

Copper & Deer Growth

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1. INTRODUCTION

The role of copper in animal metabolism has been reviewed recently (Killorn and Wilson, 1990), but there is little data available specifically relating to copper metabolism and requirements in deer. Low blood and/or liver copper concentrations are a common finding in deer from throughout New Zealand (Anon. 1990). In a survey of livers collected from deer slaughter premises in the South Island, Harrison *et al* (1989), reported that more than 40% of liver samples had a copper concentration of less 100 μmol per kg. There is a range of clinical syndromes reported to be associated with copper deficiency in deer (Wilson, 1989), but most reports are anecdotal. Enzootic ataxia (Barlow *et al*, 1964, Wilson *et al*, 1979, Mackintosh *et al*, 1986) is the only condition in which there has been a clear association with low copper levels. More recently there is evidence of hind leg joint abnormalities in deer shortly after birth (Anon 1990). However, poor growth is one of the predominant syndromes reported by veterinary practitioners to laboratories when requesting copper analyses in deer.

While poor growth has long been recognised as an effect of low copper in cattle (Cunningham, 1946) and more recently in sheep (Hogan *et al*, 1971, Whitelaw *et al*, 1979), there have been no published reports of weight gain responses in deer investigated by properly constructed and analysed trials. Indeed (Wilson, 1989) was unable to show a significant weight gain response to copper therapy on a severely copper deficient property. (Lawrence, 1987) also reported a nil response in weight gain after copper therapy, although levels in that trial were barely deficient at commencement.

Growth response trials are difficult to conduct and often give confusing and conflicting results (Phillippo, 1983) summarised the literature on 23 growth response trials conducted in cattle and noted that a positive response was reported in only 8. Some copper supplementation trials give a negative growth response (Reid and Shannon, 1987).

Recent developments in our understanding of copper deficiency in domestic animals have been summarised by (Suttle, 1986(a)(b)) Suttle introduced the concept of "dysfunction", which is the state manifest as disease, after depletion of stores as a result of reduced absorption or availability of copper, followed by a reduction in circulating concentrations of copper. Once circulating copper levels become depressed, tissue systems which require copper fail to function normally and clinical disease occurs.

In deer herds, blood copper levels usually vary considerably (Wilson, 1989 and unpublished data). In adult deer enzootic ataxia has been observed only in deer with liver copper concentrations of less than 60 $\mu\text{mol}/\text{kg}$ (Mackintosh *et al*, 1986) Yet many unaffected deer are observed on copper deficient properties with levels below 60 $\mu\text{mol}/\text{kg}$, but show no signs of disease. Such

individual variation may be genetic. Wapiti appear more susceptible to copper deficiency than red deer (Mackintosh *et al*, 1986).

If low serum copper is a pre-requisite of functional deficiency, it is likely that only a proportion of the herd will show signs of deficiency at any one time. Thus if deer are randomly allocated to treated and control groups for a growth response and copper supplementation trial, many in the control group may have blood copper levels considered adequate (Wilson, 1989). This suggests that trials may need to be designed or data examined other than by methods simply comparing mean body weights between groups.

2. **TRIAL OBJECTIVES**

The purpose of this trial has been twofold

- (a) to attempt to elicit growth rate responses in copper deficient weaner deer by treating them with oxidised copper needles, and
- (b) to ascertain whether growth rate is dependant on the copper status of the individual animal i.e. to determine if there is a blood copper level at which a response to copper treatment can be expected.

3. **METHOD**

Four farms with a previous history of low copper levels in deer were selected. Weaner stags and hinds on these farms were randomly allocated (within sex) to treatment and control groups as shown in Table 1. The treatment group received ¹10 gm oxidised copper wire (90% available copper), the control group was untreated.

Table 1: Allocation of animals on four farms to treatment and control groups

		Farm 1	Farm 2	Farm 3	Farm 4	Total
Weaner Stags	Treatment	18	11	7	20	56
	Control	17	10	7	20	54
Weaner Hinds	Treatment	6	23	59	-	79
	Control	5	22	52	-	79
	TOTAL	46	66	116	40	268

Treatments were administered on farms 1, 2 and 4 in May 1989 and again in August 1989, and on Farm 3 during August 1989. The trial animals were weighed at regular intervals throughout the trial period, and blood samples were collected and serum copper levels measured by atomic absorption spectrophotometry.

¹ Cuprax 10g capsules, Pitman-Moore, Upper Hutt

4. **RESULTS**

4.1 **Body weights**

Mean body weights of hinds and stags in the treatment and control groups are shown in table 2, and the average daily liveweight gains for hinds and stags are shown in figure 1.

Table 2 Mean Bodyweights of Weaner Hinds and Stags (kg) and Total Liveweight Gains over the trial period May 1989 - Feb 1990

		May*	June	July	Aug*	Sept	Oct	Nov	Dec	Feb	Total Liveweight Gain (kg)		
Farm 1	Female	C	38.6	42.7	-	43.3	45.1	-	55.6	-	70.6	32	
		T	35.3	39.8	-	41.4	44.1	-	54.9	-	68.4	33.1	
	Male	C	45.7	50.2	-	50.3	53.1	-	64.8	-	81.8	36.1	
		T	43.2	48.3	-	49.3	52.1	-	65.2	-	83.4	40.2	
Farm 2	Female	C	51.1	52.6	55.1	57.7	59.2	65.5	71.4	75.6	84.4	33.3	(7.8)
		T	54.2	56.1	58.0	58.9	60.8	67.5	73.0	78.1	86.8	32.6	(8.6)
	Male	C	57.1	60.6	63.4	67.5	71.3	81.8	89.4	96.4	104.8	47.7	(14.3)
		T	58.7	61.5	65.7	70.2	73.0	84.1	91.4	98.9	107.0	48.3	(13.9)
Farm 3	Female	C	-	-	-	56.5	59.3	64.9	-	-	-		(8.4)
		T	-	-	-	56.3	59.8	64.9	-	-	-		(8.6)
	Male	C	-	-	-	70.6	74.1	81.9	-	-	-		(11.3)
		T	-	-	-	66.7	71.7	80.0	-	-	-		(13.3)
Farm 4	Male	C	54.6	59.6	59.5	60.5	62.0	74.9	84.8	91.6	96.6	42	(14.4)
		T	53.5	57.7	59.2	59.5	61.9	74.5	84.1	90.3	96.9	43.4	(15)

(Bracketed figures give comparisons between farms over similar periods)

C - Control T - Treated * Copper treatments given May and August

Preliminary analysis of the data has been carried out using analysis of variance, and relationships between average daily liveweight gain and serum copper level or change in serum copper level have been calculated using linear regression

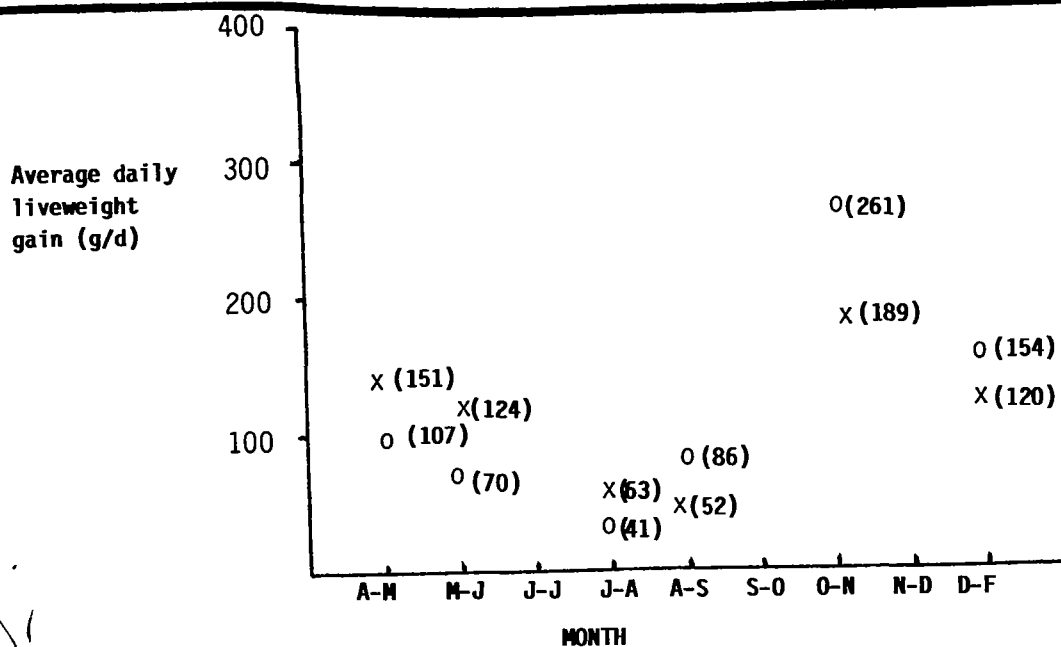
Differences were sought between sexes, farms, treatments and sampling periods, as well as over the whole trial period.

Farms: There were highly significant differences between farms for average daily liveweight gain over time with a significant farm/time interaction.

Treatments: There were no significant differences in average daily liveweight gains between treatments.

Differences between sexes: As expected, there were significant differences between the average daily liveweight gain of hinds and stags. There were also significant differences over time, as well as a significant time/sex interaction. Intriguingly, females grew faster than males in autumn, but not during spring

Figure 1 Changes in average daily liveweight gain in weaner stags and hinds over time. x = hinds, o = stags. Figures in parentheses are actual means).



4.1.1 Individual Farms:

Farm 1: There were indications that treatment in May improved average daily liveweight gains over the period May-August ($P = 0.0849$)

Farm 2: There were no significant differences between treatment groups at any time during the trial

Farm 3: Following treatment in August, there was a significant difference in growth rates of the hinds for the period August-September ($P = 0.0463$)

Farm 4: There was a significant difference in growth rates for the period June-July ($P = 0.0263$).

4.1.2 **Combined Farms:** There was a significant difference in growth rates between the treated and control groups on all farms for the period June-July ($P = 0.0488$).

4.2 Serum Copper Levels

Mean serum copper levels of both weaner hinds and stags throughout the course of the study are presented in table 3

Differences Between Sexes

There were no statistically significant differences in serum copper levels between the sexes

Farms

There were highly significant differences between farms for serum copper levels over time with a significant farm/time interaction.

Table 3 Mean Serum Copper Levels ($\mu\text{mol/l}$) of weaner hinds and stags (T = Time of Treatment)

		April	May(T)	June	July	Aug(T)	Sept	Oct	Nov	Dec	Feb
Farm 1											
Female	C	10.49	8.00	2.32	-	5.02	8.85	-	4.25	-	3.74
	T	4.51	4.91	6.97	-	10.05	14.27	-	9.14	-	7.05
Male	C	7.38	7.16	3.58	-	4.05	4.77	-	6.62	-	5.56
	T	5.21	6.74	8.14	-	10.28	10.63	-	10.4	-	8.18
Farm 2											
Female	C	12.28	10.03	-	7.14	7.19	7.29	12.53	-	5.64	14.98
	T	8.59	9.66	-	11.2	7.16	8.4	13.67	-	5.63	15.25
Male	C	9.06	10.86	-	5.93	7.58	7.08	8.07	-	3.83	15.49
	T	9.14	9.86	-	6.02	9.07	11.1	14.08	-	4.12	16.62
Farm 3											
Female	C	-	-	-	-	7.06	9.98	15.09	-	-	-
	T	-	-	-	-	7.97	8.38	12.54	-	-	-
Male	C	-	-	-	-	5.35	7.88	9.79	-	-	-
	T	-	-	-	-	7.25	10.87	13.51	-	-	-
Farm 4											
Male	C	11.62	13.34	5.39	6.78	7.88	10.64	11.19	13.73	-	10.65
	T	12.21	13.22	7.24	9.53	10.23	15.38	11.19	14.55	-	11.43

Treatments

There was a significant effect of treatment on serum copper levels between treated and control groups.

4.3 Relationship Between Growth Rate and Serum Copper Levels

There were no significant correlations between average daily liveweight gains and serum copper levels or the change in serum copper levels overall

5. DISCUSSION

The preliminary analyses of data conducted to date show that overall there were no significant growth rate responses to copper supplementation, but that during the winter (June/July) the treated deer grew significantly faster than untreated deer. The latter response was not of great enough magnitude to influence the overall effect in the 10-month trial period. It is notable that copper levels generally were lowest during the June/July period. In a previous trial Wilson (1989) was able to show that from April to November a 3.1 Kg liveweight gain was achieved in treated deer, but this also failed to achieve statistical significance. The blood copper levels measured in the study of Wilson (1989) ranged from means of 2.8 - 3.8 $\mu\text{mol/kg}$ in untreated deer, whereas those in treated deer ranged from 8.6 - 9.5 $\mu\text{mol/l}$. Thus, the levels reported by Wilson (1989) are lower than those reported in the present study

Treatment in most cases resulted in copper levels which were in excess of the now accepted standard of 8.0 $\mu\text{mol/l}$ (Mackintosh *et al*, 1986). However, on occasion, treatment was insufficient to elevate mean levels above 8.0 $\mu\text{mol/l}$. Thus in many situations treated deer still had copper levels substantially below

8.0 $\mu\text{mol/l}$ and this could potentially have reduced the mean growth rate in treated deer. Conversely, a number of deer in untreated control groups had blood copper levels in the normal range. This may well have elevated the mean growth rate in control deer. These two effects working concurrently may have reduced the difference in mean bodyweight gain between treated and control groups and prevented attainment of statistical significance when comparing mean growth rate responses.

Failure of regression analyses to show bodyweight gain effects at different blood copper levels has several possible explanations. Firstly, copper in deer may not influence growth. However, given the evidence for copper being involved with growth in sheep and cattle this is unlikely. A second explanation is that to inhibit with growth in deer, blood copper levels may need to be exceedingly low. There may have been insufficient numbers of deer with copper levels low enough to affect growth rates and to have produced a statistically significant response in the present trial. Further investigation of this possibility could be achieved by selection of farms with lower copper levels than those selected for the present study and to select animals for treatment on the basis of their blood copper levels prior to commencement of the trial. Deer with high blood copper levels at the commencement could be treated with copper to ensure that the difference in copper level between treated and control groups was very wide and that there were sufficient animals at very low levels to produce a response. Another method may be simply to not treat any animals at all and hope that there is sufficient range of blood copper levels to analyse data by regression.

A third method of re-assessing whether copper influences growth would be to house animals on copper sufficient and copper deficient diets to ensure consistently adequate or severely deficient copper levels and to monitor weight gains.

Thus, field trials conducted to date have been unable to prove a relationship between copper level and growth. Clearly more research work needs to be conducted in this field since there is a substantial concern in the deer industry and in the veterinary profession about the extent of copper "deficiency" when conventional "normal levels" are used. Existing "normal levels" have been based on observations of deer one year of age or more and the only relationship between low copper and a clinical entity that has been proven is that of enzootic ataxia which occurs only in deer with liver copper levels of less than 60 $\mu\text{mol per kg}$. It may well be that for weaner deer, which appear unaffected by enzootic ataxia a different set of "normal levels" may need to be established.

It is likely to be considerable time yet before the farmer and veterinarian have an objective assessment of required copper levels for growth. Only then can it be calculated whether or not the farmer will achieve an economic response to copper supplementation.

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