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# EFFECT OF DIETARY ALUMINUM ON ANIMAL PERFORMANCE AND TISSUE MINERAL LEVELS IN GROWING STEERS<sup>1</sup>

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## SUMMARY

Twenty-four beef-type steers with an average body weight of 226 kg were allocated to four treatments and fed for 84 days to study effects of dietary aluminum on performance and mineral composition of selected tissues. Treatments included 0, 300, 600 and 1,200 ppm supplemental aluminum as aluminum chloride. Increased dietary aluminum did not affect feed consumption, body weights, or feed conversion ratios nor plasma concentrations of phosphorus, calcium, magnesium and aluminum. Hemoglobin and hematocrit were not affected by increasing dietary aluminum; likewise concentrations of aluminum, iron, manganese, phosphorus and calcium concentrations in liver, kidney, *longissimus* muscle and brain were not influenced by treatment. Zinc concentrations were increased in liver ( $P < .05$ ) and kidney ( $P < .01$ ), by added levels of dietary aluminum. Dietary aluminum at levels up to 1,200 ppm did not influence animal performance and caused only minor changes in tissue mineral composition.

(Key Words: Aluminum, Aluminum Toxicity, Tissue Mineral Levels, Cattle.)

## INTRODUCTION

Grazing ruminants are known to consume soil in variable quantities which contains varying levels of aluminum. This element has

been shown to interfere with phosphorus availability by formation of insoluble phosphates in rats (Jones, 1938; Street, 1942), chicks (Storer and Nelson, 1968), guinea pigs and rabbits (Cox *et al.*, 1931) and man (Clarkson *et al.*, 1972), but little information is available for ruminants. Hobbs *et al.* (1954) fed cattle and sheep .5% aluminum sulfate (405 ppm aluminum) as an alleviator of flourine poisoning, without observing any deleterious effect on the animals' performance. Thompson *et al.* (1959) found no effect when 1% aluminum sulfate (810 ppm aluminum) was fed to sheep.

The objective of the present experiment was to investigate the effect of increasing levels of dietary aluminum upon performance, blood components and tissue mineral composition of growing steers.

## EXPERIMENTAL PROCEDURE

Twenty-four steers of mixed beef breeding with an average initial body weight of 226 kg were allotted to four treatments. Experimental diets included the basal (table 1) and basal plus 300, 600 or 1,200 ppm aluminum. Aluminum chloride ( $AlCl_3 \cdot 6H_2O$ ), a soluble salt of aluminum, was used and was substituted for cornstarch in the basal diet.

The steers were housed in sheltered pens and fed as a group for 84 days. The steers were weighed initially and each 14 days thereafter. Tap water was provided *ad libitum*. Feed allowance was increased gradually during the first 2 weeks and an *ad libitum* intake was provided during the remainder of the experimental period. Blood samples were collected by jugular puncture at the beginning of the trial and each 28 days thereafter. At slaughter, samples of liver, kidney, *longissimus* muscle and brain were collected for mineral analyses.

Determinations for aluminum, calcium and magnesium, in feed and plasma and aluminum, calcium, magnesium, iron, copper, zinc and

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TABLE 1. COMPOSITION OF BASAL DIET

Ingredient	Internat'l Ref. No.	Percent
Corn, yellow, grain, grnd	4-02-992	59.4
Soybean, seeds, solv-extd grnd	5-04-604	12.0
Cotton, seed hulls	1-01-599	20.0
Alfalfa, aerial part, dehy grnd, mn 17% protein	1-00-023	3.0
Sugarcane, molasses, mn 48% invert sugar mn 79.5 degrees brix	4-04-696	3.0
Corn, starch, dehy grnd	4-02-889	1.0
Salt, trace mineralized <sup>a</sup>		1.0
Calcium phosphate, dibasic, commercial	6-01-080	.1
Limestone, grnd, mn 33% calcium	6-02-632	.5
Vitamins A and D <sup>b</sup>		+
Total		100.00
Chemical composition <sup>c</sup>		
Crude protein, %		14.40
Phosphorus, %		.35
Calcium, %		.40
Aluminum, ppm		210.00

<sup>a</sup>Analysis in percent as follows: NaCl, 98.15; Fe, .30; Mn, .26; S, .10; Cu, .08; Co, .10; I, .01; Zn, 1.00.

<sup>b</sup>Vitamins added per kilogram of diet: 2,200 IU of vitamin A palmitate, 440 IU of vitamin D<sub>3</sub>.

<sup>c</sup>Dry matter basis.

manganese in liver, kidney muscle and brain samples were made by atomic absorption spectrophotometry according to methods recommended by the manufacturer (Anonymous, 1973). Phosphorus content of feed, plasma and tissues was determined by a modified colorimetric method of Fiske and Subbarow (1925). Aluminum concentration in tissues was determined in sample solutions prepared by wet-digestion with 50% nitric acid, following the method described by Slavin *et al.* (1975). Aluminum in plasma was determined by flameless atomic absorption spectrophotometry without previous digestion (Anonymous, 1973). Hemoglobin values were determined by the acid hematin method (Cohen and Smith, 1919) and hematocrit values by the microhematocrit method.

Statistical analyses of weight gains and blood parameters (minerals, hematocrit and hemoglobin) were conducted considering treatments (levels of dietary aluminum) as fixed effects and animals (random effects) nested in treatments, and time effects crossed over treatments as main plot effects. Minerals in tissues were analyzed with treatments (levels of dietary aluminum) as main effects and animals nested in treatments. Differences in treatment means were evaluated with regard to linear, quadratic

or cubic trends. Data were processed by computer programs of Statistical Analysis System (Barr *et al.*, 1976).

## RESULTS

*Animal Performance.* Average daily gains were .94, .92, .74 and .89 kg for the steers receiving 0, 300, 600 and 1,200 ppm aluminum, respectively (table 2) and were not influenced by treatment. Since animals were group fed, it was not possible to evaluate statistically the effects of treatment on dry matter intake and feed conversion. Means for feed to gain ratios were 6.29, 6.32, 7.24 and 7.11 and tended to be lower for the steers receiving less aluminum. Weight gains were comparable to expected gains for steers of similar initial live weight and feed intake (NRC, 1976).

*Blood Parameters.* No effects of dietary aluminum on levels of plasma phosphorus, calcium and magnesium were detected (table 3). Plasma phosphorus increased with feeding time ( $P < .01$ ) while plasma calcium decreased ( $P < .01$ ) but all values were considered normal. Plasma values for aluminum ranged from .100 to .118 ppm and there was no relationship between dietary aluminum levels and levels of this element in the circulatory blood. Hemato-

TABLE 2. AVERAGE WEIGHT GAIN, FEED CONSUMPTION AND FEED CONVERSION OF STEERS FED VARIOUS LEVELS OF ADDED DIETARY ALUMINUM<sup>a</sup>

Item	Aluminum (ppm of dry matter)			
	0	300	600	1200
Initial weight, kg	223.00	227.00	227.00	225.00
Final weight, kg	302.00	304.00	289.00	300.00
Daily gain, kg	.94	.92	.74	.89
Dry matter intake, kg/day	5.92	5.81	5.36	6.33
Feed/unit gain	6.29	6.32	7.24	7.11

<sup>a</sup>Six steers per treatment; feeding period 84 days.

crit and hemoglobin levels were not affected by dietary aluminum, but hematocrit increased with time ( $P < .01$ ).

*Mineral Concentration in Tissues.* Aluminum concentrations in all tissues tended to increase when dietary aluminum increased but these trends were not significant statistically (table 4). Tissue aluminum levels, when expressed on a fresh weight basis, were highest in liver, followed by brain, kidney and muscle in decreasing order. Iron, manganese, phosphorus,

calcium and magnesium concentrations were not different among treatments. Zinc concentration was increased by treatment in liver (linear,  $P < .05$ ) and kidney (curvilinear,  $P < .01$ ). Copper concentrations were only different in liver (curvilinear,  $P < .05$ ).

#### Discussion

Feed consumption, body weight gain and feed conversion by steers were not affected

TABLE 3. EFFECT OF DIETARY ALUMINUM ON PLASMA PHOSPHORUS, CALCIUM, MAGNESIUM AND ALUMINUM<sup>a</sup>

Days on experiment	Aluminum (ppm of dry matter)				SD <sup>b</sup>
	0	300	600	1200	
Phosphorus (mg/100 ml)					
0	8.60	8.23	7.87	8.50	.82
28	8.27	8.03	8.13	7.60	.56
56	7.13	6.43	6.55	6.06	.56
84	7.88	7.89	7.17	7.75	1.02
Calcium (mg/100 ml)					
0	9.32	9.45	9.78	9.59	.40
28	10.43	10.52	11.07	10.44	.56
56	9.94	9.72	10.19	10.20	.42
84	9.77	9.58	9.71	10.04	.41
Magnesium (mg/100 ml)					
0	2.86	2.86	2.91	2.94	.18
28	2.70	2.84	2.90	2.67	.21
56	2.77	2.77	2.62	2.56	.22
84	2.53	2.42	2.63	2.57	.21
Aluminum (ppm)					
84	.103	.118	.100	.120	.033

<sup>a</sup>Means represent data from six steers.

<sup>b</sup>Standard deviation calculated from residual mean squares.

TABLE 4. EFFECTS OF DIETARY ALUMINUM ON TISSUE MINERAL COMPOSITION OF STEERS<sup>a</sup>

Dietary aluminum, ppm	Tissue minerals (ppm fresh tissue)							
	Al	Fe	Zn	Cu	Mn	P	Ca	Mg
	Liver <sup>g</sup>							
0	7.6	28.2	19.4 <sup>de</sup>	59.1 <sup>df</sup>	3.00	1502	28.2	98.0
300	5.7	40.9	23.6	33.1	2.87	1841	30.8	122.8
600	10.0	30.5	21.0	48.5	3.06	1683	29.3	111.7
1200	11.2	35.7	26.6	53.5	3.30	1989	31.0	130.0
S.D. <sup>b</sup>	3.6	8.3	3.9	14.2	.71	331	3.6	21.3
	Kidney <sup>g</sup>							
0	4.5	25.5	9.1 <sup>cf</sup>	4.88	.94	1004	35.0	196.4
300	4.1	31.6	10.1	5.25	.74	1123	42.6	210.9
600	6.0	22.6	7.5	5.00	.76	890	32.8	164.5
1200	5.4	32.5	11.7	5.53	.79	1242	42.2	238.4
S.D.	3.1	6.8	2.0	.46	.23	227	12.0	43.0
	Muscle <sup>g</sup>							
0	3.8	15.4	17.8	.89	.11	1482	37.1	224.8
300	3.9	15.7	19.7	.85	.10	1454	34.7	235.5
600	5.4	15.8	22.0	.84	.11	1568	34.7	235.8
1200	4.7	14.1	17.6	.75	.13	1399	37.8	210.7
S.D.	2.4	2.2	3.8	.13	.03	183	12.2	29.8
	Brain <sup>g</sup>							
0	6.4	23.1	8.2	21.9	.41	2173	57.3	117.5
300	7.6	20.8	10.5	20.8	.40	2396	92.8	132.4
600	5.5	19.1	10.1	23.3	.37	2114	76.1	120.5
1200	7.7	24.5	9.8	20.6	.38	2222	67.2	123.7
S.D.	2.2	9.5	1.6	5.0	.07	328	28.2	15.9

<sup>a</sup>Means represent data from six steers.

<sup>b</sup>Standard deviation calculated from residual mean squares.

<sup>c</sup>Significant influence of treatment ( $P < .01$ ).

<sup>d</sup>Significant influence of treatment ( $P < .05$ ).

<sup>e</sup>Means in the same column and tissue follow a linear trend ( $P < .05$ ).

<sup>f</sup>Means in the same column and tissue follow a cubic trend ( $P < .05$ ).

<sup>g</sup>Dry matter contents of tissues in percent were: liver,  $32.71 \pm .94$ ; kidney,  $20.22 \pm 1.25$ ; muscle,  $27.08 \pm 1.81$ ; brain,  $22.46 \pm 1.32$ .

when supplemental aluminum as soluble aluminum chloride was fed at levels up to 1,200 ppm. Hobbs *et al.* (1954) fed .5% aluminum sulfate (405 ppm aluminum) to steers and Thompson *et al.* (1959), fed lambs 1% aluminum sulfate (810 ppm aluminum) and found no depression on animal performance. This led the latter researchers to suggest that ruminants were less susceptible to adverse effects of dietary aluminum than were monogastrics in which dietary aluminum depressed phosphorus utilization. In chicks (Storer and Nelson, 1968) 325 ppm Al as  $\text{Al}(\text{SO}_4)_3 \cdot 18\text{H}_2\text{O}$  produced 5%

mortality while 447 ppm Al as  $\text{AlCl}_3 \cdot 6\text{H}_2\text{O}$  effected 25% mortality. Cox *et al.* (1931) found 1,400 ppm Al reduced blood phosphorus and bone ash and bone phosphorus in guinea pigs and rabbits.

Quantitative estimates of the amounts of aluminum necessary to react with dietary phosphorus can be calculated from the reaction to form aluminum phosphate. When the aluminum:phosphorus ratio in the diet approached 1:1, Street (1942) using rats, found a 30% reduction in average daily gain accompanied by a reduction in the levels of plasma inorganic

phosphorus when aluminum hydroxide was used. This can be compared with a 92% reduction in average daily gain and a more pronounced reduction in plasma phosphorus, when rats received aluminum sulfate, a more soluble form of aluminum, to provide the same aluminum:phosphorus ratio.

If the same quantitative relationship holds for ruminants, the highest level of dietary aluminum (1,200 ppm) in the present experiment would be capable of combining with only 1,378 ppm of phosphorus, or 44% of total phosphorus present in the diet, assuming that all the aluminum present was reactive. This is unlikely since the pH and metabolic reactions in the rumen may have altered its reactivity. This would have left more than one half (56%), or about .18% dietary phosphorus which was perhaps enough to satisfy the minimum animal requirement for this element. Thompson *et al.* (1959) proposed that organic acids in the rumen may complex with aluminum, rendering it less available for phosphorus binding. Their hypothesis was based on a similar aluminum complex formed with organic acids produced by decomposition of organic matter in acid soil, which promotes a lower phosphorus retention by the soil (Struthers and Sieling, 1950).

Aluminum chloride was used in this study because it is a very soluble form of aluminum. Relatively insoluble aluminum forms such as the oxide, phosphate (Storer and Nelson, 1968) or hydroxide (Alsmeyer *et al.* (1963) have less effect on phosphorus absorption from the digestive tract.

A depression in serum inorganic phosphorus has been a frequent observation in nonruminant animals receiving high levels of dietary aluminum (Deobald and Elvehjem, 1934; Street, 1942). In the present study with cattle, there was no effect of dietary aluminum on plasma inorganic phosphate. Based on observed plasma values of these elements, it may be inferred that phosphorus absorption was not impaired to any extent. At the end of the feeding trial, aluminum levels in plasma showed no differences attributable to levels of dietary aluminum. Values found in this experiment are similar to those for humans (Kehoe *et al.*, 1940, .130 ppm aluminum; Seibold, 1960, .170 ppm aluminum).

Feeding aluminum in amounts up to 1,200 ppm did not increase the levels of this element in *longissimus* muscle and brain, but tended to increase levels in liver and kidney. Studies with

young and adult grazing cattle have shown that liver aluminum levels were related to geographic location (Mozgovaya and Arnatov, 1960). Iron, manganese, phosphorus, calcium and magnesium concentrations in tissues studied did not show differences due to increasing levels of aluminum in the ration. The observed changes in zinc and copper levels in the liver and kidney associated with dietary aluminum are difficult to explain with the information available about aluminum metabolism.

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