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Summary

An animal model utilizing Sprague-Dawley rats fed a diet containing 2% of tetraethylorthosilicate (TES) has been used to study urinary conditions and dietary factors preventing silica urinary calculi. The basal TES diet produced approximately a 50% incidence of silica urinary calculi. Supplemental dietary phosphorus and the urinary acidifying agent ammonium chloride greatly reduced the urinary calculi incidence, while supplemental dietary calcium and urine alkalinization with sodium bicarbonate increased the incidence. The protection from silica urinary calculi provided by sodium or ammonium phosphate compounds was found to be independent of their urinary acidifying effects, and the effects of phosphates and ammonium chloride were additive. While these observations have not been extended to cattle and sheep, high silica range grasses often contain suboptimum levels of phosphorus. Therefore, the feeding of phosphorus supplements free choice to animals on the range appears to be a sensible management practice that may help prevent silica urinary calculi.

(Key Words: Urinary Calculi, Silica, Phosphorus, Calcium, Ammonium Chloride, Sodium Bicarbonate.)

Introduction

Urinary calculi composed mainly of silica are common in cattle and sheep grazing western ranges and often cause urinary tract blockage and subsequent death of male or castrated male animals. The source of silica appears to be the range grasses that contain approximately 3% silica (dry basis) in the spring months and amounts often exceeding 7% in the fall and winter. While the incidence of latent silica deposits in the kidneys of grazing animals is often in the range of 20 to 90%, attempts to experimentally produce similar deposits in cattle or sheep have not met with success. Therefore, an animal model using laboratory rats fed diets containing 2% of the silicon compound tetraethylorthosilicate (TES) producing approximately a 50% incidence of silica urinary calculi has been used for the study of dietary factors influencing urinary silica deposition. Results of these studies are reported herein. While not directly applicable to the control of silica urinary calculi in ruminants, these studies provide insight concerning urinary conditions and dietary factors that may be used in future field studies for urinary calculi prevention in grazing cattle and sheep. Part of this research has been published in greater detail elsewhere (Emerick, 1984, 1986; Schreier and Emerick, 1986).

Materials and Methods

Sprague-Dawley male rats (Sasco, Inc., Omaha, NE) having initial weights of 50 to 60 g were used in these studies. Rats were individually caged under environmentally controlled conditions that included a temperature of 23 to 25° C and 12 hours of light daily. The basal diet consisted of dextrose, 69%; casein, 20%; corn oil, 5%; salts mixture P-H (ICN Nutritional Biochemicals, Cleveland, OH), 4%; vitamin mixture (ICN Nutritional Biochemicals), 2%. This diet provided .43% phosphorus, .50% calcium, .17% sulfur and .43% chloride. Dietary treatment variables were added at the expense of dextrose. Deionized water was available ad libitum.

Experimental periods were of an 8-week duration. Water consumption was measured for a 48-hour period after 2, 4 and 6 weeks and urine was collected from one-half of the rats for a 24-hour period after 3, 5 and 7 weeks on experiment. At termination, blood samples were obtained by heart puncture, the rats were killed and urinary tracts were examined for mineral deposits.

Experiment 1. Four treatments were as follows: basal diet (control), an equal molar mixture of mono- and dibasic sodium phosphate (MDP) providing .2% supplemental phosphorus, sodium sulfate providing .32% supplemental sulfur and sodium chloride providing .70% supplemental chloride. The quantities of added salts were all chemically equivalent (.2 equivalents per kg diet) and all diets contained 2% TES.

Experiment 2. In view of the interrelationship between calcium and phosphorus and the protection against silica urinary calculi provided by supplemental phosphorus, this experiment was conducted to determine whether supplemental calcium would increase the incidence of silica urinary calculi. Treatments with and without 2% TES included 0, 1 and 2% levels of calcium carbonate (CC). These provided 0, .4 and .8% additional calcium, respectively. An additional .2% supplemental phosphorus was added to all diets to reduce the calculogenic potential previously associated with the 2% TES diet.

Experiment 3. To determine the extent that the protective effect of phosphorus was due to its urinary acidifying effect, three treatments with and without 2% TES were designed to provide equal concentrations of phosphorus while providing variations in urinary acidifying and alkalinizing effects. These treatments providing .3% supplemental phosphorus (.61% total P and .50% Ca in the diet) included monobasic sodium phosphate (MP), dibasic sodium phosphate (DP) and DP + sodium bicarbonate (SB). In the order listed, these diets contribute increasing amounts of alkali to the urine.

Experiment 4. Spray-dried egg albumin was substituted for casein as a protein source in this experiment designed to further characterize the interrelationship between dietary phosphorus and urine pH in the prevention of silica urinary calculi. Egg albumin provides a lower amount of phosphorus (.29% P in the basal egg albumin diet vs .43% P in the basal casein diet) and contributes toward an alkaline urine. Treatments included an equal molar mixture of MP and DP (MDP), .75% ammonium chloride (AC), MDP + .75% AC and dibasic ammonium phosphate (DAP). The phosphate salts, when used, provided .22% supplemental phosphorus. AC has been previously used for urine acidification. DAP provided the same amount of phosphorus and a slightly lower amount of ammonium ion as the treatment MDP + AC.

Experiment 5. The egg white substituted diet described for experiment 4 was used with variations in DP and AC to determine if the calculi protective effect of phosphorus is dependent upon an acidic urine. Three levels of DP providing 0, .15 and .30% supplemental phosphorus were fed with and without .36% AC. This level of AC was shown in a preliminary study to cause slight urine acidification but to be below the critical level required for silica urinary calculi prevention.

Results and Discussion

Data are presented in table 1. Only those treatments providing 2% TES are shown. All urinary calculi formed in these treatments were confirmed by analysis to be the silica type, with silica comprising greater than 90% of the ash.

In experiment 1 the incidence of calculi amounting to 10/20 rats in the control group was reduced ($P<.05$) by dietary additions of phosphate (1/20) and chloride (3/20) but not by sulfate (7/20). Reductions in calculi associated with supplemental dietary phosphorus or chloride occurred with no increase in water consumption (data not shown) or urine volumes and with only small differences in urine pH. Urine phosphorus concentration was increased ($P<.05$) by the phosphate treatment, and urine calcium concentration tended to be increased by sulfate. Blood plasma phosphorus concentration was increased ($P<.05$) by phosphate and chloride.

In experiment 2 incidence of silica urinary calculi increased with increases in CC. A high correlation coefficient of $r = .99$ ($P<.02$) was obtained. The higher calculi incidence associated with the feeding of CC occurred in association with a 6 to 12% increase in water intake and a trend toward higher urine volumes. Other urinary differences ($P<.01$) caused by CC include a higher pH, greater calcium concentration and lower magnesium and phosphorus concentrations. In addition, blood plasma phosphorus and magnesium concentrations were reduced ($P<.01$) by CC.

In experiment 3 no urinary calculi were associated with the most acidic form of dietary phosphate (MP). The numbers of rats developing urinary calculi were greatest for the treatments having the greatest alkali-forming potential. However, all calculi found in association with these high-phosphorus diets in experiment 3 were small, averaging only 1 mg or less as opposed to 3 to 10 mg for calculi from low phosphorus treatments in the previous two experiments. In addition to higher urine pH, urinary silica concentrations were 15 to 25% higher for the DP and DP + SB treatments compared to the MP treatment.

In experiment 4 the calculi incidence of 11/24 in the controls was greatly reduced by phosphorus in the form of either DP (1/24) or DAP (3/24). No calculi occurred in either group fed .75% AC with or without supplemental phosphorus. AC and DAP had the greatest urinary acidifying effects. However, MDP also contributed to urine acidification to a small degree. Therefore, it was not possible to separate primary effects of dietary phosphate from its tendency toward urine acidification as factors contributing to prevention of silica urinary calculi. This separation was subsequently achieved in experiment 5 where the incidence of silica calculi, amounting to 10/20 in controls, was greatly reduced to 5/20 by .15% phosphorus and 2/20 by .30% phosphorus in the form of DP without an effect on urine pH. AC at a level of .36%, while significantly reducing urine pH, did not significantly reduce the incidence of urinary calculi

(8/20). No calculi occurred in treatments providing AC with either level of DP, indicating that the independent effects of dietary phosphorus and urinary acidifying agents were additive.

It is concluded that supplemental phosphorus, in the absence of corresponding increases in dietary calcium, and urinary acidifying agents including acidic forms of phosphate and AC are effective in the prevention of silica urinary calculi in the animal model used for these studies. Application of these results to ruminants has not yet been made. Attempts toward ruminant application should be made with caution in that high dietary phosphorus levels accompanied by an alkaline urine have been previously defined as a cause of phosphatic urinary calculi, a form of calculi most prevalent in feedlot cattle and sheep. Therefore, only acidic forms of phosphorus or phosphorus sources incorporated into supplements having an acid-forming potential should be used in attempts to extend the results of this animal model to grazing ruminants.

References

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TABLE 1. INFLUENCE OF VARIOUS TREATMENTS ON SILICA URINARY CALCULI INCIDENCE AND URINE AND BLOOD PLASMA PARAMETERS

Treatment	Urinary calculi incidence	Urine						Blood plasma		
		Volume		Silica	P	Ca	Mg	P	Ca	Mg
		ml/24 h	pH	mg/100 ml				mg/100 ml		
Experiment 1										
Control	10/20[j]	23.0	6.5[j]	56	105[j]	8.3[jk]	20.0	6.4[j]	10.6	1.78
MDP (.2% P)	1/20[k]	22.3	6.6[j]	56	221[k]	6.5[j]	16.4	6.8[jk]	10.5	1.65
Sulfate (.32% S)	7/20[j1]	25.2	6.8[k]	52	95[j]	10.7[k]	18.5	6.3[j]	10.5	1.62
Chloride (.7% Cl)	3/20[k1]	27.2	6.7[k]	46	87[j]	7.6[j]	15.6	7.2[k]	10.7	1.74
Experiment 2[a,b]										
0% CC	7/20	17.7	6.6	84	159	8.7	23.2	7.0	10.2	1.52
1% CC	9/20	20.5	7.3	58	54	17.2	16.0	6.4	10.3	1.58
2% CC	12/20	22.2	7.5	60	22	28.3	14.4	5.5	10.4	1.36
Experiment 3										
MP (.3% P)	0/20[j]	20.4	6.5[j]	50[j]	201	5.7	9.3[j]	6.2	9.7	1.36
DP (.3% P)	3/20[k]	21.5	6.8[k]	58[k]	208	5.0	10.3[jk]	6.6	10.0	1.33
DP (.3% P) + .5% SB	4/20[k]	20.8	7.1[1]	62[1]	240	5.6	11.9[k]	6.9	10.0	1.27
Experiment 4[c,d,e,f]										
Control	11/24[j]	19.9	7.4	55	41	21.0	25.2	7.9	10.7	1.66
MDP (.22% P)	1/24[k]	19.8	7.1	59	170	11.0	23.2	8.4	10.8	1.78
.75% AC	0/24[k]	19.6	6.5	60	52	19.1	26.2	7.3	10.8	1.65
MDP (.22% P) + .75% AC	0/24[k]	21.8	6.4	54	164	13.9	21.0	8.5	10.4	1.52
DAP (.22% P)	3/24[k]	23.8	6.7	54	151	11.2	19.6	8.6	10.5	1.70
Experiment 5[g,h,i]										
Control	10/20[j]	22.7	7.5	60	40	12.9	27.7	6.9	10.1	1.77
DP (.15% P)	5/20[j]	26.3	7.4	51	99	8.8	22.0	6.9	9.8	1.67
DP (.3% P)	2/20[k]	25.1	7.4	60	167	5.5	19.8	7.4	10.1	1.76
.36% AC	8/20[j]	21.3	7.2	61	45	16.2	28.2	7.3	10.3	1.82
.36% AC + DP (.15% P)	0/20[k]	26.0	7.2	58	98	6.7	20.4	7.3	10.3	1.78
.36% AC + DP (.3% P)	0/20[k]	26.1	7.1	51	164	6.8	19.3	7.6	10.3	1.82

[a] CC-urinary calculi incidence correlation, r = .99 (P<.02). [b] CC effect (P<.01): urine pH, Ca, Mg, P; blood plasma Mg, P. [c] MDP effect (P<.01): urine pH, Ca, Mg, P, blood plasma P. [d] AC effect (P<.01): urine pH. [e] AC x MDP interaction (P<.01): urine pH. [f] AC + MDP and DAP treatments differ (P<.01): urine pH, Ca; blood Mg. [g] DP effect (P<.05): urine volume, Ca, Mg, P. [h] AC effect (P<.01): urine pH, blood plasma Ca. [i] DP x AC interaction (P<.05): urine silica, Ca. [j,k,l] Means within the same column and experiment not having a common superscript differ (P<.05).