DELETERIOUS EFFECT OF DIETARY PHOSPHORUS SUPPLEMENT ON GRAZING CATTLE: A PHOSPHORUS-COPPER INTERACTION?

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SUMMARY

An investigation was made of the reasons why cattle grazing legume-grass pastures fertilised with superphosphate lost more weight in the dry season when fed mono-ammonium phosphate fertiliser than a complex mineral supplement containing an equivalent amount of P. Results were consistent with the theory that the poorer performance was associated with a depletion of liver Cu.
Keywords: grazing cattle, copper status, phosphorus supplement, Stylosanthes, trace elements

INTRODUCTION

Despite the widespread occurrence of dietary P deficiency in the cattle industry of northern Australia, there have been reports that cattle failed to improve liveweight (LW) gain or suffered accelerated LW loss in response to P supplementation (Winks 1986). A recent instance occured in an experiment (Miller and Webb 1988) in which steers fed a supplement of mono-ammonium phosphate (MAP) had mean LW changes of -77 and -44 kg in two dry seasons (1984 and '85) compared with -36 and +4 kg, respectively, for steers fed a P supplement containing trace elements. Both supplements gave substantial improvements in LW gain in the corresponding wet seasons. Investigations were, therefore, made in the third year to determine whether the inferior performance of MAP-fed steers during the dry season was associated with an induced deficiency of one of the elements in the complex supplement.

EXPERIMENTAL

The experimental site was on an infertile, red duplex soil, uncleared, elevation 580 m, near Mareeba, annual rainfall (summer distribution) about 800 mm. The native pasture was dominated by kangaroo grass (Themeda triandra), giant spear grass (Heteropogon triticeus) and native sorghum (Sorghum spp.). The experimental design tested the effects of introducing legumes (Stylosanthes scabra cv. Seca and S. hamata cv. Verano) and fertilisation with superphosphate at four levels (F1-F4; Miller and Webb 1988), in 12.5 ha paddocks stocked with three cross-bred zebu steers aged two to three years. This report concerns a subset of the experiment in which responses to supplementation were tested at the lowest level of fertiliser (F1) with legume. Supplementation was with either MAP fertiliser (equivalent to 5 g P/hd/d, fed twice weekly) or a mineral block (ad libitum) which provided supplementary Ca, Cu, K, Mg, Mn, Na, P, S and Zn ("Ultraphos"; Coopers-ICI, Townsville, Qld). Each treatment was replicated once. The draft of animals was changed annually in April/May. The '86 draft initially weighed 327±5 kg (n=48). Water was continuously supplied from nearby Collins weir. The animals were mustered for measurements every 28 days.

Jugular blood was collected into heparinised tubes specified for trace element determinations (Becton Dickinson, Rutherford, N.J. *07070, u*.S.A.).

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Parotid saliva was collected by suction, faeces from the rectum, liver by biopsy (Loosmore and Allcroft 1951) and kidney from a steer that died by accident. Pasture yield and composition was estimated by established methods. Mineral content was determined on separated leaf and stem fractions from green grass and legume. Samples were ashed in silica crucibles, digested with H_2O_2/HNO_3 and taken up in 2M HCl except for Mo analysis, when samples were ashed at 400°C, treated with HF to remove silica and extracted with 8-hydroxyquinoline in methyl isobutyl ketone (L.L. Conlan; unpublished). Copper, Fe, Mo, Mn, Na, K and Zn were determined by atomic absorption spectrophotometry, Cd in water, kidney and faeces by digital voltammetry and faecal Cd, Cu, Fe, Mn and Zn by ICP-emission spectroscopy after tri-acid digest. Serum sulphate was estimated nephelometrically. Statistical analysis was by multiple regression, using indicator variables for treatment effects (Daniel and Wood 1980). Homogeneity of variance was tested.

RESULTS

This report concerns the responses to supplementation during the '86 dry season (May - Oct.), unless otherwise noted. Whereas average Stylo. content of the pasture had not exceeded 5 % DM during '84 and '85, in '86 it rose to a dry season mean of 25 %. Concentrations (% DM) in green pasture at May, '85, averaged, for K, 1.10; Mg, 0.12; P, 0.065; S, 0.073 and (ug/g DM) for Mn, 149; Zn, 25. There were appreciable differences between grass and Stylo., respectively, for Ca (0.31 and 1.61 % DM), Cu (2.8 and 4.9 ug/g DM) and N (0.33 and 1.42 % DM). At July, '86, grass and Stylo. Mo were similar and averaged 0.26±0.03 ug/g DM.

Loss of LW in the '86 dry season for MAP supplemented steers (19±6 kg, n=6) was not significantly less than for steers fed Ultraphos (12±7, n=6) or unsupplemented controls (37±10, n=5). Supplement increased ($P<0.02$) plasma inorganic P from 3.78±0.09 (n=5) to 5.1±0.8 for MAP and further ($P<0.01$) to 6.8±0.5 (n=6) for Ultraphos in July. At the same time, plasma Ca was depressed ($P<0.01$) from 10.02±d0.11 mg/dl (n=5) to 9.39+0.14 (n=6) by Ultraphos but not by MAP (9.81±0.13, n=6) whereas plasma Mg was unaltered at 2.32± 0.04 (n=17). Tissue, faecal and saliva samples were obtained in Oct. No significant supplement effects were observed for serum inorganic sulphate (42±11 mg S/l, n=16), plasma or liver Zn (0.708±0.023 mg/1 and 126±20 ug/g DM, n=18, respectively), despite presence of these elements in Ultraphos. Liver Mn increased in response to the Mn in Ultraphos ($P<0.001$) from 12.0±0.9 ug/g DM (n=3; unsupplemented) and 11.7±0.5 (n=4; MAP) to 20.3+6.2 (n=6). Liver Fe concentration was unaltered by MAP (571±54 ug/g DM, n=6). Selected comparisons were made of faecal minerals for unfertilised, highly fertilised and MAP supplemented but not Ultraphos treatments. No significant differences were found for faecal levels (ug/g DM) of Cu (7.28±0.15), Zn (52.1±2.7), Mn (348±14) and Fe (138+29, n=12) and Cd was not detectable in any of these samples by ICP (detection limit 4 ug/g DM). Cadmium was not detectable by digital voltammetry in drinking water (detection limit 2 ug/1), kidney (d.l. 0.05 ug/g DM) and pooled faeces (d.l. 0.25 ug/g DM). Concentrations of all other elements in drinking water were negligible. Plasma Cu concentrations are shown in Fig. 1 for unfertilised pasture and the F1 and F4 treatments. Liver Cu (Fig. 1) and saliva Na/K values required logarithmic transformation to correct non-normality and heterogeneity of variance and hence, geometric means are reported for these parameters without error estimates. The molar Na:K ratio in parotid saliva was increased ($P<0.001$) from 5.0 (n=6) in unsupplemented animals to 17.5 (n=6) in those receiving Na in Ultraphos.

DISCUSSION

The most striking result was the dramatic depression of liver Cu in animals on the fertilised treatments compared with unfertilised and the significant further depression of liver Cu concentration by the MAP supplement (Fig. 1). This depression occurred despite a high Stylo. and, therefore, increased Cu content of the pasture. The unfertilised controls were in extremely poor condition, having lost approximately 29 % of initial LW when liver was biopsied (unpublished). It is surprising, therefore, that liver Cu levels in these animals were normal, while plasma Cu was low (Fig. 1). Plasma Cu but not faecal Cu levels were increased by fertiliser + Stylo. There was no extra effect on plasma Cu by P supplement, either alone or with Cu, resolveable from the data. It is presumed, therefore, that plasma Cu levels responded to the additional Cu contained in the Stylo., which is consistent with previous findings that Cu in plant material has a higher availability to the animal than Cu in inorganic supplements (Schlink and Hoffmann 1986). It is, similarly, presumed that some factor in the fertilised Stylo. and MAP promoted loss of liver Cu without inhibiting Cu absorption.

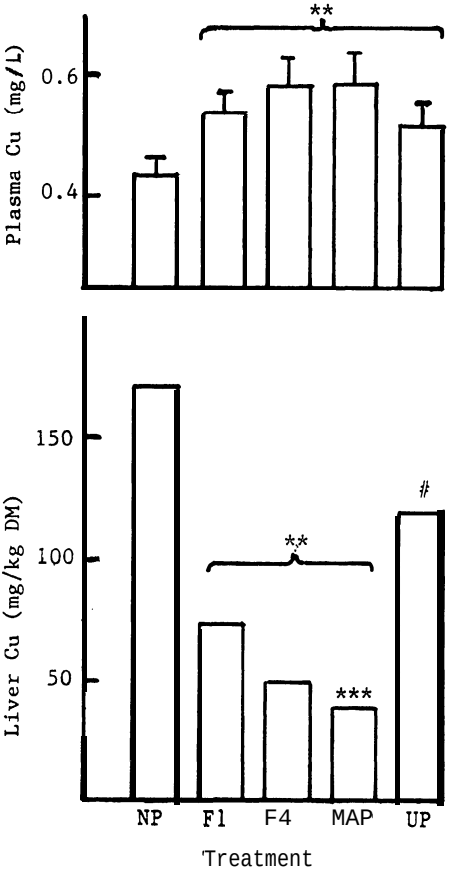

Fig. 1. Concentration of Cu at the end of the dry season (Oct., '86) in liver (geometric mean) and blood plasma (mean ± S.E.) from steers grazing unimproved native pasture (NP), legume + least fertiliser (F1) or most fertiliser (F4) and F1 + monoammonium phosphate supplement (MAP) or a complex mineral supplement (UP). Means are for six steers, except NP, which is for 12 animals. ** Fertilised differs from unfertilised: $P<0.01$; *** MAP < fertilised unsupplemented: $P<0.001$: # UP > MAP; $P<0.001$

The primary limitation to animal production during the dry season was probably N, rather than P content of the pasture. However, the negative response to MAP during the first two dry seasons implies that some other element was also involved. Unfortunately, the negative response to MAP did not recur during our detailed studies in the third year, when there was a large increase in **Stylo.** in the pasture. Consequently, the cause of the deleterious effect of MAP cannot be identified; It is speculated, however, that a greater drop in liver Cu than that seen here (Fig. 1), may have occured in previous years when there was less Cu-containing **Stylo.** in the pasture. It is possible, therefore, that the deleterious effect of MAP in previous years was due to an induced Cu deficiency.

The mechanism of the reductions in liver Cu reported here is unclear. Biochemical analyses eliminated dietary deficiencies of Fe, Mg, Mn, S and Zn but suggested Na deficiency. However, similarity of LW response to MAP and Ultraphos (containing Na) in the ensuing wet season (unpublished results) suggest that Na intake was not limiting. The simplest hypothesis is that loss of liver Cu was due to a component common to fertilised **Stylo.** and MAP. Possible antagonisms to Cu were investigated. Lack of treatment effects on levels of faecal Fe, Mn and Zn, serum sulphate and pasture Mn, Mo and Zn suggest that antagonism of Cu by dietary excess of these elements was not involved. Similarly, Cd, which contaminates phosphatic fertilisers (including MAP), is taken up by pasture plants (Williams and David 1973) and induces loss of liver Cu (Mills 1974), was undetectable in the drinking water, faeces or kidney, making it unlikely that antagonism of Cu by Cd was involved. Although the higher Ca level in **Stylo.** than grass could account for the reduction of liver Cu due to fertiliser, it could not account for the MAP effect (Fig. 1). We therefore propose that the significant depression of liver Cu levels by fertiliser or supplement (Fig. 1) was due to increased dietary **P.** This hypothesis is currently being tested with pen-fed animals in our laboratory.

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