

EFFECT OF DIETARY COPPER ON DROUGHTMASTER STEERS

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SUMMARY

Droughtmaster steers were fed unrestricted for 93-99 d on diets of *Urochloa mosambicensis* or *Digitaria decumbens* hay containing 10 to 155 ppm Copper, or supplemented with copper sulphate to increase dietary levels to 249 ppm. The steers showed no symptoms of copper toxicity. When the copper was derived entirely from the forage, liver copper levels peaked at 28 days whereas the copper level in the livers of the group receiving  $\text{CuSO}_4$  increased during the whole period. Liver containing the highest concentration of copper, 449 ppm DM, were observed with cattle fed forage containing 155 ppm copper although the group supplemented with  $\text{CuSO}_4$  had a greater copper intake.  
(Key words: steers, Pangola, tissue, copper)

INTRODUCTION

The maximum intake of copper tolerated by cattle has not been well defined. It has been estimated that cattle can tolerate up to 100 ppm of dietary copper for an extended period (NRC 1980). Kidder (1949) induced symptoms of copper toxicity such as icterus, haemoglobinuria and death by administering 3g copper sulphate ( $\text{CuSO}_4$ )/day as a drench for 122 days. Chapman et al. (1962) administered the same dose in gelatin capsules without ill effect. The chemical form of the supplement administered appears to influence the toxicity of copper to cattle and studies to date have all used daily dosing of copper sulphate to induce copper toxicity.

Problems exist in disposing of effluent waters rich in heavy metals such as copper from piggeries and industrial plants (Poole et al. 1983). One solution could involve the removal of such metals in pasture plants harvested and fed to cattle. However little pertinent information on the effects of such a practice on the health of cattle and on heavy metal accumulation in the tissues is available. During studies of trace element metabolism, the opportunity arose to irrigate by both spraying and flooding areas of Pangola grass (*Digitaria decumbens*) with industrial effluent water containing appreciable levels of copper. Hay from these pastures was fed to cattle without and with copper sulphate ( $\text{CuSO}_4$ ) to determine the effects of a range of dietary copper levels on the accumulation of that metal in the tissues.

EXPERIMENTAL

Twelve Droughtmaster steers (305 + 45 kg) were treated with anthelmintic and individually housed on shaded concrete during a four week training period. During this period a steer refused feed and was removed from the experiment.

The steers were then randomly allocated to one of four hay diets, milled through a 2.5 cm screen, and they remained on these diets until slaughtered. No unirrigated Pangola hay was available, so the "control" group was fed *Urochloa mosambicensis* hay (Diet 1) with a moderate copper content. The other diets were flood irrigated Pangola [*Digitaria decumbens*] hay (Diet 2); spray irrigated Pangola hay (Diet 3) and flood irrigated Pangola hay sprayed 'before feeding with copper sulphate ( $\text{CuSO}_4$ ) (Diet 4) to provide an extra 236 ppm

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copper/kg DM of feed. The steers were fed daily, approximately 13% in excess of their daily dry matter intakes and provided free access to reticulated water which contained less than 0.002 ppm copper.

Feed intakes were recorded daily, while the animals were weighed and blood sampled weekly. Liver biopsies were taken prior to the trial and at days 28 and 56 of the trial. At day 70 the steers were placed into metabolism crates for a ten day adjustment period followed by an eight day balance study. Urine was collected into sulphuric acid. Faeces and urine were collected daily and subsampled for bulking over the eight day balance period. At the completion of the balance study the steers were returned to their individual pens until slaughtered between days 93 and 99 of the trial, Tissues were recovered for chemical analysis and histopathology.

Feed and faeces were dried at 70°C and ashed at 550°C. Nitrogen was estimated colorimetrically after microKjeldal digestion. Blood packed cell volume was measured by microhaematocrit and mean corpuscular fragility by erythrocyte resistance to haemolysis in isotonic saline. Tissue samples were freeze dried and elemental analysis carried out by atomic absorption after HNO<sub>3</sub> digestion. Data were analysed by analysis of variance using the method of least squares.

## RESULTS

Nitrogen levels in Urochloa and spray irrigated Pangola were appreciably higher than in the flood irrigated Pangola and this contributed to the significantly higher nitrogen intakes observed with diets 1 and 3 (Table 1), as the intakes of organic matter (OM) did not differ significantly.

Table 1 Production data from steers consuming varying levels of copper ( $\pm$  SD in parentheses)

	Diet 1	Diet 2	Diet 3	Diet 4
No. of steers	2	3	3	3
Duration (d)	98.5	99	93	99
<u>Feed composition</u>				
<u>(/kg dry matter)</u>				
Nitrogen (g)	8.65	5.54	8.46	6.21
Organic matter (OM) (g)	860	921	925	897
Copper (mg)	10	31	155	249
<u>Data from feeding experiment</u>				
OM intake (kg/d)	4.6 <sup>a</sup> (0.3)	3.7 <sup>b</sup> (0.8)	4.8 <sup>a</sup> (0.7)	4.0 <sup>b</sup> (0.4)
Nitrogen intake (g/d)	46 <sup>a</sup> (4)	22 <sup>b</sup> (5)	44 <sup>a</sup> (7)	28 <sup>b</sup> (2)
Weight change (g/d)	-64 <sup>a</sup> (4)	-79 <sup>ab</sup> (158)	97 <sup>a</sup> (71)	-239 <sup>b</sup> (71)
Copper intake (mg/d)	50 (4)	110 (25)	771 (147)	857 (58)
<u>Balance study</u>				
OM digestibility (%)	53 <sup>a</sup> (2)	56 <sup>a</sup> (6)	64 <sup>b</sup> (3)	56 <sup>a</sup> (4)
Nitrogen balance (g/d)	2.7 <sup>a</sup> (1.2)	-3.5 <sup>ac</sup> (3.4)	11.7 <sup>bd</sup> (3.0)	-5.6 <sup>bc</sup> (0.8)

Means in rows with different superscripts are significantly different ( $P < 0.05$ ).

During the whole feeding period, steers on spray irrigated Pangola gained weight, whereas weight losses were observed with the other steers, those receiving CuSO<sub>4</sub> losing significantly more than the

others. The balance study indicated that the spray-irrigated Pangola was significantly more digestible than the other diets, and steers on this forage showed a significantly higher nitrogen balance.

Despite the range of intakes of copper, from 50 to 857 mg/day, the steers showed no visual or histopathological evidence of copper toxicosis. The initial blood copper levels were 8.62, 0.81, 0.73 and 0.69 ppm and the final blood copper was 0.71, 0.70, 0.77 and 0.65 for diets 1, 2, 3 and 4 respectively while packed cell volumes and mean corpuscular fragility did not differ significantly during the trial.

A non-significant increase in liver copper was seen with increasing levels of copper intake (Table 2). With diets 1, 2 and 3 liver copper levels had attained maximal values by day 28 and remained at these levels for the duration of the trial. By contrast, in the diet containing copper sulphate, liver copper levels increased in a linear fashion for the 99 days conforming to the relationship

$$y = 10.3 + 4.0 x \quad (r = .956)$$

where  $y$  = liver copper (ppm DM) and  $x$  = days. The availability of copper from a ration can be expressed as fractional absorption (A) of dietary copper where

$$A = \frac{([\text{Initial} - \text{Final liver Cu}] \times \text{Liver DM})}{\text{Copper intake}}$$

Table 2 Tissue copper levels (ppm DM) from steers consuming varying levels of copper ( $\pm$  SE in parenthesis)

	Diet 1	Diet 2	Diet 3	Diet 4
<u>Liver copper levels</u>				
Initial	93 (107)	101 (28)	120 (50)	44 (23)
Day 28	161 (163)	294 (191)	456 (76)	50 (23)
Day 56	182 (122)	296 (186)	418 (106)	275 (41)
Final	161 (80)	319 (141)	449 (113)	399 (160)
<u>Final tissue levels</u>				
Skin	1.4 (0.2)	1.5 (0.7)	1.9 (0.4)	1.3 (1.3)
10th rib ash	3.9 (0.3)	4.5 (0.1)	4.7 (0.1)	4.6 (0.5)
Spleen	4.6 (2.9)	5.6 (1.7)	4.7 (1.9)	3.1 (0.3)
Kidney	13.4 (2.4)	14.5 (4.2)	16.8 (2.5)	15.7 (0.9)
Kidney fat	0.2 (0.1)	0.6 (0.2)	0.3 (0.2)	0.8 (0.6)
Thyroid gland	1.7 (0.2)	1.8 (0.2)	1.7 (0.3)	1.5 (0.2)
Pancreas	4.5 (0.2)	4.5 (0.4)	5.2 (1.0)	4.1 (0.5)
Adrenal gland	6.5 (1.6) <sup>a</sup>	3.1 (0.2) <sup>b</sup>	6.8 (1.2) <sup>a</sup>	7.8 (1.6) <sup>a</sup>
Heart	17.1 (1.0)	14.5 (0.6)	16.1 (0.9)	15.4 (0.6)
Lung	7.5 (0.8)	6.9 (0.9)	9.9 (6.8)	7.1 (0.9)
Spinal cord	5.2 (1.4)	4.2 (1.1)	5.0 (1.7)	4.8 (0.4)
Brain	9.9 (0.7)	13.2 (1.6) <sup>b</sup>	13.5 (2.4)	14.5 (3.3) <sup>ac</sup>
M. psoas major	2.9 (0.5) <sup>ac</sup>	4.7 (0.8) <sup>b</sup>	3.4 (0.5) <sup>ab</sup>	2.3 (0.7) <sup>ac</sup>
M. semitendinosus	2.3 (0.9)	2.3 (0.1)	3.4 (1.4)	3.4 (1.9)
Diaphragm	4.1 (1.0)	4.7 (0.2)	5.2 (0.4)	5.1 (1.6)

Means in rows with different superscripts are significantly different ( $P < 0.05$ )

The values of A ( $\pm$  SD) for the duration of the trial were .012 (.003), 0.011 (0.007), 0.004 (0.001) and 0.003 (0.001) for diets 1, 2, 3 and 4 respectively. These values of A for diets 1, 2 and 3 are of limited use because of the biphasic increase in liver copper with time. The values of A in the first 28 days were  $0.047 \pm 0.036$ ,  $0.071 \pm .058$  and  $0.014 \pm .005$  for diets 1, 2 and 3 respectively. The value of A between day 28 and trial end approached zero for diets 1-3. There were no significant changes in liver zinc and iron with time or dietary treatment.

The copper levels of tissues measured were significantly lower than those for the liver regardless of treatment. The only tissues to show significant differences between treatments were the adrenal glands and the Psoas major muscles; these differences appear to be unrelated to the-copper levels.

#### DISCUSSION

This experiment revealed that the source of dietary copper affected its pattern of retention in the liver, the sustained increase with copper supplied as copper sulphate contrasting with the biphasic retention of copper supplied as part of the forage. This observation casts doubts on the use of current methods to predict retention of copper in the liver. While the ARC (1980) absorption coefficient of 0.06 may be appropriate to determine short term changes in liver copper, copper retention could be grossly over-estimated over a longer period of time in situations where the copper is available only from forage. The present results suggest that a linear increase in liver copper levels may only be assumed when copper is added as a supplement.

The reasons for the biphasic retention of copper are not known nor are the reasons for the plateau levels of liver copper which differed between diets and which did not fully reflect the differences in levels of copper in the forage diets. Variations in levels of molybdenum and sulphur in the different forages could possibly be involved. Further, the fact that three of the four forages supplied submaintenance levels of energy and probably protein suggest that some caution in the widespread application of the present results is warranted. However it is of interest, in confirmation of the results of Chapman et al. (1962), that steers were able to consume a diet containing 249 ppm copper for 99 days and to achieve levels of 399 ppm copper in the liver without showing signs of copper toxicosis. Although the cattle showed no symptoms of copper toxicity, the liver copper levels were in excess of the National Health and Medical Research Council's maximum permitted copper concentrations in edible offal. All other tissue had copper levels below the regulation requirements.

Most studies with copper in cattle have been concerned with eliminating copper deficiencies by raising liver copper stores. This experiment investigated copper in excess of normal level in a hay diet and confirmed the steer's ability to tolerate high levels of copper intake. There was a marked difference in the response of the liver to copper from a "natural source" and a supplement of copper sulphate. There is a need for further study of these differences and of the role of copper offered in amounts higher than those currently recommended to overcome clinical copper deficiencies.

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