Factors Influencing Urinary Calculi in Lambs

Donald W. Hoar

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FACTORS INFLUENCING URINARY CALCULI IN LAMBS

BY

DONALD W. HOAR

A thesis submitted
in partial fulfillment of the requirements for the
degree Doctor of Philosophy, Major in
Animal Science, South Dakota
State University

1969

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FACTORS INFLUENCING URINARY CALCULI IN LAMBS

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/ Thesis Adviser

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principally due to an increase in urinary pH. On the other hand, sodium bicarbonate increased urine pH but did not promote stone formation in lambs fed a diet with 15% hay. Lower urinary phosphorus concentrations (0.8-24 mg./100 ml. with 15% hay diet vs. 19-99 mg./100 ml. with all-concentrate diet) appeared to be the limiting factor in calculi formation with diets containing hay.

With a level of phosphorus (0.28%) that was only slightly in excess of requirements, a low calcium to phosphorus ratio (0.5-1:1) did not promote ovine urinary calculi formation. However, when dietary phosphorus was increased to 0.47%, a calcium to phosphorus ratio greater than 2:1 appeared to be necessary for urinary calculi protection when ground limestone was used to increase dietary calcium. With the use of calcium chloride, a 1.2:1 ratio completely prevented calculi formation. An increase in dietary phosphorus generally reduced weight gains and feed consumption, and additional dietary calcium overcame these effects.

Urine and serum potassium and sodium were not related to phosphatic urolithiasis. In addition, lambs fed the calculogenic diets showed no increase in nondialyzable urinary constituents including protein, hexose and hexosamine. However, in combination with phosphatic ovine urolithiasis there were increases in urine and serum phosphorus and decreases in urine and serum calcium. Elevated levels of dietary calcium and phosphorus were reflected in higher serum values for the corresponding elements.

Experiment III involved a study of interrelationships between two levels of potassium (0.41 and 1.01%), two levels of phosphorus
(0.24 and 0.57%) and two levels of calcium (0.28 and 1.20%) on feedlot performance and phosphatic urinary calculi formation in ewe lambs. The feeding of 1% potassium chloride in a high phosphorus (0.57%) diet depressed weight gains, but additional calcium overcame this depression. While the incidence of urinary calculi was not increased by the higher level of potassium, the average size of the urinary mineral deposits was larger.
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Introduction.

The problem of bladder and urethral stone is one of the first known diseases of man. A well-formed bladder stone was found with the skeleton of a 16-year-old Egyptian boy who probably lived 7,000 years ago (Shattock, 1905). Joly (1929) has also reported that bladder stones were common in Greek and Roman Times and lithotomy, cutting for stone, was used as a preventative method.

The first reported occurrence of urinary calculi in farm animals was by Youatt in 1893 in England (Beveridge, 1942). Urinary calculi is now recognized as a major problem in the raising of livestock. Ensminger et al. (1955) reported that the urinary calculi problem, nationwide, ranked number five and accounted for 5.5% of all nutritional diseases in cattle. For the Midwest they rank it as the number two problem, accounting for 18.6% of cattle suffering from nutritional diseases. It is not only a problem in the feedlot, but on the range as well. To combat the problem in both humans and farm animals, several species of animals including cattle, sheep, cats, dogs, foxes, mice, mink and rats have been used in research work. There are no reports in the literature of a urinary calculi problem in swine and horses, although Cornelius and Bishop (1961) report an analysis of a stone from a horse.

In addition to the name urinary calculi, other terms used to describe the same problem include urolithiasis, water belly, gravel, kidney stones, bladder stones and urethral stones. Strictly speaking,
stones are a resulting condition rather than a disease.

The most positive evidence of calculi is partial or complete blockage of the urinary tract. The symptoms associated with urinary calculi have been reported by Pontius et al. (1931). Affected animals stop eating and the back becomes arched, especially when they strain to urinate. Straining causes the tail to be held away from the body, and secondary rectal prolapse is not uncommon. The animals become dull, listless, twitch their tails and, in advanced stages, show evidence of great pain. They are inclined to lie down much of the time and when they do get up, they frequently stamp their hind feet and kick at their abdomen. With partial blockage, urine slowly dribbles from sheath, and preputial hairs may contain masses of calculous material. Upon complete blockage the bladder or urethra will rupture and the animal dies of uremic poisoning. Complete urethral blockage and uremia may occur after only 2 weeks on calculi-provoking diets (Cornelius and Moulton, 1960).

As reported by Connell et al. (1959), Udall (1959) and Udall et al. (1965), peak calculi incidence in animals is present in mid-winter from October to March, usually coinciding with the onset of cold weather. In cattle, obstructive calculi occur most often in steers but are found occasionally in bulls (Connell et al., 1959). The urethra in steers and wethers, as a result of castration, is of smaller diameter, and its obstruction may be brought about by a relatively small stone which may be restricted in its passage by the S-curve or sigmoid flexure of the penis. Scratching of the urethral lining induces reflex spasm in the surrounding musculature, and these spasms may
impede and fix the stone. The susceptibility of males, as compared to females, to obstructive urolithiasis would seem to be due to differences in anatomy rather than to less frequent urolith development in females (Connell et al., 1959; Emerick and Embry, 1964). They (Connell et al., 1959) also reported that it appears to be muscular strength of the ureters that creates the pressure and ruptures the bladder in urethral obstruction, rather than kidney secreting pressure.

As reported by Connell et al. (1959), calculi in the bladder can produce obstruction in any of three ways: (1) by single uroliths attaining obstructive size, (2) by agglomeration and cementing together of numerous small uroliths or (3) by numerous small stones of subobstructive size passing into the urethra. Stones can begin forming anywhere between the renal glomerulus and the prepuce, although most obstructive calculi have their beginning at the renal papilla level.

Factors influencing calculi formation.

Among the principal factors that have been reported to influence urinary calculi, and by proper manipulation help to prevent it are: urine pH, urine volume and mineral composition of the urine and diet. These factors may act alone or in combination. In recent years, research on the mechanisms of stone formation has centered on three main aspects: (1) the relation between the concentrations of the precipitating ions in urine and the solubility of the salts formed, (2) the role of urinary proteins and mucoproteins and (3) the part played by inhibitors of crystallization.

Urine pH. Higgins (1933) working with rats, was one of the first
to show that renal calculi are partially dependent upon urine pH. This was followed by the use of urinary acidification for prevention of some types of urinary calculi in humans. Higgins (1935) and Keyser (1935) independently reported on the value of acidification in the treatment of (presumably) phosphate stones. On the other hand, Oppenheimer and Pollack (1937) reported no benefit from urinary acidification in the treatment of urinary stone, although they probably treated many patients having oxalate stone. It is also clear that pH alone is not the determining factor in stone formation. The pH of the urine of feedlot steers forming phosphate stones is alkaline but may be no different from those not forming stones (Frank et al., 1961).

Most types of calculi appear to be pH dependent, except the silicious type. Silica uroliths have been reported to be formed in both acidic and alkaline urines (Bezeau et al., 1961; Bailey et al., 1963). These workers concluded that although pH of the urine may be important in calculi formation, the incidence of silicious calculi is not a simple function of the urine pH.

The solubility of calcium phosphate and hence the formation and growth of phosphatic or apatite calculi are influenced principally by pH of the urine (Elliot et al., 1961). Therefore, most urine specimens which consistently have a urine pH above 6.6 will be saturated with calcium phosphate unless the urinary calcium excretion is less than 50 mg. per 24 hours.

Several materials have been used to change urinary pH. These include ammonium chloride, calcium chloride, magnesium chloride, sodium bicarbonate and potassium bicarbonate. The chlorides tend to acidify
the urine while the bicarbonates promote a higher urine alkalinity. Bushman et al. (1967, 1968) have shown that 1-1.5% ammonium chloride or calcium chloride lowers urine pH, thus decreasing ovine phosphatic calculi formation. In their studies, a lower level (0.5%) of calcium chloride or ammonium chloride appeared to be ineffective in calculi prevention.

Supplementing 1 gm. of ammonium chloride per mink per day was effective in preventing the formation of magnesium ammonium phosphate urinary calculi (Leoschke and Elvehjem, 1954). A lower level of one-half gm. of ammonium chloride per mink per day was ineffective. Vermeulen et al. (1951) has also shown that 1.5% ammonium chloride will prevent phosphatic calculi formation in rats. They also reported that the acidification of the urine resulted in dissolution of existing calculi in most instances.

Various acid producing substances have been used in human studies to prevent oxalate calculi (Winer, 1959). This worker used sodium acid phosphate, ammonium chloride, betaine hydrochloride or acid ash foods such as cranberry juice to achieve intermittent urine acidification. Intermittant alkalinization may be obtained by use of palatable alkalis or alkaline ash foods, with cantaloupe having the greatest effect. Zinsser et al. (1968) have also used various acidifying agents, chlormerodrin, ammonium chloride and cranberry juice, to prevent calcium citrate calculi in humans. Chlormerodrin is an antibacterial agent that diminishes activity of bacterial urease.

The feeding of ammonium chloride for calculi prevention in sheep and cattle has been cleared by the F.D.A. (1968). These
regulations permit the use of approximately 28.4–42.5 gm. of technical grade ammonium chloride per head daily for fattening cattle and 7.1 gm. for sheep. On the basis of work done at this station (Bushman et al., 1967, 1968), these levels appear to be too low for optimum effectiveness under conditions conducive to a high incidence of calculi.

For production of phosphatic calculi in rats, Vermeulen et al. (1951) fed sodium bicarbonate to increase urine alkalinity. The feeding of 1.5% ammonium chloride in a basal laboratory chow to Harlan male rats acidified the urine, and prevented the formation of magnesium ammonium phosphate stones on implanted zinc disc (Gill et al., 1959). However, 1.5% sodium bicarbonate somewhat augmented stone growth under these conditions.

In cats (Carbone, 1965), struvite calculi appeared in the urine in direct relation to the physiologic elevation of urine pH above 6.8. The struvite crystals were made to appear in the urine of a normal cat by administering the carbonic anhydrase inhibitor, acetazolamide. This compound inhibits hydrogen ion secretion in renal tubules increasing the pH to 7.0 or higher. Sodium bicarbonate has also been administered to cats to increase the formation of struvite crystals (Carbone, 1965; Rich and Kirk, 1968). Rich and Kirk (1968) stated that, even though urinary acidification decreases the amount of crystals, prolonged acidification does not prevent recurrence of urinary tract obstruction.

As stated above, an alkaline urine tends to promote phosphatic calculi formation, but this is not the case with uric acid calculi.
Vermeulen and Fried (1965) have reported that alkalinization of human urine will cause *in vivo* dissolution of uric acid calculi.

**Urine volume.** The concentration of normal urinary constituents is obviously subject to wide variations, depending upon the urine volume. In estimating the degree of saturation, the concentration of the various constituents is of greater importance than the 24-hour output.

An increase in urine volume has been used to prevent silica urolithiasis (Bailey, 1967a, b; Forman et al., 1958; Newsom et al., 1943). This increase in urine volume has been accomplished by feeding a large amount of sodium chloride. A dilution of the urinary constituents in this manner prevented the calculi producing materials from reaching their saturation point. Udall (1959) and Elam et al. (1957) have shown that the feeding of 10% sodium chloride in a pelleted diet prevented the formation of both bladder and renal calculi of the phosphatic type in sheep. Not only does sodium chloride increase water consumption, but it has also been proposed to have a specific effect of displacing magnesium and phosphorus from nucleation centers on the matrix, thereby preventing crystal development and phosphatic urolith formation (Udall et al., 1965). This mechanism of action, assigned to the chloride ion, remains unconfirmed as a factor in calculi prevention.

It has also been shown in rats that diuresis will dissolve urinary minerals previously deposited on foreign-body bladder implants (Grove et al., 1950). In this instance, diuresis was obtained by substituting a 10% glucose solution for tap water, increasing urine volume fivefold. Yet, several workers (Robbins et al., 1965a; Lindley et al.,
1953; Taysom et al., 1951) have shown that animals afflicted with calculi had larger urine volumes than those remaining free of stones. This appeared to be due to the greater water consumption associated with higher mineral intakes for the afflicted animals. Elliot et al., (1961) have shown that urinary dilution is of questionable value in reducing the formation of calcium phosphate stones because of the concomitant dilution of all other substances in urine which increase the solubility of calcium phosphate.

As reported by Prince et al. (1956), there is a definite seasonal incidence of urinary calculi in humans. The incidence of acute urolithiasis (calcium oxalate and/or calcium phosphate) appears to follow the average mean temperature with the greatest number of cases occurring from April to October and the fewest in February. They expressed the opinion that the average mean temperature existing from April to October results in dehydration and urinary concentration with resultant increases in stone formation during these months. Therefore, if the patients can be made to take large quantities of liquid to increase urine volume, particularly in the hot months, the number of cases of calculi may be reduced.

**Dietary and urinary mineral composition.** The mineral composition of the diet and urine is of utmost importance in calculi formation. To some degree, a mineral in great excess in the diet and/or urine determines the type of stone formed. However, several workers (Bailey, 1967a; Emerick et al., 1959, 1963; Forman and Sauer, 1962; Whiting et al., 1958) have shown that feeding inorganic sources of silica to animals does not influence the incidence of silicious calculi.
Prediction of the incidence of phosphatic urolithiasis in sheep has been attempted by using a multiple regression analysis based on the dietary mineral intake (Lamprecht et al., 1969a). On the basis of their analysis, the maximum phosphatic urolithiasis producing diet is one composed of a high level of phosphorus, low level of calcium and magnesium and a 0.64% potassium level. Serum values (phosphorus, calcium, magnesium and potassium) along with urine excretion values for 24-hour volume, specific gravity, pH, titratable acidity and total calcium, magnesium, phosphorus and potassium have been used to predict urolithiasis with about 75% accuracy under limited experimental conditions (Lamprecht et al., 1969b).

Because of the specificity of diet-urine-calculi relationship, this subject as it pertains to phosphatic calculi is discussed in detail under the heading "phosphatic urinary calculi".

Miscellaneous. Several other factors influence calculi formation, but they may be of limited or doubtful importance. Vermeulen et al. (1955) express the belief that three basic factors are essential for stones to form: (1) precipitation of crystalloids, (2) retention of crystalloids and (3) presence of matrix or binding substance. Other factors, as stated previously, have been implicated in the control of conditions for precipitation. Butt and Hauser (1952) and Butt (1952) concluded that the urine of humans prone to stone formation was deficient in protective colloids, and that this was the main factor in stone formation. They report that urinary colloidal activity of Negroes is high compared to that of the white race, and the former is less prone to stone formation. Work with hamsters has shown that
sequestering of urinary stone salts in a quasi-colloidal form is adverse to stone formation (Vermeulen, et al., 1960). On the other hand, growth of stones in vitro indicated that there was no participation or facilitating influence of normal urinary colloids in stone growth (King and Boyce, 1963).

All stones appear to have an organic matrix composed principally of mucosubstances and distributed from the center to surface of calculi (Boyce and King, 1963). The matrix consists of both amorphous and regularly arranged material in the form of laminations (Finlayson et al., 1961). In fully crystallized calculi the matrix accounts for 2.5% of the dry weight, but in "matrix" calculi the comparable average is 65% with a range of 42-84% of the dry weight (Boyce and King, 1959).

A comparison of the crystalline and matrix structures suggested that the size, shape, laminations, radial striations and color of calculi are primarily determined by the mucoprotein matrix (Boyce et al., 1958; Boyce and King, 1959). This matrix is formed by aggregation and molecular orientation of uromucoid. The mechanism of this molecular orientation is unknown but is intimately associated with binding of calcium, phosphate and water (hydroxyl) ions. Therefore, they concluded that the organic matrix material is a prerequisite to concretion formation, and crystal deposition is a secondary phenomenon. On the other hand, Finlayson et al. (1961) have suggested that stones form by coprecipitation of conjugated proteins and inorganic salts. When precipitation occurs in urine, protein is invariably carried down with the precipitate as a co-precipitate. They considered stone formation to be an ex-solution of stone salt plus matrix plus time for
growth of incipient stone. Therefore, the amount of matrix deposited in a stone would depend on the concentration of the co-precipitable material and the affinity of the material for stone crystals. Cornelius et al. (1965) and Packett and Coburn (1965) have suggested that the bulk of urinary mucoprotein in mammals originates from renal biosynthesis rather than from the serum.

Several workers have shown that urinary mucoprotein values are elevated in animals fed calculi-provoking diets (Packett and Coburn, 1965; Udall et al., 1958; Sulkin and Boyce, 1956; Cornelius et al., 1959; Boyce et al., 1954; Cornelius and Koulton, 1960). Bailey (1968) has also reported that the concentration of nondialysable urinary solids was much greater in cows fed prairie hay, which predisposes to silica urolithiasis, than in cows fed alfalfa hay. However, only two animals were used in this experiment, and calculi were not actually formed. Other reports, principally those by Fried and Vermeulen (1964), Packett and Coburn (1965) and Vermeulen et al. (1965), point out that nondialyzable organic constituents of urine probably play only a secondary role in urinary calculi formation, and that stone formation is fundamentally a process of crystallization rather than mineralization of an organic matrix.

Diethylstilbestrol has also been implicated as a cause of urinary calculi. Early work has shown that this estrogenic hormone, which is largely used to increase weight gains in feedlot cattle and lambs, will increase the incidence of stone formation (Bell et al., 1954; Wilkinson et al., 1955; Marsh, 1961; Udall and Jensen, 1958; Udall et al., 1965). With the use of diethylstilbestrol, hyperplasia of
the prostate and/or other accessory sex organs could well contribute to mucoid secretions and a diminished size of the urethral lumen making an animal more prone to calculi formation. In most of these reports diethylstilbestrol was implanted in wethers at the rate of 15-30 mg. These are much higher levels than those now recommended (3 mg. as an implant or 2 mg. daily in the feed). An increase in obstructive urolithiasis in lambs without any increase in nonobstructive cases was reported by Udall and Jensen (1958) to accompany the use of 30 mg. diethylstilbestrol implants. Emerick and Embry (1964) have reported that diethylstilbestrol added at currently recommended levels, 3 mg. implant or 2 mg. per lamb daily in the diet, has no influence on the incidence of calculi. McDonald and Eddings (1957) reported that oral administration of low levels of diethylstilbestrol reduced urinary calculi development in male rats.

Feeding 22 mg. of chlortetracycline per kg. of diet to sheep reduced the incidence of phosphatic urinary calculi (Packett et al., 1958). They postulated that the antibiotic tended to reduce minor kidney infections, thus reducing potential nuclei for calculi formation, and/or it might reduce bacterial urease production in the kidney, thereby preventing a calculogenic increase in urinary pH.

Several workers (Bassett et al., 1946; Higgins, 1933, 1951; Schmidt, 1941; Eveleth, et al., 1948; Cordonnier and Miller, 1951) have implicated vitamin A deficiency as a cause of urinary calculi. An increase in susceptibility to infections and changes in epithelial tissue, including the lining of the urinary tract, during periods of a vitamin A deficiency provide a basis for this. However, experiments designed.
to determine if vitamin A deficiency is a specific causative factor, have largely yielded negative results (Limley et al., 1953; Beeson et al., 1943; Whiting et al., 1958; Swingle and Marsh, 1956; Taysom et al., 1951).

Losses from urinary calculi generally involve only male or castrated male animals. However, Emerick and Embry (1964) and Udall (1959) have shown that while no losses occurred in ewes from urinary tract blockage, several were found to have urinary calculi at slaughter. Overall, there was no significant difference between ewes and wethers in percent incidence of urinary calculi found at time of slaughter. On the other hand, only the females of the white Wistar rat strain developed calculi, and this was believed to be due to differences in estrogen levels (Cousins and Geary, 1966). Deferred castration has also been shown to lower the incidence of calculi in cattle due to a slightly larger urethra diameter (Marsh and Safford, 1956).

A dietary vitamin B<sub>6</sub> deficiency has been shown to increase the incidence of calcium oxalate calculi in rats (Gershoff et al., 1959; Andrus et al., 1960; Gershoff and Andrus, 1961; Faragalla and Gershoff, 1963). Occurrence of this type of calculi, resulting from a vitamin B<sub>6</sub> deficiency, was shown to be reduced by feeding high levels of magnesium. Oxalate calculi are generally encountered in rats, cats and humans and are not a problem in large farm animals.

Phosphatic urinary calculi.

The type of calculi that will be discussed in detail is the phosphatic type, this being the type studied in sheep, as reported herein. Yet, several other types of calculi exist in both humans
and/or other animals. These types include calculi composed principally of silica, oxalate, uric acid, urates, citrates, xanthine, cystine, fibrin or cholesterol. Some aspects of certain of these types of calculi were discussed in previous sections.

The development of urinary calculi composed principally of phosphates is a problem most often associated with sheep and cattle in the feedlot as opposed to the silicious calculi that commonly develop in animals maintained under range conditions. The phosphatic type of calculi, also known as struvite (magnesium ammonium phosphate) and apatite (basic calcium phosphate), is composed of the mineral components plus an organic matrix (Romanowski, 1965). The solubility of calcium phosphate in aqueous solutions is markedly reduced by a rise in pH and apatite will not crystallize from urine below a pH of about 6.6 and struvite will not crystallize below a pH of 7.1 (Elliot, 1968).

As early as 1951, high dietary phosphorus supplementation was recognized to cause a high incidence of calculi (Taysom et al., 1951; Schneider et al., 1952). Haag and Palmer (1928) found phosphatic calculi in rats receiving diets low in calcium carbonate and high in magnesium carbonate and phosphoric acid, but they pointed to high magnesium as the causative factor. Renal damage and calcification in rats has also been observed following the ingestion of a diet containing an excess of inorganic phosphate (MacKay and Oliver, 1935).

It has been well documented by many workers (Lindley et al., 1953; Emerick et al., 1959; Emerick and Embry, 1963; Packett and Hauschild, 1964; Robbins et al., 1965a; Bushman et al., 1965a, b) that high dietary phosphorus, with consequently high urinary phosphorus levels,
is one of the principal causative factors in phosphatic urinary calculi formation. The feeding of various phosphates, monosodium phosphate, disodium phosphate or sodium tripolyphosphate, but not dicalcium phosphate, has been shown to promote calculi formation (Bushman et al., 1965a). However, phosphoric acid fed under conditions that result in an acidic urine does not necessarily contribute to calculi formation (Elam et al., 1956; Crookshank et al., 1960).

Feeding a pelleted diet (0.18% calcium and 0.71% phosphorus), with a high ratio of concentrate to roughage (4:1) and containing 2.5% of dipotassium phosphate, consistently produced a high incidence of urolithiasis in yearling wethers (Cornelius et al., 1959). Pelleting a known calculogenic diet has increased the incidence of calculi in wether lambs. This increase in calculi appeared to occur in response to higher urine phosphorus levels (Crookshank et al., 1965; Packett et al., 1958). Slinger et al. (1966) has shown that pelleting enhances the availability of plant phosphorus for chicks.

Additional dietary calcium has been shown to depress the incidence of phosphatic urinary calculi, but generally it has not completely prevented the problem (Emerick and Embry, 1963; Robbins et al., 1965a; Bushman et al., 1965a, b; Packett et al., 1968). This reduction in calculi incidence is probably caused by the precipitation of calcium phosphates in the G.I. tract, thus less phosphorus is available for absorption and subsequent urinary excretion (Gill et al., 1959).

"Therefore, in addition to the absolute amount of dietary phosphorus present, the dietary calcium to phosphorus ratio is of importance in determining whether stones will form. With a 1.3-2.9:1 dietary
calcium to phosphorus ratio no calculi developed in lambs, but a 31-73% calculi incidence occurred when the ratio was 0.5-0.7:1 (Emerick and Embry, 1963). Robbins et al. (1965a) also reported that the occurrence of phosphatic urolithiasis in lambs tended to increase as the ratio of calcium to phosphorus decreased. With a 5:1 ratio there was a low incidence of calculi, but when the ratio was changed to 0.3:1, calculi incidence was greatly increased. Studies by Bushman et al. (1965b) have shown that the optimal calcium to phosphorus ratio appeared to be 2:1 or greater with a 0.55% dietary phosphorus level.

Magnesium has also been implicated in urinary calculi formation (Crookshank et al., 1967; Packett et al., 1968). Crookshank et al. (1967) postulated that the development of phosphatic urolithiasis may be associated with renal retention of magnesium. However, Bushman et al. (1965b) found increased magnesium retention to be a normal response to increases in serum phosphorus levels. While both serum and urine magnesium levels have been found to be relatively high in lambs predisposed to calculi (Kunkel et al., 1961; Packett and Hauschild, 1964; Packett et al., 1968), Bushman et al. (1965b) found supplemental dietary magnesium to offer some degree of protection. They (Bushman et al., 1965b) concluded that although dietary magnesium was more effective than an equal amount of calcium in reducing urinary phosphorus, it was no more effective in reducing the incidence of urolithiasis.

Various salts have been used to provide protection against calculi formation. Dietary ammonium chloride, as reported under the heading
"urine pH", lowers the incidence of urinary calculi. In addition, other ammonium compounds including ammonium sulfate and a commercial preparation of an ammonium polyphosphate solution offer some protection (Crookshank, 1969). However, the feeding of diammonium phosphate produced a nonsignificant increase in the incidence of calculi.

The addition of potassium chloride to the diet has yielded conflicting results. Crookshank (1966) reported 1% potassium chloride to be protective against calculi formation and concluded that potassium salts offered more protection than corresponding sodium salts. However, Bushman et al. (1968) reported that 1% potassium chloride added to a high phosphorus calculogenic sheep diet increased the incidence of urinary calculi and depressed weight gains. Recently, Lamprecht et al. (1969a) have postulated that potassium has a curvilinear effect on the incidence of calculi.
I. OVINE PHOSPHATIC UROLITHIASIS AS RELATED TO THE PHOSPHORUS AND CALCIUM CONTENTS AND ACID-BASE-FORMING EFFECTS OF ALL-CONCENTRATE DIETS

A high urinary phosphorus level has been previously delineated as an important measurable factor contributing to the formation of phosphatic urinary calculi (Emerick et al., 1959; Packett and Haushild, 1964; Robbins et al., 1965a; Bushman et al., 1965a, b). The addition of elevated levels of calcium to high-phosphorus, calculogenic diets has been shown to lower serum and urine phosphorus concentrations and reduce the incidence of phosphatic urolithiasis in lambs (Emerick and Embry, 1963, 1964; Bushman et al., 1965a, b). Whether calculi may result from feeding calcium at levels that are low in relation to phosphorus when phosphorus approximates recommended levels (N.R.C., 1964) has not been determined.

Phosphatic urinary calculi are generally recognized to undergo most rapid formation in an alkaline urine, and the feeding of acid-forming salts including ammonium chloride and calcium chloride has been utilized for urinary calculi prevention (Crookshank et al., 1960; Bushman et al., 1967, 1968). Several studies (Wise et al., 1961, 1965; Nicholson et al., 1963; Oltjen et al., 1965) conducted with cattle have involved feeding alkaline buffers, presumably to increase feed consumption and/or utilization of high- or all-concentrate diets. While kidney lesions have been reported in some instances (Nicholson and Cunningham, 1961; Nicholson et al., 1962), little attention appears to have been given to the potential calculogenic effects of these diets. The research described herein was conducted to determine the relative
importance of low dietary calcium, elevated dietary phosphorus and alkali-forming effects of the diet to urinary calculi formation in lambs fed an all-concentrate diet.

Experimental

This experiment utilized 240 crossbred wether lambs averaging 28.1 kg. and was conducted over a 101-day period during the summer. The experimental design was a 2 x 2 x 2 factorial involving two levels of sodium bicarbonate (0 and 2%), two levels of calcium (0.14 and 0.28%) and two levels of phosphorus (0.28 and 0.55%). The eight treatments were replicated three times with 10 lambs per lot, initially. The lambs were allotted on the basis of weight. Prior to being placed on experiment, the lambs were vaccinated for prevention of enterotoxemia, drenched with thiabendazole and implanted with 3 mg. diethylstilbestrol. This level of diethylstilbestrol has been shown to be without effect on urolithiasis in previous studies (Emerick and Embry, 1964).

The experimental diets (table 1) consisted principally of ground shelled corn and soybean meal, and contained an average of 12.9% crude protein ($N \times 6.25$) and 0.13% magnesium by analysis. During a preexperimental period of 2 weeks, the lambs were fed chopped alfalfa hay ad libitum and 0.23 kg. of ground shelled corn per lamb daily. At the start of the experiment, they were fed 0.23 kg. of the experimental diets plus 0.46 kg. of chopped alfalfa hay daily and were converted to a full feed of the all-concentrate experimental diets over a 10-day period. Thereafter, they were fed once daily in amounts so feed would be available at all times with water being given ad libitum.

For mineral analysis, feed samples were treated with concentrated
<table>
<thead>
<tr>
<th>Component</th>
<th>Control series</th>
<th>2% Sodium bicarbonate series</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium, %</td>
<td>0.14 0.28</td>
<td>0.14 0.28</td>
</tr>
<tr>
<td>Phosphorus, %</td>
<td>0.28 0.28</td>
<td>0.28 0.28</td>
</tr>
<tr>
<td>Ground shelled corn, %</td>
<td>89.23 88.84</td>
<td>87.23 86.84</td>
</tr>
<tr>
<td>Soybean meal, %</td>
<td>10.00 10.00</td>
<td>10.00 10.00</td>
</tr>
<tr>
<td>Trace mineral salt, %</td>
<td>0.50 0.50</td>
<td>0.50 0.50</td>
</tr>
<tr>
<td>Ground limestone, %</td>
<td>0.27 0.66</td>
<td>0.27 0.66</td>
</tr>
<tr>
<td>Disodium phosphate, %</td>
<td>- - 1.38</td>
<td>- - 1.38</td>
</tr>
<tr>
<td>Sodium bicarbonate, %</td>
<td>- - -</td>
<td>2.00 2.00</td>
</tr>
<tr>
<td>Vitamin A, %</td>
<td>+ + +</td>
<td>+ + +</td>
</tr>
</tbody>
</table>

*a* Air-dry basis.

*b* Calcium and phosphorus values are based on analyzed values for the basal diet (0.14% Ca and 0.28% P) with adjustments being made in ground limestone and disodium phosphate to obtain the higher level of each.

*c* 44% crude protein.

*d* Anhydrous Na$_2$HPO$_4$ furnished by Stauffer Chemical Co., Victor Chemical Division, New York, N.Y.

*e* Vitamin A, 2200 I.U. per kg. of diet.
nitric acid prior to being ashed at 600°C in a muffle furnace. Analyses were performed on the acid-soluble ash. Calcium and magnesium were determined by atomic absorption spectroscopy in the presence of 0.5% (w/v) lanthanum. Phosphorus was determined by the A.O.A.C. (1965) method.

A blood sample was obtained by jugular vein puncture from each lamb after 21 days on experiment, and the serum was stored frozen until analyzed. A 24-hour urine sample collected under toluene was obtained from one lamb per lot daily until samples had been obtained from all lambs surviving at the time collections were made. These collections were made in steel metabolism cages during the period 41-59 days after initiation of the experiment. An additional blood sample was obtained from each lamb at the time they were placed in the metabolism cages. Serum and urine calcium and magnesium were determined by atomic absorption spectroscopy in the presence of 0.5% lanthanum, and serum and urine phosphorus were determined by the method of Fiske and Subbarow (Hawk et al., 1954).

The pH values of the 24-hour urine collections were determined using a Beckman Zeromatic pH meter. The volume of the urine was recorded, concentrated hydrochloric acid was added at the rate of 2% (v/v) and the acidified urine was filtered through multiple layers of cheese cloth. An aliquot was stored frozen for later inorganic analyses. Another sample (10 ml.) was immediately placed in 1.6 cm. dialysis tubing and dialyzed at 7°C for 5 days against a continuous change of deionized water. After dialysis, the solutions were made slightly alkaline with 3N sodium hydroxide and were stored frozen until
analyzed. For analysis, the samples were thawed, clarified by centrifugation, and protein was determined by the Folin-Ciocalteu method (Layne, 1957) and hexose by the anthrone reaction method of Tuller and Keiding (1954). Hexosamine was determined by the method of Rondle and Morgan (1955) following hydrolysis with 4N hydrochloric acid in a boiling water bath for 4 hours.

Lambs developing urinary tract blockage were slaughtered during the course of the experiment. Those remaining were slaughtered after 101 days on experiment, and the bladders and kidneys of all lambs were examined for calculi. All calculi were characterized according to type.

Average daily gain and feed consumption data were calculated only for those lambs finishing the experiment. Feed consumption data were corrected for death losses by subtracting an average value up to that time.

Statistical analysis of urinary calculi incidence was made by the Chi-square method with all other data being analyzed by the least squares method (Steel and Torrie, 1960).

Results and Discussion

Data from this experiment are presented in table 2. No apparent problems were encountered in getting the lambs to consume the all-concentrate diets. However, maximum consumption was not attained until after 3 weeks. Average daily feed consumption for all treatments was in the range of 1.06 to 1.13 kg. per lamb with an average daily gain of 0.211 kg.

In a comparison of lambs fed the two levels of calcium (0.14 vs. 0.28%), those receiving the higher level made slightly higher (P < .1)
<table>
<thead>
<tr>
<th></th>
<th>Control series</th>
<th>2% Sodium bicarbonate series</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.14 0.28 0.14 0.28</td>
<td>0.14 0.28 0.14 0.28</td>
</tr>
<tr>
<td>Calcium, %</td>
<td>0.28 0.28 0.55 0.55</td>
<td>0.28 0.28 0.55 0.55</td>
</tr>
<tr>
<td>Phosphorus, %</td>
<td>0.14 0.28 0.14 0.28</td>
<td>0.14 0.28 0.14 0.28</td>
</tr>
<tr>
<td>No. of lambs b</td>
<td>30 29 28 27</td>
<td>30 29 29 29</td>
</tr>
<tr>
<td>Av. daily gain, kg.</td>
<td>0.211 0.226 0.195 0.206</td>
<td>0.217 0.225 0.204 0.204</td>
</tr>
<tr>
<td>Kg. feed per kg. gain</td>
<td>1.12 1.16 1.08 1.15</td>
<td>1.16 1.18 1.06 1.14</td>
</tr>
<tr>
<td>Carcass grade</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum values, mg./100 ml.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>10.1 10.0 9.6 9.5</td>
<td>9.6 9.8 9.2 9.4</td>
</tr>
<tr>
<td>Magnesium</td>
<td>3.0 2.9 2.7 2.8</td>
<td>2.9 2.9 2.8 2.9</td>
</tr>
<tr>
<td>Phosphorus e</td>
<td>7.8 7.6 8.7 8.5</td>
<td>8.7 8.0 9.8 9.2</td>
</tr>
<tr>
<td>41-59 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>9.8 10.2 8.8 9.6</td>
<td>9.9 10.2 8.7 9.7</td>
</tr>
<tr>
<td>Magnesium</td>
<td>4.0 3.7 3.7 3.7</td>
<td>3.6 3.6 3.9 3.8</td>
</tr>
<tr>
<td>Phosphorus e</td>
<td>8.6 8.0 9.8 9.1</td>
<td>8.0 7.9 9.8 9.2</td>
</tr>
<tr>
<td>Urine data</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume, ml./24 hr.</td>
<td>574 610 859 884</td>
<td>737 750 1017 1046</td>
</tr>
<tr>
<td>Inorganic values, mg./100 ml.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>3.3 3.5 1.8 1.8</td>
<td>2.2 1.8 1.4 1.3</td>
</tr>
<tr>
<td>Magnesium</td>
<td>56 39 41 56</td>
<td>48 29 26 26</td>
</tr>
<tr>
<td>Phosphorus f</td>
<td>19 34 93 99</td>
<td>20 21 65 70</td>
</tr>
<tr>
<td>Non-dialyzable organic values, mg./ml.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>644 709 440 512</td>
<td>543 436 376 372</td>
</tr>
<tr>
<td>Nuclease</td>
<td>123 129 87 81</td>
<td>91 93 67 68</td>
</tr>
<tr>
<td>Hexosamine</td>
<td>137 146 97 96</td>
<td>104 110 81 85</td>
</tr>
<tr>
<td>Urinary calculi incidence, % of animals</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obstructive</td>
<td>4 1 23 16</td>
<td>0 1 19 27</td>
</tr>
<tr>
<td>Total</td>
<td>4 1 24 23</td>
<td>0 1 19 27</td>
</tr>
</tbody>
</table>

---

a Data obtained during a 101-day experimental period, June 1 - September 9.
b Originally 30 lambs per treatment, but some were lost from causes apparently unrelated to treatment.
c Choice = 11, -rize = 14.
d Calcium effect was significant (P < .05).
e Sodium bicarbonate effect was significant (P < .05).
f Phosphorus effect was significant (P < .01).
g Phosphorus effect was significant (P < .05).
h Sodium bicarbonate effect was significant (P < .01).
i Includes losses due to urinary obstruction and animals found to have urinary mineral deposits at slaughter.
j Phosphorus and sodium bicarbonate effect were significant (P < .005).
average daily gains (0.207 vs. 0.215 kg.), and had small but significant (P < .05) increases in carcass grades. Water available to the lambs provided calcium equivalent to 0.02 to 0.03% of the diet. Even with the additional calcium provided by the water, the lower level of calcium intake was below the 0.18 to 0.23% calcium indicated by the N.R.C. (1964) to be required by lambs in the 27 to 45 kg. category.

In a comparison between phosphorus levels, the higher level (0.55%) appeared to reduce weight gains and feed consumption, but the differences were not significant. The feeding of a similar level of dietary phosphorus to lambs was reported by Bushman et al. (1965b) to significantly depress weight gains. Sodium bicarbonate had no apparent effect on feed consumption or weight gain.

Increasing the phosphorus level from 0.28 to 0.55% or including 2% of sodium bicarbonate in the diet resulted in significant (P < .005) increases in the incidence of urinary calculi. While levels of calcium above 0.3% have been shown to provide some protection against the calculogenic effects of high-phosphorus diets (Emerick and Embry, 1963; Bushman et al., 1965a), reducing the calcium level from 0.28 to 0.14% in the current trial resulted in no increase in calculi.

Combining data from groups of lambs fed the two calcium levels, those fed 0.28% phosphorus without sodium bicarbonate had an average calculi incidence of only 8%. All cases in this instance involved non-obstructive calculi disclosed at the time of slaughter. In corresponding low-phosphorus groups fed 2% sodium bicarbonate, a 58% average calculi incidence was observed with only one obstructive case occurring during the course of the experiment. Lambs fed the higher level of
phosphorus (0.55%) without sodium bicarbonate had a total calculi incidence of 8.5%. Obstructive cases alone accounted for only a 9% incidence. The high incidence of calculi attributed to feeding 0.55% phosphorus tended to preclude a further increase for the corresponding groups fed sodium bicarbonate where an 88% total incidence was observed. However, the incidence of obstructive cases rose to 22% in this instance. The higher incidence of obstructive urinary calculi in lambs fed sodium bicarbonate was undoubtedly related to the larger size of the individual deposits in these lambs. Urinary mineral deposits recovered averaged 102 and 34 mg. from lambs fed diets with and without sodium bicarbonate, respectively. In all instances the calculi were the phosphatic type and contained both calcium and magnesium.

Higher serum (P < .05) and urine (P < .01) phosphorus concentrations and a larger (P < .01) 24-hour excretion of urinary phosphorus were associated with the feeding of the higher level of phosphorus. These lambs also had lower serum calcium concentrations after 21 (P < .01) and 41-59 (P < .05) days, respectively. This was associated with a lower (P < .01) urinary calcium concentration and a sodium bicarbonate x phosphorus interaction (P < .01) for urinary calcium values. However, due to differences in urine volume, the effect of dietary phosphorus in reducing urinary calcium excretion was less apparent in the 24-hour excretion values. Bushman et al. (1967) have previously concluded that variations in urinary calcium excretion appear to play no major role in phosphatic urinary calculi formation.

Lambs fed diets with 2% sodium bicarbonate had lower (P < .05) serum calcium values than corresponding non-bicarbonate treatment
groups at 21 days, but not at the later sampling period. However, one of the more obvious effects of sodium bicarbonate feeding was an alka­linization of the urine, average urine pH values being 8.4 and 7.8 (P < .01) for lambs with and without sodium bicarbonate, respectively. For all groups, average pH of the urine greatly exceeded the values of 6.6 to 6.8 above which magnesium and calcium phosphates have been shown to precipitate from urine (Elliot et al., 1961; Carbone, 1965).

When compared with lambs fed the higher calcium level, those fed 0.14% calcium had lower (P < .1) serum calcium values at the 41-59 day sampling period. This effect appeared to be the result of a decline in serum calcium during the course of the experiment for these lambs, this being further evidence of the inadequacy of the 0.14% calcium level used in this study. There was no apparent effect of variations in dietary calcium on serum phosphorus concentrations. However, feeding the higher level of calcium resulted in a larger (P < .05) 24-hour excretion of phosphorus via the urine. The effect of the higher calcium level on phosphorus excretion, in this instance, is opposite the effect previously demonstrated with levels of calcium above those required for optimum performance (Bushman et al., 1965b), and is possibly due largely to the greater feed intake and slightly larger urine volume associated with the higher calcium treatments.

Average urinary phosphorus concentrations observed for groups of lambs fed the lower level of phosphorus were in the range of 19 to 34 mg. per 100 ml., and corresponding values for those fed the higher phosphorus levels were 65 to 99 mg. per 100 ml. These values obtained with lambs fed an all-concentrate diet are notably higher than those
observed in some previous studies (Bushman et al., 1965a, b, 1967, 1968), where roughage was incorporated into diets with similar levels of phosphorus. Further, the incidence of urinary calculi (av., 85%) associated with use of the 0.55% phosphorus diet, as reported herein, is higher than the approximate 50% incidence observed with lambs fed similar phosphorus and calcium levels in the studies previously cited.

Serum magnesium values for samples collected at either 21 or 41-59 days were not influenced by any of the treatments, but they averaged about 0.9 mg. per 100 ml. higher for the second set of samples. Urine magnesium concentration was significantly (P < .05) decreased by the higher level of dietary phosphorus or by including sodium bicarbonate in the diet. In both instances, the lower concentration of magnesium appeared to be due to larger urine volumes. The total amounts of magnesium excreted in 24 hours did not differ between treatments.

The total amounts of various nondialyzable urinary constituents, including protein, hexose and hexosamine, were relatively constant for lambs on all treatments. However, concentrations of some of these appeared to be inversely related to urine volume which was increased by the higher level of calcium (P < .05) and phosphorus (P < .1) and tended to be increased by sodium bicarbonate (nonsignificant). An exception is the slightly but significantly (P < .05) higher total urinary excretion of nondialyzable hexose that accompanied the feeding of the higher level of calcium. Because of the inverse relationship with urine volume, concentrations of these organic urinary constituents tended to be lowest in groups having the highest incidence of urinary
calculi. Although similar organic fractions are normally found in urine and in matrices of urinary calculi (Cornelius, 1963), no conclusive evidence has been reported showing a diet to be calculogenic by virtue of its influence on excretion of these materials. The observation reported above, that concentrations of nondialyzable organic constituents tended to be lowest in groups of lambs having the highest incidence of urinary calculi, supports the conclusion reached previously (Packett and Coburn, 1965; Vermeulen et al., 1965) that these materials probably play only a secondary role in urinary calculi formation. Finlayson et al. (1961) have aptly proposed co-precipitation as the method for incorporation of urinary proteins into calculi.

The most important, if not the only, calculogenic urinary deviations associated with the feeding of sodium bicarbonate or elevated levels of dietary phosphorus to lambs in this experiment appear to be an increase in urine alkalinity by the former and an increase in urinary phosphorus concentrations by the latter. While either condition appeared to promote phosphatic urinary calculi under the conditions described herein, Bushman et al. (1967, 1968) have shown that the calculogenic effect of high urinary phosphorus concentrations in sheep can be overcome by the feeding of acid-forming salts producing relatively small decreases in pH of the urine. This may indicate an interdependence between these two factors, i.e., urinary phosphorus concentrations and urine alkalinity, in the formation of phosphatic urinary calculi. However, minimum urinary phosphorus concentrations required for the formation of phosphatic urinary calculi in lambs fed diets having various acid-, alkali-forming capacities have not been defined. High urinary phosphorus
levels, whether promoted by high dietary phosphorus levels or by factors increasing phosphorus availability, and alkali-forming effects of the diet continue to be the most important factors elucidated thus far as causes of phosphatic urinary calculi.

Summary

A 2 x 2 x 2 factorially designed experiment utilizing 240 wether lambs was conducted to determine the relative importance of a suboptimum level of calcium, an elevated level of dietary phosphorus and an alkali-forming effect of the diet in promoting phosphatic urinary calculi. Treatment variables incorporated into an all-concentrate, corn-soybean meal diet included 0.28 and 0.55% phosphorus, 0.14 and 0.28% calcium and 0 and 2% sodium bicarbonate.

With a level of phosphorus (0.28%) that is only slightly in excess of requirements, a low calcium to phosphorus ratio (0.5-1:1) did not promote urinary calculi. Variations in phosphorus or sodium bicarbonate did not significantly affect feed consumption or weight gains, but the higher level of each gave a high incidence of urinary calculi. With the feeding of low (0.28%) and high (0.55%) levels of phosphorus without sodium bicarbonate, 8 and 85%, respectively, of the lambs developed urinary calculi. With the inclusion of sodium bicarbonate in the diet, 58 and 88% of lambs in corresponding groups were afflicted.

The calculogenic variables, including sodium bicarbonate and an increase in dietary phosphorus, had the principal effects of increasing urine alkalinity by the former and serum and urinary phosphorus concentrations by the latter. Lambs on the calculogenic treatments showed
no increase in nondialyzable urinary constituents including protein, hexose and hexosamine.
II. INFLUENCE OF VARIATIONS IN CALCIUM SOURCE, PHOSPHORUS LEVEL AND ACID-BASE-FORMING EFFECTS OF THE DIET ON FEEDLOT PERFORMANCE AND URINARY CALCULI FORMATION IN LAMBS

A previous study (Experiment I) has shown that sodium bicarbonate added to an all-concentrate diet promotes phosphatic urinary calculi. It has also been well documented that an elevated dietary phosphorus intake results in a concomitant increase in urinary phosphorus excretion and urinary calculi formation (Elam et al., 1956; Emerick et al., 1959; Bushman et al., 1965a, b; Robbins et al., 1965a). On the other hand, calcium carbonate, calcium chloride and ammonium chloride have been successfully used to reduce the incidence of phosphatic urinary calculi (Bushman et al., 1967, 1968). These workers have shown that calcium chloride gives more protection against calculi formation than is provided by calcium carbonate. Calcium carbonate is believed to exert its effect through a reduction in urinary phosphorus excretion. The effect of calcium chloride in reducing phosphatic urolithiasis appears to be due, in part, to a reduction in urine pH.

This experiment was conducted to compare the effects of calcium carbonate (ground limestone) and calcium chloride on feedlot performance and phosphatic urolithiasis in lambs fed diets contributing to elevated urinary phosphorus concentrations and/or urine alkalinity.

Experimental

An experiment utilizing 360 Texas lambs of mixed breeding was conducted during the summer months over a period of 87 days. The lambs, averaging 30.5 kg., were randomly allotted according to weight to 36 pens with six wethers and four ewes per pen. Each treatment was
replicated three times. During a 15-day preexperimental period, they were fed ground shelled corn and corn silage in a ratio of 1:10 (as-fed basis). During this period, the lambs were vaccinated for prevention of enterotoxemia, drenched with thiabendazole and implanted with 3 mg. diethylstilbestrol. At the start of the experiment, the experimental diets were substituted for the ground shelled corn and were subsequently increased, with gradual elimination of the silage, to provide a full feed of the experimental diets within 5 days. After this time, the lambs were fed once daily in amounts so that feed was available at all times. Water was available ad libitum.

The basal diet, presented in table 3, was shown by analysis to contain 12.0% protein (N x 6.25), 0.22% phosphorus, 0.31% calcium, 0.15% magnesium, 0.27% potassium and 0.21% sodium. The mineral variables comprising the experimental treatments are shown in table 4. The lower level of each mineral was inherent in the basal diet. The higher level of phosphorus (0.47%) was achieved by adding 1.15% disodium phosphate (anhydrous). Ground limestone (0.64%) or calcium chloride (0.92%) was added to the basal diet to provide diets containing 0.56% calcium. In addition, ground limestone (1.92%) was used to provide diets with 1.06% calcium, but calcium chloride was not used at the higher level because of possible palatability problems. All mineral additions were made at the expense of corn.

After the lambs had been on the experimental diets for 26-27 days, a blood sample was obtained by jugular vein puncture. A blood sample was also obtained after 84 days. Serum was collected and stored frozen until analyzed.
### TABLE 3. COMPOSITION OF BASAL DIET - II^a^  

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ground shelled corn, %</td>
<td>81.5</td>
</tr>
<tr>
<td>Ground alfalfa hay, %</td>
<td>15.0</td>
</tr>
<tr>
<td>Soybean meal, %c</td>
<td>3.0</td>
</tr>
<tr>
<td>Trace mineral salt, %</td>
<td>0.5</td>
</tr>
<tr>
<td>Vitamin A, I. U./kg. d</td>
<td>1000</td>
</tr>
<tr>
<td>Chlortetracycline, mg./kg. e</td>
<td>22</td>
</tr>
</tbody>
</table>

^a^ Provides 0.22% phosphorus and 0.31% calcium.

^b^ Air-dry basis.

^c^ 44% crude protein.

^d^ Nopcay "30", Nopco Chemical Co., Newark, N.J.

^e^ Aureomycin-50, American Cyanamid Co., Agricultural Division, Princeton, N.J.
### TABLE 4. EFFECT OF DIETARY MINERAL VARIATIONS ON FEEDLOT PERFORMANCE AND CALCULI INCIDENCE - II

<table>
<thead>
<tr>
<th>Sodium bicarbonate, %b</th>
<th>--</th>
<th>--</th>
<th>--</th>
<th>--</th>
<th>--</th>
<th>--</th>
<th>--</th>
<th>2.0</th>
<th>2.0</th>
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<tbody>
<tr>
<td>Phosphorus, %b</td>
<td>0.22</td>
<td>0.22</td>
<td>0.22</td>
<td>0.22</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.22</td>
<td>0.22</td>
<td>0.22</td>
</tr>
<tr>
<td>Calcium, %b</td>
<td>0.31</td>
<td>0.56</td>
<td>1.06</td>
<td>0.56</td>
<td>0.31</td>
<td>0.56</td>
<td>1.06</td>
<td>0.56</td>
<td>0.31</td>
<td>0.56</td>
<td>1.06</td>
</tr>
<tr>
<td>Calcium source</td>
<td>--</td>
<td>CaCO₃</td>
<td>CaCO₃</td>
<td>CaCl₂·2H₂O</td>
<td>--</td>
<td>CaCO₃</td>
<td>CaCO₃</td>
<td>CaCl₂·2H₂O</td>
<td>--</td>
<td>CaCO₃</td>
<td>CaCO₃</td>
</tr>
<tr>
<td>No. of lambsc</td>
<td>30</td>
<td>30</td>
<td>29</td>
<td>30</td>
<td>29</td>
<td>30</td>
<td>28</td>
<td>30</td>
<td>30</td>
<td>29</td>
<td>29</td>
</tr>
<tr>
<td>Av. daily gain, kg.</td>
<td>0.258</td>
<td>0.256</td>
<td>0.287</td>
<td>0.260</td>
<td>0.226</td>
<td>0.257</td>
<td>0.263</td>
<td>0.244</td>
<td>0.262</td>
<td>0.275</td>
<td>0.264</td>
</tr>
<tr>
<td>Av. daily diet, kg.</td>
<td>1.34</td>
<td>1.33</td>
<td>1.42</td>
<td>1.35</td>
<td>1.29</td>
<td>1.40</td>
<td>1.37</td>
<td>1.29</td>
<td>1.39</td>
<td>1.40</td>
<td>1.43</td>
</tr>
<tr>
<td>Kg, feed per kg. gain</td>
<td>5.22</td>
<td>5.22</td>
<td>4.95</td>
<td>5.22</td>
<td>5.69</td>
<td>5.46</td>
<td>5.22</td>
<td>5.29</td>
<td>5.30</td>
<td>5.08</td>
<td>5.43</td>
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<tr>
<td>Dressing percent</td>
<td>48.3</td>
<td>49.4</td>
<td>49.4</td>
<td>49.2</td>
<td>49.7</td>
<td>49.7</td>
<td>49.6</td>
<td>47.5</td>
<td>48.5</td>
<td>48.5</td>
<td>49.5</td>
</tr>
<tr>
<td>Carcass graded</td>
<td>11.3</td>
<td>11.5</td>
<td>11.5</td>
<td>11.1</td>
<td>11.2</td>
<td>11.5</td>
<td>11.4</td>
<td>11.0</td>
<td>11.3</td>
<td>11.4</td>
<td>11.2</td>
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<table>
<thead>
<tr>
<th>Urinary calculi incidence, No, of animals</th>
</tr>
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<tr>
<td>Obstructive</td>
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<tr>
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<td>Total</td>
</tr>
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<td>0</td>
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<td>1</td>
</tr>
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<td>15</td>
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<td>15</td>
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<td>8</td>
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<tr>
<td>0</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>1</td>
</tr>
</tbody>
</table>

---

**a** Data obtained during an 87-day experimental period, June 13 - September 7.

**b** Air-dry basis.

**c** Originally 30 lambs per treatment, but some were lost from causes apparently unrelated to treatment.

**d** Choice = 11, Prime = 14.

**e** Included losses due to urinary obstruction and animals found to have mineral deposits at slaughter.
A 24-hr. urine sample was collected under toluene from two wethers per treatment per day until samples had been obtained from all lambs surviving at the time collections were made. These collections were obtained in steel metabolism cages during the period 34-48 days after initiation of the experiment. The pH of the 24-hr. urine sample was determined using a Beckman Zeromatic pH meter. Titratable alkalinity was measured by titrating a 25 ml. aliquot with 0.05 N hydrochloric acid to a pH of 6.5. Concentrated hydrochloric acid was added to the remainder of the sample at the rate of 2% (v/v). The acidified urine was filtered through multiple layers of cheese cloth, and an aliquot was stored frozen for later analyses.

Serum, urine and feed mineral analyses were performed as described for Experiment I with the exception that serum, urine and feed potassium and sodium were determined by atomic absorption spectrophotometry (Perkin-Elmer, 1968) using a Perkin-Elmer model 303 Atomic Absorption Spectrophotometer.

Some lambs developing urinary tract blockage during the course of the experiment were slaughtered when death appeared imminent. Those remaining after 87 days on experiment were slaughtered, and the bladders and kidneys of all lambs were examined for calculi. The calculi obtained were characterized according to type.

Some lambs were also lost during the experiment due to factors apparently unrelated to experimental treatments. Average daily gain and feed consumption data were calculated only for those lambs finishing the experiment. Feed consumption data were corrected for death losses by subtracting an average value up to that time.
Statistical analysis of urinary calculi incidence was made by the Chi-square method with all other data being analyzed by the orthogonal single degree of freedom comparison (Steel and Torrie, 1960).

Results and Discussion

Data obtained from this experiment are shown in tables 4, 5 and 6. Average daily gain was significantly \((P < .01)\) reduced by increasing the level of dietary phosphorus from 0.22 to 0.47% in the presence of 0.31% calcium. The weight gain depressing effect of this level of phosphorus was almost completely overcome \((P < .01)\) by increasing dietary calcium. When fed at equal calcium levels, no significant differences were observed between the two calcium sources on the basis of weight gains. However, calcium chloride tended to be less effective than ground limestone in overcoming the weight gain depressing effect of phosphorus. Increasing the calcium level to 1.06% using ground limestone provided no further increase in weight gains except when fed in the absence of added phosphorus and sodium bicarbonate where a non-significant increase was observed. Other workers (Bushman et al., 1965b), supported by data reported herein for Experiment I, have shown that additional dietary calcium is effective in partially overcoming the weight gain depression associated with the feeding of diets containing approximately 0.5 to 0.6% phosphorus. However, weight gain benefits derived from the feeding of high levels of calcium in diets having a lower phosphorus content have generally been lacking.

Although no significant differences in feed consumption were observed, these data followed the same trend as was observed for average daily gains. As reported previously in Experiment I, sodium
### Table 5. Effect of Dietary Mineral Variations on Blood Mineral Values - II

<table>
<thead>
<tr>
<th>Calcium source</th>
<th>--</th>
<th>CaCO$_3$</th>
<th>CaCO$_3$</th>
<th>CaCl$_2$·2H$_2$O</th>
<th>--</th>
<th>CaCO$_3$</th>
<th>CaCO$_3$</th>
<th>CaCl$_2$·2H$_2$O</th>
<th>--</th>
<th>CaCO$_3$</th>
<th>CaCO$_3$</th>
<th>CaCl$_2$·2H$_2$O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium bicarbonate, %b</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phosphorus, %b</td>
<td>0.22</td>
<td>0.22</td>
<td>0.22</td>
<td>0.22</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.47</td>
<td>0.22</td>
<td>0.22</td>
<td>0.22</td>
</tr>
<tr>
<td>Calcium, %b</td>
<td>0.31</td>
<td>0.31</td>
<td>0.31</td>
<td>0.31</td>
<td>0.56</td>
<td>0.56</td>
<td>0.56</td>
<td>0.56</td>
<td>0.56</td>
<td>0.31</td>
<td>0.31</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Serum values, mg./100 ml.

26 - 27 days

| Calcium | 11.0 | 11.9 | 11.4 | 10.9 | 9.7 | 10.5 | 10.4 | 10.4 | 11.0 | 11.2 | 11.0 | 11.8 |
| Magnesium | 2.8 | 2.9 | 2.6 | 2.7 | 3.2 | 3.1 | 2.7 | 2.9 | 2.9 | 2.5 | 2.6 | 2.6 |
| Phosphorus | 6.1 | 5.6 | 5.7 | 6.5 | 8.6 | 7.9 | 7.6 | 7.3 | 6.8 | 6.4 | 6.9 | 6.0 |
| Potassium | 20.5 | 21.3 | 21.1 | 21.0 | 21.0 | 20.4 | 20.9 | 21.5 | 20.7 | 21.0 | 20.7 | 20.7 |
| Sodium | 331 | 341 | 328 | 339 | 336 | 351 | 333 | 344 | 334 | 335 | 349 | 356 |

84 days

| Calcium | 10.2 | 10.7 | 11.0 | 11.1 | 9.1 | 10.6 | 10.3 | 10.9 | 9.7 | 10.9 | 10.8 | 11.0 |
| Magnesium | 2.8 | 2.6 | 2.6 | 2.7 | 3.7 | 3.2 | 2.8 | 2.8 | 2.8 | 2.6 | 2.5 | 2.7 |
| Phosphorus | 8.4 | 7.0 | 7.6 | 7.9 | 9.2 | 8.6 | 8.7 | 7.9 | 7.3 | 7.7 | 8.1 | 7.9 |
| Potassium | 22.3 | 22.2 | 22.3 | 21.7 | 21.8 | 21.8 | 21.3 | 22.0 | 22.4 | 21.1 | 21.0 | 23.0 |
| Sodium | 352 | 351 | 349 | 342 | 350 | 345 | 346 | 344 | 360 | 347 | 343 | 353 |

---

*a* Data obtained during an 87-day experimental period, June 13 - September 7.

*b* Air-dry basis.
<table>
<thead>
<tr>
<th>Mineral Values</th>
<th>pH</th>
<th>Volume, ml./24 hr.</th>
<th>Titratable Alkalinity</th>
<th>Mineral values, mg./100 ml.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium bicarbonate, g</td>
<td>8.4</td>
<td>508</td>
<td>33.4</td>
<td>Calcium</td>
</tr>
<tr>
<td>Phosphorus, g</td>
<td>2.2</td>
<td>760</td>
<td>0.0</td>
<td>Magnesium</td>
</tr>
<tr>
<td>Calcium, g</td>
<td>0.3</td>
<td>681</td>
<td>54.5</td>
<td>Phosphorus</td>
</tr>
<tr>
<td>Calcium source</td>
<td>CaCO₃</td>
<td>--</td>
<td>40.5</td>
<td>Potassium</td>
</tr>
</tbody>
</table>

### Notes:
- Data obtained during an 87-day experimental period, June 13 - September 7.
- Air-dry basis.
- mL. of 0.05 HCl to titrate 25 mL. of urine to pH 6.5.
bicarbonate had no apparent effect on weight gains, feed consumption or feed efficiency. Only small differences between treatments were observed in this experiment for dressing percent and carcass grade.

Only two cases of calculi were found at slaughter in the low phosphorus groups. All other cases of calculi occurred in lambs fed the high phosphorus level (significant, $P < .005$). All calculi formed in this experiment were of the phosphatic type containing both calcium and magnesium phosphates. A 52% incidence of calculi occurred in lambs fed 0.47% phosphorus and 0.31% calcium. The incidence did not differ greatly (50% incidence) when calcium, supplied by ground limestone, was increased to 0.56%, but when the same level of calcium was furnished by calcium chloride there was complete prevention of urinary calculi. Partial protection was provided by the 1.06% calcium level, supplied by ground limestone (incidence of 29% vs. 52%). Adding 2% sodium bicarbonate to a 15% alfalfa hay diet did not promote stone formation in this experiment. This differs from results obtained previously (Experiment I) using an all-concentrate diet.

The elevated dietary phosphorus level (0.47%) significantly increased ($P < .01$) serum phosphorus and magnesium and lowered ($P < .01$) serum calcium at both sampling periods (table 5). Serum calcium was increased for lambs fed the higher level of calcium, and was significantly ($P < .01$) higher than the controls at the 84-day bleeding period. At this time, serum calcium values had a tendency to be higher for lambs fed calcium chloride when compared with those fed ground limestone. Increases in dietary calcium also resulted in lower ($P < .01$) serum magnesium values at the 84-day sampling period with
only a trend in this direction being apparent at the first (26-27 day) sampling. When fed with the low level of phosphorus (0.22%), additional calcium had no effect on serum phosphorus. However, when fed with the higher level (0.47%) of phosphorus, the elevated levels of both calcium sources depressed (P < .01) serum phosphorus at both sampling times. Neither serum potassium nor sodium at either sampling time were significantly affected by any of the levels of calcium, phosphorus or sodium bicarbonate.

Urinary pH was significantly (P < .01) increased by feeding 2% sodium bicarbonate (table 6). This small increase (av. = 0.2 pH units) did not promote urinary calculi. However, sodium bicarbonate was fed only with the lowest level of dietary phosphorus, and it appears that the low urinary phosphorus values associated with the feeding of these diets, as discussed below, may have been a limiting factor in calculi production. In contrast, calcium chloride fed with the high-phosphorus calculogenic diet tended to lower urine pH and greatly reduced calculi incidence. The urinary pH of all lambs exceeded the values of 6.6 to 6.8 above which magnesium and calcium phosphates have been shown to precipitate from urine (Elliot et al., 1961; Carbone, 1965). Titrat­able alkalinity of the urine was increased (P < .01) by feeding 2% sodium bicarbonate, whereas additional phosphorus decreased (P < .01) this value. There was a tendency for calcium chloride to reduce titratable alkalinity.

Additional dietary calcium fed as ground limestone increased (P < .01) urinary calcium concentration only in the absence of the higher level of phosphorus and sodium bicarbonate. However, calcium
chloride increased \( (P < .01) \) urinary calcium concentration in all instances. Sodium bicarbonate and phosphorus reduced \( (P < .01) \) urinary calcium concentration with the former also lowering \( (P < .05) \) the total amount of calcium excreted in 24 hours. Urinary magnesium (mg./100 ml.) was lowered by sodium bicarbonate \( (P < .05) \) and by additional phosphorus \( (P < .01) \). However, the total amount of magnesium excreted in 24 hr. was not affected by any of the dietary treatments.

Urinary phosphorus concentration and 24-hr. excretion values were increased \( (P < .01) \) by the addition of phosphorus to the diet. Lambs fed the highest level of phosphorus \( (0.47\%) \) and \( 0.56\% \) calcium, supplied by ground limestone, had the highest average urinary phosphorus concentration. The factors contributing to the difference in urinary phosphorus excretion between this group and the corresponding high phosphorus, low calcium group are not understood at this time. However, the failure of ground limestone at the \( 0.56\% \) calcium level to reduce urinary phosphorus excretion in this instance is consistent with its failure to offer protection against urinary calculi. Elevating dietary calcium to \( 1.06\% \) in the high phosphorus diet reduced urinary phosphorus concentration and depressed calculi formation.

Potassium concentration of the urine was decreased \( (P < .01) \) by sodium bicarbonate or additional phosphorus. This appeared to be due largely to a higher \( (P < .05) \) urine volume for lambs on these treatments. With the higher level of phosphorus, additional calcium increased \( (P < .05) \) the total amount of potassium excreted in 24 hr., but the values appeared to bear no relationship to the extent of
protection provided by the various levels or sources of calcium.

The failure of sodium bicarbonate to promote urinary calculi in this experiment conflicts with results obtained in Experiment I. One of the major differences between the two experiments is the feeding of 15% alfalfa hay in this experiment as opposed to the all-concentrate diet fed previously. Udall et al. (1958) reported that alfalfa hay in the diet decreases protein-bound hexosamine in the urine, thus decreasing calculi formation. However, one of the most obvious differences noted in the urinary data between Experiments I and II was that urinary phosphorus was much higher, and the increase in urinary pH attributed to the feeding of 2% sodium bicarbonate was greater, when an all-concentrate diet was fed. Average urinary phosphorus concentrations for groups of lambs fed an all-concentrate diet, as reported herein in Experiment I, were 19-99 mg./100 ml., while those obtained in this experiment with diets containing 15% alfalfa hay and comparable calcium and phosphorus levels were only 0.8-24 mg./100 ml. The low urinary phosphorus values associated with the feeding of diets containing alfalfa hay may have been a limiting factor in the lack of calculi formation in sodium bicarbonate fed lambs in this experiment. This difference in urinary phosphorus excretion remains unexplained at the present.

A dietary calcium to phosphorus ratio of 2:1 or higher has been recommended by Bushman et al. (1965b) for the prevention of phosphatic calculi. With the higher level of phosphorus, the lower level of calcium used in the experiment reported herein gave a calcium to phosphorus ratio of 0.7:1. With the higher level of calcium the ratio
was 2.3:1. When the added calcium was supplied as ground limestone, even the highest level provided only partial protection, but with calcium chloride providing the added calcium (approximately 40% of the total calcium) a ratio of 1.2:1 provided complete protection. The difference is undoubtedly related to the acid-forming characteristics of the latter.

Summary

An experiment using 360 lambs was conducted to compare the effects of calcium carbonate (ground limestone) and calcium chloride on feedlot performance and phosphatic urolithiasis when fed in a 15% alfalfa hay diet containing added phosphorus or sodium bicarbonate. The incomplete factorial involved 0 and 2% sodium bicarbonate, 0.22 and 0.47% phosphorus and 0.31, 0.56 and 1.06% calcium in the diet. At the 0.56% calcium level, both ground limestone and calcium chloride were used, while only ground limestone was utilized at the higher calcium level. Sodium bicarbonate was fed only with the low phosphorus series of treatments.

The higher level of phosphorus (0.47%) reduced weight gains, but additional calcium, in the form of ground limestone or calcium chloride, overcame this depression. Calcium chloride appeared to be less effective than ground limestone in overcoming the weight gain depressing effect of high dietary phosphorus, but was more effective in preventing urinary calculi. The greatest incidence (50-52%) of calculi was obtained by feeding 0.47% phosphorus and 0.31% or 0.56% calcium furnished by ground limestone. However, by increasing dietary calcium to 1.06% with ground limestone, calculi incidence was reduced.
(29% versus 50%). With the feeding of calcium chloride (0.56% calcium level), calculi were completely prevented. The feeding of sodium bicarbonate in these 15% alfalfa hay diets did not promote calculi formation, although it did cause a slight increase in urine alkalinity.
Conflicting reports have appeared in the literature concerning the relationship between dietary potassium and ovine urolithiasis. An increased intake of calcium and/or potassium was reported by Robbins et al. (1965a) to decrease the incidence of urinary calculi in wether lambs fed calculogenic diets having phosphorus contents of 0.20 to 0.35%. Using similar dietary conditions, Crookshank (1966) concluded that potassium salts offered more protection than corresponding sodium salts. On the other hand, Bushman et al. (1968) found 1% potassium chloride to increase the incidence of urinary calculi in wether lambs fed a high-phosphorus (0.55%), calculogenic diet. A high phosphorus intake with a consequential elevation of urinary phosphorus has been shown by several workers (Emerick et al., 1959; Packett and Hauschild, 1964; Bushman et al., 1965a, b; Robbins et al., 1965a) to be associated with phosphatic urolithiasis. Elevated levels of dietary calcium (Emerick and Embry, 1963, 1964; Bushman et al., 1965a, b) and certain salts, including sodium chloride (Elam et al., 1957; Udall, 1959; Udall and Chow, 1963), ammonium chloride (Crookshank et al., 1960; Bushman et al., 1967, 1968) and calcium chloride (Bushman et al., 1967, 1968), have been shown to offer some degree of protection.

The experiment reported herein was conducted to determine the influence of added dietary potassium as potassium chloride on feedlot performance and formation of phosphatic urinary calculi in lambs fed diets containing various levels of phosphorus and calcium.
Experimental

This experiment utilized 240 Texas ewe lambs of mixed breeding and was conducted over a 95-day period in the summer. Although it is rare to have obstructive urinary calculi in ewes, the incidence of non-obstructive calculi, as determined at slaughter, has been shown to be similar in ewes and wethers fed high-phosphorus, calculogenic diets (Emerick and Embry, 1964). In order to assess the influence of supplemental potassium on feedlot performance, it was deemed desirable to avoid large losses of animals from obstructive calculi in any of the experimental groups. The lambs, averaging 27.3 kg., were allotted on the basis of weight to eight treatments replicated three times with 10 lambs per pen initially. Approximately 2 weeks before the start of the experiment, the lambs were vaccinated for prevention of enterotoxemia and drenched with thiabendazole.

The experimental design was a 2 x 2 x 2 factorial involving two levels of potassium (0.41 and 1.01%), two levels of phosphorus (0.24 and 0.57%) and two levels of calcium (0.28 and 1.20%). The basal diet consisted of 76.6% ground shelled corn, 20% ground alfalfa hay, 0.5% trace mineral salt and 2.9% soybean meal (44% crude protein) plus 227 I.U. vitamin A per kg. of diet. The lower level of each treatment variable was inherent in the basal diet. In addition, it contained 12.3% crude protein (N x 6.25) and 0.16% magnesium. The higher levels of phosphorus and calcium were obtained by adding 1.52% anhydrous disodium phosphate and 2.27% ground limestone, respectively. Addition of 1% potassium chloride provided the higher level of potassium. All mineral additions were made at the expense of corn.
Initially, lambs were fed 0.32 kg. of the experimental diets plus 0.46 kg. of chopped alfalfa hay daily. The chopped alfalfa hay was eliminated and corresponding increases in the experimental diets were made over a 5-day period. Thereafter, they were fed the experimental diets once daily so feed was available at all times. Water was given ad libitum.

A blood sample was obtained by jugular vein puncture from each lamb after 30 days on experiment, and the serum was stored frozen until analyzed. Serum and feed mineral analyses were performed as described for experiment 1, with the exception of serum potassium, which was determined by flame photometry (Coleman, 1956).

One lamb was lost due to factors apparently unrelated to experimental treatment. Feed consumption data were corrected in this instance by subtracting an average value for the lamb removed. At the termination of the trial, the lambs were slaughtered and the urinary bladders and kidneys were examined for calculi.

Statistical analyses were performed by methods outlined by Steel and Torrie (1960). Data from nine lambs per pen, or 27 lambs per treatment, were used in the analyses of variance for feedlot performance and blood values, the extra data being withdrawn at random to provide equal numbers. Statistical analyses of urinary calculi incidence among treatments were made by the Chi-square method.

Results and Discussion

Data obtained from this experiment are shown in table 7. Increasing the phosphorus content of the low calcium diet from 0.24 to 0.57% tended to reduce weight gains and feed consumption with the greatest
TABLE 7. DATA ON FEEDLOT PERFORMANCE, SERUM VALUES AND URINARY CALCULI INCIDENCE - III

<table>
<thead>
<tr>
<th></th>
<th>Control series</th>
<th></th>
<th>1% KCL series</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphorus (g)</td>
<td>0.24 0.24</td>
<td>0.57 0.57</td>
<td>0.24 0.24</td>
<td>0.57 0.57</td>
</tr>
<tr>
<td>Calcium (g)</td>
<td>0.28 1.20</td>
<td>0.28 1.20</td>
<td>0.28 1.20</td>
<td>0.28 1.20</td>
</tr>
<tr>
<td>No. of lambs</td>
<td>30 30</td>
<td>29 30</td>
<td>30 30</td>
<td>30 30</td>
</tr>
<tr>
<td>Av. daily gain, kg.</td>
<td>0.197 0.197</td>
<td>0.180 0.189</td>
<td>0.194 0.200</td>
<td>0.168 0.200</td>
</tr>
<tr>
<td>Av. feed consumption, kg./day</td>
<td>1.20 1.23</td>
<td>1.15 1.20</td>
<td>1.18 1.24</td>
<td>1.13 1.21</td>
</tr>
<tr>
<td>Feed per kg. gain, kg.</td>
<td>6.09 6.23</td>
<td>6.44 6.32</td>
<td>6.10 6.20</td>
<td>6.75 6.05</td>
</tr>
<tr>
<td>Carcass grade</td>
<td>11.8 11.9</td>
<td>11.5 11.4</td>
<td>11.6 11.4</td>
<td>11.3 11.6</td>
</tr>
<tr>
<td>Urinary calculi incidence</td>
<td>1 0</td>
<td>16 5</td>
<td>0 0</td>
<td>17 5</td>
</tr>
<tr>
<td>Serum values, mg. per 100 ml.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phosphorus</td>
<td>7.05 6.66</td>
<td>8.66 8.17</td>
<td>6.94 6.40</td>
<td>8.90 7.85</td>
</tr>
<tr>
<td>Calcium</td>
<td>10.6 11.0</td>
<td>9.4 10.1</td>
<td>10.7 10.7</td>
<td>9.6 10.0</td>
</tr>
<tr>
<td>Potassium</td>
<td>21.5 21.8</td>
<td>22.2 22.1</td>
<td>22.4 22.8</td>
<td>22.6 22.4</td>
</tr>
</tbody>
</table>

a 95-day experimental period.

b Air-dry basis.

c Initially 30 lambs per treatment, but one was lost due to causes unrelated to treatments.

d Choice = 11, prime = 14.

e Represents animals having urinary mineral deposits at slaughter; no obstructive cases were observed.
reduction in weight gains occurring when the diet also contained 1% potassium chloride. Increasing dietary calcium from 0.28 to 1.20% resulted in significantly (P < .05) higher weight gains and feed consumption. In this regard, the added calcium completely overcame the weight gain depressing effect of the higher level of phosphorus with and without 1% potassium chloride.

Average weight gain of lambs fed the high-phosphorus, low-calcium diet with 1% potassium chloride was 6.7% below that of the corresponding group not fed potassium chloride. Using a diet having phosphorus and potassium contents exceeding those in the current study (0.64% P, 0.67% K vs. 0.57% P, 0.41% K), Bushman et al. (1968) obtained a 27% reduction in weight gains of wether lambs by feeding an additional 1% potassium chloride. It is not known if the differences in phosphorus and potassium levels may have contributed to the difference in results between the two studies.

No obstructive cases of urinary calculi were observed in the ewe lambs used in this experiment. Therefore, the urinary calculi incidence represents mineral deposits found in urinary bladders and kidneys at the time of slaughter. Calculi were found in only one of the lambs fed the lower (0.28%) level of phosphorus. Lambs fed diets with 0.57% phosphorus and 0.28% calcium had a 55 to 57% incidence of calculi. Increasing the calcium level to 1.20% of the diet reduced the incidence to 17%. The increase in urinary calculi formation attributed to the higher level of dietary phosphorus and the reduction resulting from an increase in dietary calcium were significant (P < .005).

Feeding 1% potassium chloride had no effect upon the number of
animals that developed calculi, but it appeared to promote the formation of larger calculous deposits. Average sizes of the deposits were 120 and 83 mg. for lambs on treatments with and without potassium chloride, respectively. In work reported previously (Bushman et al., 1968), a significant increase in the incidence of urinary calculi attributed to the feeding of 1% potassium chloride to wether lambs was accompanied by an apparent increase in obstructive cases. In the current study, the larger size of calculous mineral deposits in ewe lambs fed potassium chloride is further support of a detrimental effect of this salt when fed in conjunction with an elevated level of dietary phosphorus.

With an increase in dietary phosphorus (0.24 vs. 0.57%), serum phosphorus values were significantly ($P < .05$) increased and serum calcium values were significantly ($P < .05$) decreased. Raising the level of dietary calcium from 0.28 to 1.20% caused an increase ($P < .05$) in serum calcium and a decrease in serum phosphorus ($P < .1$) and magnesium ($P < .01$). Groups of lambs exhibiting higher serum calcium values in response to the higher level of dietary calcium included one exception, i.e. those fed 1.20% calcium, 0.24% phosphorus and 1% potassium chloride. This appeared to contribute to a significant ($P < .05$) potassium x calcium interaction that occurred in addition to the more commonly recognized phosphorus x calcium interaction ($P < .05$) regarding serum calcium values.

Serum potassium values were significantly ($P < .05$) increased by the addition of 1% potassium chloride to the diet. This increase amounted to an average of only 0.64 mg. potassium per 100 ml., but
was accompanied by very little within-treatment variation. Dietary potassium had no apparent effect on serum calcium, phosphorus or magnesium. However, there was a tendency for the higher level of either dietary calcium or phosphorus to increase (nonsignificant) serum potassium values. A significant \((P < .05)\) interaction between calcium and phosphorus relative to their effects on serum potassium was observed. Dietary calcium is known to reduce intestinal absorption of phosphorus (Gill et al., 1959), and this may have negated to some extent the effect of phosphorus on serum potassium.

Data appearing in the literature previously indicate a potassium x phosphorus and/or calcium interrelationship. Gillis (1948) showed that chicks receiving diets deficient in phosphorus benefited from the addition of potassium as measured by growth and bone calcification. Also, potassium x phosphorus and potassium x calcium interactions pertaining to growth of rats were reported by Robbins et al. (1965b). However, their high-phosphorus diets appeared to contribute to urinary calculi formation in rats only under conditions of a potassium deficiency. The latter observation appears to conflict with results reported for sheep. Elam et al. (1956) found combinations of potassium carbonate and phosphoric acid to be more calculogenic than either one alone added to the basal diet, but they recognized the role that differences in urine reaction may have played in their studies. In a study appearing not to involve differences in urine reaction, Bushman et al. (1968) found that 1% of potassium chloride fed to wethers in a high-phosphorus, calculogenic diet significantly reduced weight gains and increased the incidence of phosphatic urinary calculi.
Although urinary calculi incidence in the studies reported herein was not increased in ewe lambs by the feeding of 1% potassium chloride, the average size of the calculous deposits was larger. This observation, as well as growth and blood serum data, indicate the existence of a potassium-phosphorus interrelationship that is detrimental to lambs fed potassium chloride in a high-phosphorus, calculogenic diet.

**Summary**

Two levels of potassium (0.41 and 1.01%), two levels of phosphorus (0.24 and 0.57%) and two levels of calcium (0.28 and 1.20%) were used in a 2 x 2 x 2 factorially designed experiment to determine the influence of excess dietary potassium on feedlot performance and the formation of phosphatic urinary calculi in lambs fed various levels of phosphorus and calcium. A total of 240 Texas ewe lambs were allotted to the eight treatments.

There was a tendency toward lower weight gains and feed consumption for lambs fed the higher level of dietary phosphorus. This effect was most pronounced in the presence of the highest level of potassium (1% potassium chloride added to the diet). Added calcium completely overcame these effects. No obstructive urinary calculi were observed in these ewe lambs. However, those fed the highest level of phosphorus and the lowest level of calcium had a 55 to 57% calculi incidence as determined at slaughter. While the incidence of urinary calculi was no different in the lambs fed the higher level of potassium, the average size of the urinary mineral deposits was larger amounting to 120 and 83 mg., for lambs on treatments with and without 1% potassium chloride, respectively. Increasing the level of calcium to 1.20% of the diet
reduced the calculi incidence to 17%. These data support the existence of a detrimental potassium-phosphorus interrelationship expressed in lambs fed elevated levels of each.
SUMMARY AND CONCLUSIONS

The most important factors among those shown to influence phosphatic urinary calculi formation appear to be dietary factors contributing to elevated urinary phosphorus concentrations and/or urine alkalinity. The urine of ruminants is normally alkaline and in these experiments ranged from an average pH of 7.8 for lambs fed an all-concentrate basal diet to 8.4 for lambs fed a similar diet containing 15% alfalfa hay. However, urinary phosphorus concentrations were notably higher (0.8 - 23.6 vs. 19 - 99 mg./100 ml.) in lambs fed the all-concentrate diet. Neither of these basal diets, an all-concentrate diet or one containing 15% hay, promoted calculi. Additional dietary phosphorus rendered both of these diets calculogenic, but sodium bicarbonate had a calculogenic effect only with the all-concentrate diet. Lambs fed diets made calculogenic by virtue of added phosphorus or sodium bicarbonate showed no increase in nondialyzable urinary protein, hexose and hexosamine. Serum and urine potassium and sodium appeared to have no important relationship to urinary calculi formation in sheep, except when they contribute to urine alkalinity.

The feeding of sodium bicarbonate with either an all-concentrate or 15% hay diet did not influence feed consumption or weight gains in lambs. In addition to its calculogenic effect, excess dietary phosphorus tended to depress weight gains. Additional dietary calcium was partially effective in overcoming the weight gain depressing effect of excess phosphorus, and offered some degree of protection against urinary calculi presumably by reducing urinary phosphorus excretion.
With dietary phosphorus levels approximating 0.4 - 0.5%, the calcium to phosphorus ratio should exceed 2:1 to provide a high degree of protection against calculi when ground limestone serves as the source of supplemental calcium. Calcium chloride provided more protection against calculi formation than was obtained from the use of ground limestone at equivalent calcium levels. Calcium chloride appears to provide protection against calculi formation by depressing urinary phosphorus excretion and lowering urine pH.

While a low calcium to phosphorus ratio in the presence of an elevated level of dietary phosphorus appears to be an important prerequisite for urinary calculi, a low calcium to phosphorus ratio (0.5-1:1) did not promote calculi when accompanied by a dietary phosphorus level approximating requirements. The feeding of 1% potassium chloride in a high-phosphorus, low-calcium diet appears to contribute to an exaggeration of the calculogenic and weight gain depressing effects of this diet. This effect is also overcome by the use of additional calcium.
LITERATURE CITED


