TRACE ELEMENTS IN DEER

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Introduction

Trace element deficiencies have been recognised in ruminant livestock in New Zealand for many years. Symptoms of the deficiency conditons have been reported and control and therapy for these disorders have proved to be effective. The most significant advance in the recognition of trace element deficiencies has been the abilty to analyse tissue and blood samples and so determine the mineral status of the animal. This has necessitated the correlation of symptoms with measured mineral status and subsequent responses to the appropriate therapy. The reliability and accuracy of the diagnosis resulting from analyses is a function of the number of measurements which have been correlated. Trace elements for which deficiencies are of consequence in sheep and cattle production in N.Z. are copper, cobalt, selenium and iodine. With the advent of agricultural diversification and the intensive farming of other ruminant species such as goats and deer, there has been a tendency to adopt trace element reference levels for sheep and cattle and apply them to the other species. In the absence of data it is a logical and necessary development, but extreme care should be taken before making strong assumptions about relationships between trace element status and the symptoms observed in animals. Where there are no animal remedies specifically licensed for use in a particular species, it behoves the veterinarian to exercise extreme caution before recommending the use of any products to control or prevent disease.

Present Status

Current reference values for trace elements in red deer (Table 1) have been compiled from data presented by McAllum (pers. comm.) and Grace (1982). These are based on values from other species.

Table 1 Trace element levels for red deer

	Liver levels		Blood levels	
Selenium (*)	nmol/kg	ug/kg	nmol/l	ug/l
responsive	<250	<20	<130	<10
marginal	250-450	20-35	130-250	10-20
adequate	>450	>35	>250	20
Vitamin B12 (**)	nmol/kg	mg/kg	pmol/l	ug/l
responsive	<110	<0 15	<185	<0 25
marginal	110-220	0 15-0 30	185-370	0 25-0 5
adequate	>220	>0 30	>370	>0 50
Copper (***)	umol/kg	mg/kg		
responsive	<45	<3		
marginal	45-95	3-6	not recommended	
adequate	>95	>6		

Adapted from McAllum (pers comm) and Grace (1982)

(*) for sheep and cattle (and probably deer)
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The only available published data on trace element levels in the livers of deer in N.Z. are those of Reid, McAllum and Johnstone (1980) and they are for copper. This information is presented in Table 2.

Table 2. Observed liver copper values in N.Z. red deer.

	Farmed deer			Feral
	Foetus/Neonate	Immature	Adult	
umol/kg	5589	360	165	642
mg/kg	356	23	10.5	40.9

Adapted from Reid, McAllum & Johnstone (1980)

Survey of A.H.D. (Lincoln) data

Survey In order to assess the present situation, we have examined, with the cooperation of staff at Lincoln Animal Health Laboratory, all their records from March 1980 to January 1984 in which trace element analyses in deer were requested. This laboratory services the northern part of the South Island. To address the question initially, trace element deficiency was defined as those concentrations considered to be responsive to treatment in cattle and sheep (Grace, 1982), or where there was post-mortem evidence consistent with a trace element deficiency.

The review addressed the following questions:

1. Which trace elements appear to be 'deficient' in deer?
2. In which age group are 'deficiencies' most common?
3. At what serum and/or liver concentrations are the 'deficiencies' clinically evident?
4. In what regions of the northern South Island are the 'deficiencies' most prevalent?

Results One hundred and thirty requests for trace element analyses were examined comprising 700 samples from 500 deer. The following values give the distribution of those requests and the classes of deer involved:

trace element - copper (Cu) 66%, vitamin B12 22%, selenium (Se) 12%;
class of deer - hinds 43%, stags 21%, calves 16%, class not stated 19%;
region - over 50% from Nelson/Westland area.

Copper 38% of cases (33/86) examined for Cu had samples 'deficient' in Cu (less than 4.5 umol/l serum or less than 45 umol/kg liver), and 12% of cases (10/86) had Cu levels that would be considered marginal for adult cattle (4.5 - 8.0 umol/l serum or 45 - 95 umol/kg liver). 42% of the 'marginal' or 'deficient' samples were from hinds, 11% from stags, 16% from calves and 31% from animals of unreported age and/or sex. Ataxic animals were reported in 27% of the cases (23/86), and of these, 16 had Cu levels which were 'deficient' or

'marginal', and 7 had Cu levels within the 'normal' range
Of the 43 cases with 'deficient' or 'marginal' samples, 24 were from the Nelson region (8 of these from one property and 3 from another), 9 from Westland, 8 from South Canterbury and 2 from North Canterbury.
There had been a number of requests to test for Cu after the administration of Cu injections (cattle dosage). On at least two Nelson properties, responses to the injections were variable. On one property a hind was ataxic and seven other hinds were 'deficient' in serum Cu six months after Cu injections. Three months later no detectable Cu could be found in the sera of some of these deer. A second injection of Cu was then given and one month later half of the deer still had sera 'deficient' in Cu. Two months after a third injection, about one half of the deer remained 'deficient' and ataxic deer were still seen on the property. During 1983, this same herd of deer was given additional Cu injections. The deer had adequate serum Cu levels two months after supplementation, but no measurable Cu could be detected in their sera three months after the supplementation (n = 15).
A second property in the same region had a similar problem. Six fawns were 'deficient' in serum Cu levels three months after Cu injections and when grazing pastures which had been topdressed with Cu. Only after a third injection five months later did all serum samples analysed contain 'adequate' Cu.
Two cases were reported which appeared to be suggestive of Cu poisoning.

Cobalt - vitamin B12 Vitamin B12 'deficiency' (vitamin B12 levels less than 185 pmol/l serum and less than 110 nmol/kg liver) was diagnosed in seven (5.4%) cases measured and was 'marginal' (vitamin B12 levels 185 - 370 pmol/l serum and 110 -220 nmol/kg liver) in another 4 (3%) cases. No symptoms other than poor growth rates were recorded in these cases. In one case both Cu and vitamin B12 serum levels were in the 'deficient' ranges.

Selenium One case reported very high serum selenium levels. There were no cases with selenium levels below the values accepted as 'normal' for sheep and cattle.

Discussion of survey The survey showed that:

1. Cu 'deficiency' is the most frequently reported trace element anomaly in the northern S.I. region.
2. Cu and vitamin B12 'deficiencies' occurred in all age groups and in both sexes.
3. The only signs of possible vitamin B12 'deficiency' were poor growth and/or poor condition.
4. Where there was clinical evidence for Cu deficiency (ataxia), most of the samples submitted for analysis were within the ranges recorded as 'deficient' or 'marginal' in cattle, and a number of these cases also had extensive demyelination of spinal cord tissue. Some of the ataxic deer which had adequate Cu levels may have been affected with rye-grass staggers as suggested in the submission reports.
5. There was insufficient evidence to indicate whether areas where trace element deficiencies in sheep and cattle have been reported corresponded to those areas where 'deficiencies' were reported for deer.

Copper deficiency

Value of serum/liver values In deer copper appears to be the trace element of major concern to both the veterinarian and the farmer. Existing data on liver Cu concentrations suggest that farmed deer maintain lower liver Cu levels than do feral deer, and measurements of liver Cu in these deer have produced values (see Table 2) markedly lower than is normally expected in sheep or cattle (Reid et al, 1980). Reid and his co-workers (1980) also observed that despite the low adult liver Cu levels, offspring appeared to be extremely well protected against low Cu status at birth. These observations together with other data available, such as the survey mentioned earlier, would indicate that caution must be applied to any extrapolation of data on Cu metabolism from sheep to deer.

The problem with Cu deficiency is not simply one of low dietary Cu intake. Other dietary constituents, particularly sulphur (S) and molybdenum (Mo), can have a marked effect on Cu metabolism and requirement as has been shown in sheep and cattle.

Lincoln College trial A recent trial at Lincoln College (Freudenberger, Familton & Sykes, unpublished) attempted to examine Cu depletion and repletion in deer and sheep. By using silage based diets enhanced with the Cu antagonists S and Mo, very low liver Cu levels (16ug/kg liver DM) could be induced in deer. Deer (4) and sheep (4) were offered an unsupplemented silage diet, containing Cu 7ug/kg DM, S 3.1 g/kg DM and Mo 1.9 mg/kg DM, for 10 weeks. Further groups of sheep and deer (6 of each species) were offered the same silage but with additional S (3g S/kg DM) and Mo (4.8mg Mo/kg DM) to reduce Cu availablity further (S-Mo supplemented silage). Liver biopsy samples were taken initially and at 6 and 11 weeks after the trial had commenced. The rates of loss of liver Cu (ug/g DM/day) were 0.27 and 0.81 in deer and 3.12 and 4.01 in sheep on unsupplemented and S-Mo supplemented silage, respectively. The very much slower rates of Cu loss in deer may reflect the fact that liver Cu stores in sheep were initially 10-fold higher than in deer, even though the animals were from the same property. Moreover, within each species there was a highly significant linear relationship between initial liver Cu concentration and the rate of decrease of concentration. However the slope of this relationship was different in the two species (see Fig 1). It was also noted that deer did not develop insoluble Cu complexes in plasma to the same extent as sheep, which was suprising considering that the basal diet and supplementation were similar for the two species. This suggests, perhaps, that there is a fundamental difference between sheep and deer in metabolism of S and Mo and may explain the differences observed in the relative losses of liver Cu.

One important similarity between sheep and deer which is of importance to the veterinarian is that there appears to be little correlation between plasma Cu levels and liver Cu status in deer. Cu values for blood samples (n=39), collected at the same time as liver biopsies were obtained, are plotted

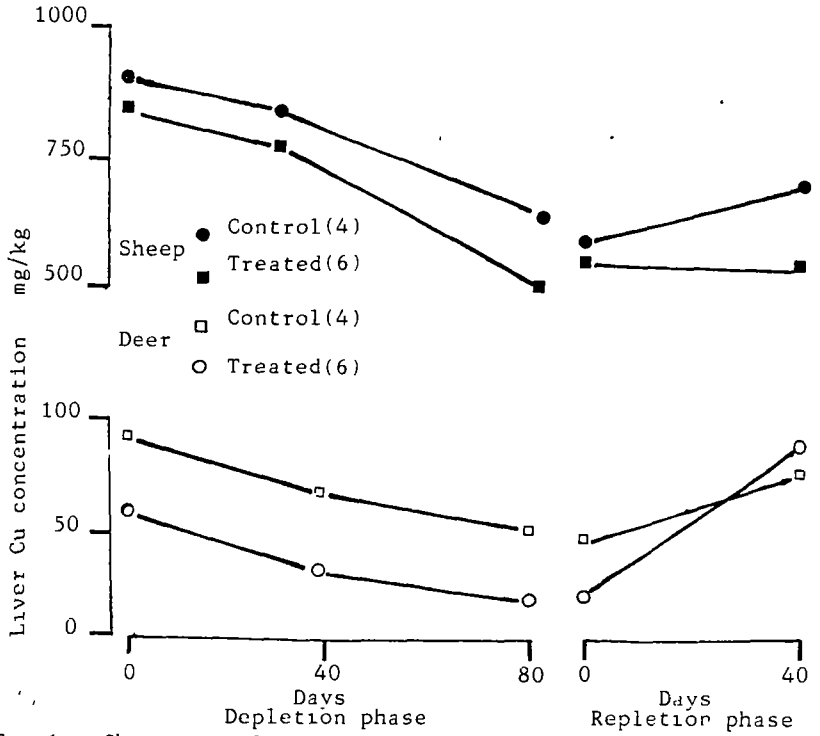

Fig 1 Changes in liver copper values obtained from sheep and deer during the Lincoln College depletion repletion trial (during repletion all groups contained five animals).

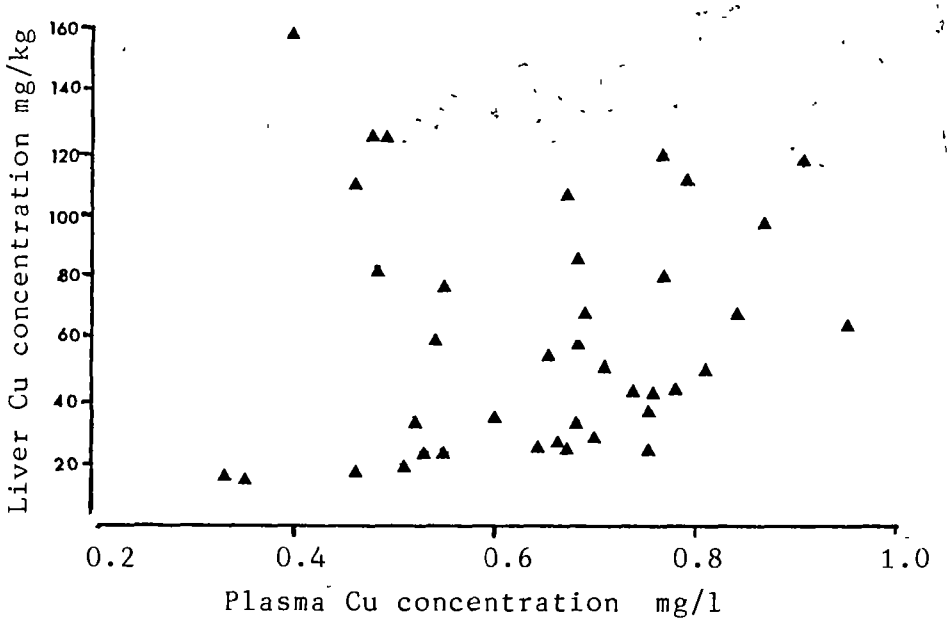

Fig 2: Relationship between liver and serum copper obtained from 10 deer on four occasions during the Lincoln College trial.

against the values from liver in Fig.2.

Enzootic ataxia Enzootic neonatal ataxia in lambs is associated with depressed Cu levels in both the lambs and their dams and appears to be associated with lowered activity of cytochrome oxidase in brain tissue (Underwood, 1971). It has been suggested by Howell et al.(1964) that the lesion may be one of myelin aplasia rather than of demyelination. The condition can be prevented by administration of Cu to ewes.

A condition resembling enzootic ataxia has been reported in deer in N. Z. (Wilson, Orr & Key, 1979) and other countries (Barlow & Butler, 1964). This syndrome occurs in older animals and is characterised by a spongy change and a deficiency of myelin in the dorso-lateral columns of the spinal cord. Although Cu 'deficiency' has been associated with this condition the cause is uncertain (Hartley, 1983). The evidence is inconclusive and low Cu status is probably not the only factor involved in the aetiology (Barlow, 1980; McTaggart et al., 1981).

Attempts have been made by Mason et al.(1984) to reproduce the disease by increasing the dietary intake of Mo and S, but no evidence of ataxia or change in the plasma Cu concentration was noted. The numbers of animals (2) and the failure to measure liver Cu make definitive conclusions difficult. Nagy, Chappell and Ward (1975) concluded that mule deer were more tolerant to increased dietary Mo than sheep or cattle by at least a factor of 10, but this trial was conducted over a short period of time and no mention was made of the copper status of the animal. It is obvious that further long-term depletion trials are required before it is possible to speculate on the aetiology of enzootic ataxia in red deer.

Discussion

It is clear when reviewing the present state of knowledge on trace elements in deer, that there is a paucity of information on this subject. Also there is a danger that extrapolation of reference values and deficiency symptoms from other species may lead to errors in diagnosis and inappropriate medication.

There is a need for fundamental information on the following aspects of trace element metabolism in deer:

(1) the requirements and availability of these elements (which can be established from feeding trials);
(2) reliable values for the diagnosis of deficiencies;
(3) effective methods of supplementation.

The acheivement of these objectives is an urgent requirement of the deer farming industry and in particular of veterinarians servicing this industry.

References

Barlow, R.M. (1980). Enzootic ataxia of deer. In: The comparative pathology of zoo animals. Ed Montali,R.J. and Migaki,G., Smithsonian Institute Press: Washington, pp

65-71.
Barlow, R.M. & Butler, E.M. (1964). An ataxic condition in red deer (Cervus elaphus). J. Comp. Path. 74: 519-529.
Grace, P.(Editor) (1982). Laboratory diagnosis of trace element deficiency disease. Surveillance 9: Special Issue, M.A.F. An. Health Div: Wellington.
Hartley, W.J. (1983). Holes in nervous tissue, what are they? In: Diseases of muscle and peripheral nerve. Proc. 13th A.G.M. N.Z.Vet.Soc. for Vet. and Comp. Path. p43.
Howell, J.McC., Davison, A.N., & Oxberry, J. (1964). Biochemical and neuropathological changes in swayback. Res. Vet. Sci. 5: 376-384.
Mason, J., Williams, S., Harrington, R., & Sheahan, B. (1984). Some preliminary studies on the metabolism of 99MO-labelled compounds in deer. Irish Vet J. 38: 171-175.
McTaggert, H.S., Lowe, V.P.W., Barden, P.J. & Gellatly, J.B.M. (1981). Copper status of red deer on the island of Rhum. Vet. Rec. 109: 155-157.
Nagy, J.G., Chappell, W. & Ward, G.M. (1975). Effects of high molybdenum intake in mule deer. J. Anim. Sci. 41: 412.
Reid, T.C., McAllum, H.J.F. & Johnstone, P.D. (1980). Liver copper concentrations in red deer (Cervus elaphus) and wapiti (C.canadensis) in New Zealand. Res. Vet. Sci. 28: 261-262.
Underwood, E.J. (1971).Copper. In: Trace elements in human and animal nutrition. 3rd Edn. New York and London: Academic Press. pp83-87.
Wilson, P.R., Orr, M.B., & Key, E.L. (1979). Enzootic ataxia in red deer. N. Z. Vet J. 27: 252-254.