

nutrition and to determine the requirement of fattening lambs for this mineral element.

LITERATURE REVIEW

Role of Potassium Within the Animal Body

Potassium and sodium are the major monovalent cations found within the body and a discussion of one must automatically include the other. Potassium exists mainly in the cellular fluid while sodium is a major extra-cellular cation. The relationship between these two ions has been a focal point of interest for many years. The concept of tissue cell walls being impermeable to these cations has been disproven by isotope techniques (6). More recent theories concerning behavior of these ions include a "sodium pump" mechanism and intra-cellular binding of potassium (6, 26). In the "sodium pump" concept energy is required for active transport of sodium out of the cell and in "potassium binding" energy is also required for the formation of the poorly ionized salt, potassium glucose diphosphate. Since these cations form a major portion of the ionic equivalents within the extra- and intra-cellular phases, they perform a very import role in maintaining osmotic balance and electrical neutrality. Similarly, sodium and potassium are intimately involved in the buffering system within the body.

Metabolically, potassium functions in many enzyme systems. Among the enzymes for which potassium has been found

to be important are; phosphotransacetylase, acetyl CoA synthetase, pantothenate synthetase, pyruvate phosphokinase and myosin ATP-ase (6). The demands of osmolarity and electroneutrality, caused by a depletion of potassium, result in an increased entry of sodium into the cells. Black (6) has suggested that, since sodium is antagonistic to the potassium effect on enzyme systems, the consequences of intra-cellular depletion of potassium may be partially due to the increase in cellular sodium concentration. In vitro and in vivo studies have shown that such depletion also results in irregularities in intermediary metabolism, particularly carbohydrate and protein synthesis (43).

Utilizing in vitro experiments Fenn (20) showed that deposition of glycogen in the liver is accompanied by deposition of potassium. Further, Asford and Dixon (1) demonstrated that anaerobic glycolysis was inhibited and aerobic glycolysis stimulated by the addition of potassium to a medium of incubating rabbit brain slices. Similarly, Hastings quoted by Welt et al. (43) reported a decreased glucose uptake, increased glucose output and a decreased glycogenesis when rat liver slices were depleted of potassium. However, studies with intact animals have produced inconsistent results regarding the relationship between carbohydrate metabolism and potassium intake. Contrary to the liver slice

experiments of Hastings and coworkers potassium depletion has been shown to result in increased liver glycogen levels in rats (22). On the other hand, another report indicated glycogen was practically absent in rats deprived of potassium for 90 to 120 days (23). However, as suggested by Welt et al. (43), the apparent contradiction in these two reports regarding liver glycogen in rats may be explained by a decreased liver glycogen in the latter case mediated by starvation as poor appetite is associated with potassium deprivation.

In many of the earlier studies involving potassium metabolism a decrease in growth was found to be associated with potassium deficiency. Later studies suggest that this decreased growth rate is related to protein metabolism. Cannon et al. (11) subjected rats to a protein depletion diet until they lost 20 to 30% of their initial weight. After depletion two groups, of 10 depleted rats each, were fed a basal ration adequate in all respects except for potassium, and an amino acid solution. One group received potassium and the other no potassium. Weight gains and feed consumption were recorded, and although feed intake was quite similar, the group receiving no potassium supplementation gained 25% less weight than the group receiving potassium supplementation. This data suggests that potassium had an effect upon protein anabolism. Similar results were reported by Muntwyler et al. (35) who compared

rats consuming rations both adequate and low in potassium. Rats consuming the low potassium ration exhibited poor growth and lower nitrogen retention both before and after realimentation with protein. Muntwyler et al. (35) suggested that, since nitrogen intake of the potassium deficient rats was approximately equal to controls, there was impaired nitrogen anabolism as a consequence of potassium deficiency.

Consequences of Potassium Depletion

It is important at this point to make clear what is meant by a deficit or depletion of potassium. Scribner and Burnell cited by Welt et al. (43) developed the concept of potassium "capacity". As the potassium within the cells is intimately related to protein and glycogen the quantity of these two is the major determinant of the potassium "capacity". Consequently, a cell deficit of potassium would not become apparent if glycogen and protein were lost along with potassium. Burnell and Scribner (9) claim that at any given level of total body potassium, there is an inverse relationship between serum pH and serum potassium concentration. Thus as the manifestation of a potassium deficit is complicated by many factors it is not unreasonable that, as reported by Welt et al. (43), the correlation between a potassium deficit and its level in serum is poor when the deficit is estimated by either muscle analysis or by determination of total

exchangeable potassium, utilizing potassium 40. However, Telle et al. (41) reported a significant correlation between level of potassium in the ration and serum potassium concentrations in lambs. In addition to serum potassium changes in relation to the potassium capacity, other serum compositional changes during potassium depletion include a frequent but not invariable decrease in chloride (43), a decreased hematocrit (41) and an increase in bicarbonate (43).

Associated with potassium deficiency are anorexia and reduced rate of growth (3, 30, 34, 41). However, paired feeding experiments have shown that the slower rate of growth cannot be completely accounted for by the concomitant reduction in feed intake (11, 31). As mentioned earlier the relationship of potassium to intermediary metabolism may be the factor causing this effect.

Other deficiency symptoms in rats include roughening and thinning of the fur, a striking alertness and a peculiar pica (36). Meyer et al. (33) observed similar symptoms when rats were fed low levels of both sodium and potassium. However, when these ions were fed in ratios (Na/K) of 2, 20 and 200 to 1 the rats exhibited listlessness and diarrhea. Although Orent-Keiles and McCollum (36) were able to maintain rats on a low level of ration potassium (0.01% K) for 327 days, Meyer et al. (33) observed death within 14 days in rats

fed a high ration level of sodium (1.0% Na) and a low ration level of potassium (0.005% K). Similarly, Bell and Eirfle (3) found that mice, fed a semi-purified diet with no supplementary potassium, became emaciated and died after five - six days on test. Brink (8) observed decreased weight gains, emaciation, loss of wool, muscular stiffness and lack of appetite in sheep consuming a ration containing 0.1% potassium. Similar results were reported by Telle et al. (41), and in addition, two deaths occurred among the lambs receiving the lowest potassium ration (0.1% K).

In potassium deficiency there is movement of sodium into the cell to compensate for the decrease in potassium. However, the replacement is not complete and reports show that approximately 2/3 of the potassium lost is replaced by sodium (43). Hydrogen ions have been implicated as the other cation to make up this deficit (14, 35), and also certain basic amino acids have been suggested (19). The end result of these electrolyte shifts is extra-cellular alkalosis and impaired renal function.

A concomitant increase in sodium and decrease in potassium have been observed in skeletal and heart muscle with very little change in kidney tissue during potassium depletion (33, 35, 43). However, data concerning liver electrolyte changes during potassium depletion are not so consistent.

Most workers have reported the liver potassium content to be quite stable with no change despite significant total body potassium deficits (43). In contrast to this, Meyer et al. (33) observed a significant increase in liver potassium concentration of rats maintained on a potassium deficient diet.

Muntwyler et al. (35) reported that an important feature of potassium depletion is its continuous renal loss. A balance study conducted on a healthy 35 year-old man by Blahd and Bassett (7), substantiates this suggestion. These workers observed a negative potassium balance following the initiation of a potassium depletion trial, and as the trial progressed the urinary excretion of potassium fell to a relatively constant minimal level. This reduction in urinary potassium output was, however, not adequate to prevent loss of potassium from the body and a negative cumulative potassium balance resulted. In contrast to this observation Orent-Keiles and McCollum (36) maintained rats on a potassium deficient diet (0.01% K) for 327 days with the animals being in equilibrium as far as potassium was concerned at the end of the trial.

Muntwyler et al. (35) reported an effect of low potassium intake on the nitrogen balance of rats. A less positive balance was observed in all groups receiving low potassium. In addition, the levels of sodium and chloride in

the ration seemed to influence nitrogen utilization. The work of Devlin and Roberts (16) corroborates this sodium effect, as they reported an increased urinary nitrogen excretion by lambs fed a low sodium ration. As mentioned earlier Cannon et al. (11) also reported an adverse effect of low potassium intake on cellular nitrogen utilization.

On reviewing the literature concerned with the pathology of potassium depletion Welt et al. (43) reported that the majority of kidney lesions are of tubular origin and of two types: (1) an intra-cellular accumulation of large granules, and (2) swelling and hyperplasia of the tubular epithelium. The former lesions are found in the inner medulla and papilla and their nature is not definitely known; although Pearse and MacPherson (37) suggested that they might be altered mitochondria. The dilation of kidney tubules commonly observed in potassium deficiency is thought to be caused by the hyperplastic lesions. Telle et al. (41) observed areas of interstitial nephritis and a few hyaline mineralized casts in the kidney collecting tubules of potassium deficient lambs.

Potassium depletion has been shown to result in myocardial lesions in rats (35), mice (43), pigs (43), and cats (15) but not in sheep (41) or dogs (15). Skeletal muscle necrosis has also been reported as a consequence of potassium lack (8, 41).

It has been demonstrated that the ratio of sodium to potassium is very important in potassium deficiency. Meyer et al. (33) reported no relationship between structural changes in kidney, thymus, spleen and liver tissues of rats and the levels of sodium and potassium in the ration. On the other hand, the microscopic lesions were observed in heart tissue and they became more severe as the sodium level of the potassium deficient ration was increased. This finding was later substantiated by Cannon et al. (12). These workers divided 15 protein depleted rats into three groups: one group received a ration devoid of both sodium and potassium; a second group received supplementary sodium; and the remaining group received both added sodium and potassium. All rats fed the sodium-potassium deficient diet lived and heart lesions were observed in only two of the five rats. In group two, three of the five rats died and severe heart lesions were observed in all the rats. The third group showed optimum growth with no heart lesions developing. In a later experiment these same workers subcutaneously injected protein depleted-potassium deficient rats with 5 ml of a hypertonic sodium chloride solution (4.66%). Death of all rats was observed within 24 hours.

Potassium Requirements

Most natural food stuffs contain sufficient potassium

to meet human and other animal requirements. However, the use of purified diets in experimental work and the development of all concentrate rations for ruminants have resulted in considerable interest in the potassium requirement of various species of animals. Orent-Keiles and McCollum (36) reported that a ration containing 0.01% potassium was inadequate to maintain normal growth of rats. Similarly, Miller (34) produced greatly retarded growth rates in rats by reducing the ration level of potassium below 0.1%. Later studies by Kornberg and Endicott (31) indicated that a ration potassium level of 0.17% was adequate to uniformly prevent pathological lesions and promote normal growth in rats. A ration containing 0.2% potassium produced normal growth when fed to mice (3). Graw et al. (24), cited by Bell and Eirfle (3), indicated that the chick requires from 0.16 to 0.20% potassium depending on the stage of growth. A slightly higher requirement is displayed by the turkey. Sullivan (40) reported that a potassium ration level of at least 0.275% was necessary for survival of 50% of the turkeys tested. Du Toit et al. (18) did not notice any deficiency symptoms when cattle were fed a herbage containing 0.34% potassium. Brink (8) found that a ration containing 0.3% potassium was not adequate for the optimum growth of lambs and that a level of 0.5% was required. On

the other hand, Telle et al. (41) showed the potassium requirement of lambs for growth to be about 0.3% of the ration. Devlin and Roberts (16) suggested that the potassium requirement for maintenance of wether lambs was 1.2 g per day.

EXPERIMENTAL PROCEDURE

Experiment Ia

A 56-day feeding trial was conducted to study the potassium requirement of fattening wether lambs. Sixty-four western range lambs averaging 74.1 lb each were adjusted to a semi-purified ration consisting of the following ingredients (expressed as per cent): dried brewers grains, 66.9; corn starch, 15; solka floc, 11; animal tallow, 5; dehydrated alfalfa, 1.0; mineral-vitamin supplement, 1.1[1]. The basal ration contained 0.046, 0.15 and 0.12% potassium, sodium and chloride[2], respectively. After an initial 11-day adjustment period the lambs were randomly alloted to four groups of 16 lambs each, and were equilibrated on a 0.5% potassium ration for an additional seven days. Ration potassium levels of (expressed as a per cent of the air dry ration) 0.1, 0.3, 0.5 and 0.7% were made by adding various levels of K_2CO_3 to the basal ration. These ration treatments were randomly assigned to the four groups. At the initiation of the trial feed intake was restricted and then the quantity offered was

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[1]The mineral-vitamin supplement contained the following (expressed in g per lb): dicalcium phosphate 294.2; sodium chloride 159.8; and vitamins A and D to supply 3,000 I. U. and 500 I. U. per day, respectively.

[2]Calculated from values listed in Feeds and Feeding 22nd ed.