TRACE ELEMENTS IN DEER

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INTRODUCTION

The domestication of deer has brought with it several health issues and one of these is trace element deficiencies The NZ farmed deer industry is still quite young and as a consequence, the amount of information known about trace elements in deer is still quite limited However, what is obvious is that copper deficiency is the major problem and that the farming of deer has resulted in widespread copper depletion in deer Based on what we know from other ruminants grazing pasture in NZ, the other trace elements that have a potential of being deficient are selenium and cobalt The aim of this paper is to

- briefly review what is known about trace elements in NZ farmed deer
- outline an approach for establishing the mineral status of deer on the farm

Copper

Clinical Syndromes

Bringing deer down from the mountains, where there diet is varied and domesticating them on NZ grassland farms has been associated with a marked reduction in liver and blood copper levels[17] This has led to several clinical syndromes being recognised

Enzootic ataxia

This is a well recognised clinical syndrome associated with copper deficiency Information to date indicates

It is uncommon in deer under 9 months of age and is most common between 1-3years although quite mature deer may be affected

Wapiti are probably more susceptible than red deer Preliminary information suggests Wapiti have lower serum and liver copper levels than red deer grazing the same area [13,17]

It is characterised by demyelination of the spinal cord and brain stem Once ataxia is observed, copper supplementation does not result in clinical improvement

Both hinds and stags are affected

Clinical signs begin as a slight unsteadiness, swaying action of the hind limbs and may progress to total paralysis

Poor growth

Poor growth from copper deficiency is widely suspected by veterinarins but controlled trials to demonstrate this have produced equivocal results No significant body weight response was demonstrated on one severely copper deficient farm [20] and others have reported only short periods of increased growth which became insignificant over longer periods[10] In one extensive study, involving several groups of deer of different ages, there was a signficant growth response over six months in one group of yearling stags but there was no effect in an earlier time period and in younger and older deer[8]

The table below summarises some of the pertinent findings in these trials

AGE OF DEER	SERUM CU/ LIVER CU	PASTURE CU, MO, S AND FE.	GROWTH RESPONSE	TIME OF YEAR
3-12 months[5]	0 9 - 4 umol/l	Cu 9-10 Mo 3-6 S 0 34-0 4 Fe 130-260	+ 10 kg over 7 months	April - June Aug - Nov
4-12 months[20]	2 8 - 3 8 umol/l	Cu 15-20 Mo 0 3-0 4 S 0 38-0 39 Fe 400-800	+ 3 1 kg over 8 months	June - Nov
5 - 14 months[10]	2 3 - 15 umol/l	Not reported	Small response on 2 of 4 farms for a short period but no diifference by end of trial	June-July Aug - Sep
9 - 15 months[6]	50 - 100 umol/kg	Not reported	+ 4 kg over 6 months	Aug - Feb
4 - 12 months[11]	360 - 500 umol/kg	Cu. 9 Mo 0 20 S 0 41	Nil	

The most marked association between copper deficiency and growth was on a peat farm in the Waikato[5] Red deer stags that were supplemented with copper grew an extra 10kg from

weaning to 12 months of age. Unsupplemented deer were very hypocupraemic throughout the trial with mean levels falling from 4umol/l to <1umol/l over the trial perid There was a significant correlation between average daily liveweight gain and serum copper levels for the periods April-June and August to November Between August and November, for every one umol/l increase in serum copper there was a liveweight gain of 0 5±0.12 kg This compared with 0 36±0 16kg for the April to June period There was no growth response between June and August despite serum copper levels of unsupplemented deer averaging <3umol/l over this period.

Although trial information is still limited it appears that serum copper levels may need to be quite low before a significant response to supplementation occurs The accepted normal range for serum copper in deer is between 8-22umol/l Levels may need to be considerably below 8umol/l in many animals within a group before growth is significantly affected In cattle, a similar situation exists Animals are copper depleted if their serum copper levels are below 8umol/l but dysfunction may not occur until levels are <3umol/l [18]

It is also possible that the degree of response is influenced by the reason for the deficiency In cattle, molybdenum induced copper deficiency produces more severe animal performance effects than deficiencies induced by high iron or low copper intakes [16] Too few trials have been done to evaluate this in deer but the greatest response did occur on the farm with the high molybdenum levels

The effect of copper on growth rates may also depend on the route of administration. In a trial reported by Familton and Harrison [6] a growth response to copper supplementation occured only with copper oxide wire particles and not when an injectable product was used despite both treatments producing similar liver copper levels It is speculated that the response to COWP was due to its anthelmintic effect COWP has been the supplement used in all trials reported where there has been a positive growth response

Osteochondritis

This symptom has recently been described in detail[1,19] The pertinent facts are

- found in young deer from 1-7 months of age
- affected deer are lame and sometimes have a 'bunny-hopping' gait and/or a 'cow-hocked' stance Hocks and carpi are frequently swollen They are often also in poor body condition with dull coats
- typically there is multiple joint involvement with most severe lesions in the carpal, tarsal, stifle and hip joints
  the postural abnormalities ('bunny-hopping' and 'cow-hocked' stance) are usually from femoral epiphyseal fractures
- copper supplementation of affected calves may improve their body condition and general health but will not reverse the underlying joint condition
- Can affect more than 30% in a group
- Some affected farms also report cases of enzootic ataxia in adult deer

Osteoporosis of copper deficiency

Bone fragility as a consequence of osteoporosis is often a feature of copper deficiency in sheep and pigs and has also been reported less commonly in calves, foals and dogs

Thinning of the cortices of long bones has been reported in deer calves with osteochondrosis but no shaft fractures were reported and it could not be established whether the thinning was due to copper deficiency or secondary to reduced food intake[19] There have been occasional reports of spontaneous midshaft limb fractures in deer of low copper status[4] Such reports have generally been singletons which is different to that described in lambs where several cases generally occur Furthermore, there have been no pathological descriptions of affected bones as to their degree of osteoporosis Consequently, there is as yet insufficient information to incriminate copper deficiency as a cause of osteoporosis and spontaneous fractures

Estimating the copper status of deer

Blood and liver are the two tissue types generally used for estimating the copper status of animals Studies in deer indicate that the relationship between copper in the liver and serum is similar to that described for cattle and sheep[4,14] Liver is the storage organ for copper and when copper intake is below requirement, liver copper stores are depleted in order to maintain the serum copper in the normal range As liver stores become progressively depleted, an increasing proportion of deer will be unable to maintain serum levels within the normal range and so become hypocupraemic

Production related reference ranges for serum and liver copper are still being established However correlations between these parameters and clinical cases of enzootic ataxia suggest deer with liver copper levels below 100umol/kg are at risk of developing this disease[4,14] Data from 426 paired liver/serum samples indicate that if liver copper levels are >100umol/kg, almost all deer have serum copper levels between 8-22 umol/l[4,14] This is currently used as the reference interval for serum copper Almost all deer with serum copper levels <8umol/l have liver copper levels <100umol/kg Approximately 50% of deer with liver copper levels <100umol/kg have serum copper levels <8umol/l

As a result of the above, the following situations are relevant

- Almost all deer with adequate liver stores have serum copper levels in the reference range (8-22umol/l)
- Increasing liver levels in deer that already have adequate stores will have a negligible effect on serum levels
- Low serum levels almost invariably indicate that liver reserves are low
- Individual normal serum levels give little indication of liver stores Liver levels can range from <100 to >1000 umol/kg when serum levels are within the normal range
- Clinical and subclinical copper responsive diseases are unlikely to occur in the absence of low serum copper levels. This is because blood is the medium for distributing copper to the

metabolic sites where it is needed. For these sites to become depleted and therefore dysfunction to occur serum copper levels must be low

Analysis of 10 blood samples should be sufficient to detect a copper deficiency If 3 or more samples are <8umol/l, it is probable that approximately 70% of deer in the herd will have liver copper concentrations of less than 100umol/kg[14]

Serum copper versus ferroxidase

The relationship between measurements of elemental copper in serum and copper in the form of the enzyme ferroxidase has been established for deer The two correlate very well so that either is a reliable measure of the amount of copper in serum

Supplementation

Copper oxide wire particles, (COWP), are the most commonly used form of copper supplementation in deer This is because their duration of effect is longer than copper edta injections and any possible injection lesions are avoided The risk of toxicity is also very low with this product

Trials have been done using different dose rates with different products and in different aged deer In one study, 10g of COWP to weaner red deer stags increased liver levels from 100 to 850umol/kg at 2 months[20]. Levels had fallen substantially by four months to the extent that a few deer had liver levels below the threshold level of 100umol/kg Mean levels remained adequate, however, for 6 months In another trial where both 5 and 15 gms of COWP were used, peak levels of approximately 400 and 600 umol/kg occured at four weeks post dosing but, in both groups, levels had returned to control levels by four months[8] In the trial on peat with high pasture molybdenum levels, 10g COWP were given at the beginning of the trial in April and again in July, September and October Of the seventeen supplemented deer that were repeatedly sampled over this period, the number with serum levels <8umol/l were June 11, August 4, October 4 and November 7 Two of the deer remained hypocupraemic throughout the trial[5]

In older deer, responses appear more predictable 10 gms of COWP increased liver reserves of rising one year stags from approximately 130umol/kg to 500umol/kg and levels remained higher than unsupplemented deer for 4-6 months[8] Similar levels of increase occured in mature stags given 10 g COWP[8] The levels were maintained above unsupplemented animals for six months 20 gms increased liver copper levels to a greater extent than 10 gms but the difference was small Giving 80 grams of COWP to mature stags gave a substantial increase in liver copper levels compared with 20 grams but the rate of decay was rapid so that there was no difference in levels at four months

The increased variability of COWP in weaner deer has also been reported in young cattle and sheep It is hypothesised that the smaller rumen and reticulums lowers the retention time of particles in these compartments and hence in the abomasum This was supported in one study by

the fact that peak levels in the liver occured at 4 weeks compared with 6 weeks in more mature deer[8]

Trials have been done comparing the efficacy of copper edta injections with COWPs 50 mg of Cu-EDTA produced a similar initial increase in liver Cu to 5gms of COWP om rising one year stags Higher levels were maintained for longer with the COWPs A similar pattern was observed between 100 mg of Cu-EDTA and 10 or 15gms of COWPs

The percentage of a Cu-EDTA dose that translocates to the liver appears to be dependent on the liver copper levels In one study, where two copper injections were administered 6 weeks apart, 90% of the first dose was present in the livers one week later compared with approximately 60% of the second injection[9] The average liver copper level at the first dose was approximately 260umol/kg whereas levels were much higher when the second injection was given (ranged from 700-1277umol/kg)

Abscess formation has been a problem in the field with the use of copper injections However, the incidence of local reactions at injection sites in controlled trials has been low[9]

Selenium

Little is known about the role of selenium in deer

Reports of selenium responsive conditions in farmed deer are rare Cases of white muscle disease have been reported and it has been suggested that selenium deficiency was a predisposing factor in post capture myopathy There is some anecdotal evidence that selenium responsive illthrift in growing deer may occur but as yet this has not been confirmed with controlled trials From a practical perspective it is generally assumed that deer have similar selenium requirements to sheep and cattle and supplementation programmes have been modelled on these

White muscle disease

This is occasionally diagnosed in the field and in veterinary diagnostic laboratories A summary of findings is

- Calves generally are between 1-6 months of age
- Cases have occurred in both red and fallow breeds
- In some cases only cardiac muscle is involved, in others only skeletal and in some both cardiac and skeletal muscle is affected There is no obvious age relationship with distribution of lesions
- Liver selenium levels of affected calves have ranged from 370-590 nmol/kg
- Liver vitamin E levels of affected calves have ranged from 0 7-2 4 micro mol/kg
- Whole blood selenium levels from four dams on one farm were 84-140nmol/l

The liver selenium reference range for sheep is >450nmol/kg and for cattle is >850nmol/kg The reference range for liver vitamin E is >11 5 umol/kg From the above cases it appears that the

liver selenium reference range for cattle is more applicable to deer than the sheep reference range As in sheep and cattle, vitamin E may also play a part in the pathogenicity of white muscle disease in deer

Selenium responsive illthrift

There is just one report of a controlled trial that examined the effect of selenium on growth [12] No growth response occurred in deer grazing pasture with a selenium content of 30-57ppb The unsupplemented calves had mean blood selenium levels that varied from 730 at the start of the trial to a low of 316 in the winter Extrapolating from the results of this single trial suggests deer are unlikely to have selenium requirements markedly different from sheep and cattle and that their whole blood levels are likely to be comparable for comparable selenium intakes

Selenium responsive infertility

The effect selenium deficiency may have on reproduction in deer has not been established

There is an obvious need to establish more definitive guidelines for the selenium requirements of deer and the levels of selenium in blood and liver at which responses can be expected However, until this is done, reference ranges for cattle seem a reasonable compromise

Whole blood selenium versus glutathione peroxidase.

There is a good relationship between selenium in whole blood and glutathione peroxidase activity in the range where this has been determined between 300-1000nmol/l whole blood selenium[12] For a given whole blood selenium level, deer have a lower GPX activity than sheep but similar to cattle The interelationships between the different forms of selenium in blood (serum selenium, whole blood selenium, whole blood gpx) and liver selenium under steady intake has not been established; needless to say how these interelationships change when supplements are given is also unknown

Supplementation

50 mg of selenium as barium selenate injected subcutaeously increases whole blood and GPX activities for at least 12 months in growing calves[12] GPX activities increased from 5-7 kU/l to 20-25kU/l Monthly oral doses of sodium selenate at 1mg/10kg also significantly increased whole blood Gpx activities above levels in unsupplemented calves[12] This limited data suggests dose rates and routes of administration as used in sheep and cattle are probably safe and efficaceous in deer

Cobalt

Little is known of the cobalt requirements of deer With deer farms being geographically widespread throughout New Zealand, deer are grazing soil types that are known to vary from

adequate to severely deficient for other ruminants The results of two controlled supplementation trials in weaner and yearling deer grazing soil types considered deficient for sheep have been reported[3] In one trial, the pasture cobalt content averaged 0 04mg/kg which is about 50% of the level required for growing lambs and about the minimum required for growing cattle Serum vitamin B12 levels of unsupplemented animals during the trial ranged from 75-83pmol/l 2mg of hydoxycobalamin at approximately monthly intervals significantly increased serum B12 levels over control animals throughout the trial but there was no difference in weight gain The same trial protocol was repeated on a farm where pasture cobalt levels varied from 0 12-0 055mg/kg Again serum vitamin B12 levels increased significantly following the injection of 2mg of hydroxycobalamin but again there was no effect on weight gain It was concluded from these trials that growing red deer hinds have a lower requirement for cobalt than lambs and therefore serum vitamin B12 reference ranges applicable for sheep are not applicable for deer

Where possible, weight gain response trials should be encouraged, especially when serum vitamin B12 levels are less than 120pmol/l

RECOMMENDATIONS FOR INVESTIGATING THE MINERAL STATUS OF A DEER FARM

* Information used to develop these recommendations is derived from three sources
* Published literature on minerals in deer
* Published literature on minerals in other species
* Personal experiences in a diagnostic laboratory over the last 15 years

There are obviously some large gaps in our knowledge, especially as relates to cobalt and selenium requirements of deer How, therefore, should a veterinarian approach defining the trace element status of deer on a farm and what recommendations should be given regarding supplementation The following are guidelines based on information known to date As we learn more, these guidelines will need to be revised and some may in fact be wrong, but they are a start

Copper

As this is likely to be the most significant mineral deficiency, most effort should be focussed on this The time of year when copper availability is lowest is generally during the winter and early spring when molybdenum and iron intakes are the highest The age group of deer with probably the highest requirements at this time are the rising one year growing deer and the pregnant hinds Therefore, sampling these two age groups at this time should determine whether copper deficiency is likely to be a problem on the farm Liver is the preferred sample as it measures copper stores However, most veterinarians do not feel competent to biopsy deer and therefore blood is usually taken Probably 10 should be collected

At the same time, at least one pasture sample representative of each soil type on the farm should be collected and the minimum number of analyses should include copper, molybdenum, sulphur, iron, selenium and cobalt

If all serum values are within the reference range, and pasture copper, molybdenum, sulphur and iron levels are within the optimal range then no immediate action is necessary The farmer should be encouraged, however to collect livers from any deer that die suddenly of traumatic causes and from at least one line of deer going for slaughter This will provide information on how adequate the copper status of the deer on this farm is Annual July/August monitoring of serum copper levels of growing deer should continue for 2-3 years just to make sure there are no significant between season fluctuations

If some serum levels are low, then a more detailed investigation of all age groups is necessary Bood samples should be collected from all groups of deer on the farm to deterimine how widespread the deficiency is On many deer farms, this will mean blood serum copper levels will be established for

- Pregnant hinds
- Rising one year stags/hinds
- Rising two year stags/hinds
- Mature stags

Based on these results, a supplementation programme should be developed for the groups that are of low copper status Probably most critical in this regard are the rising one year stags and hinds and the pregnant hinds The important thing, once you have recommended a supplementation programme is to set up a monitoring programme This is not just for academic reasons The efficacy of supplements is known to be quite variable and some of the variables appear to be molybdenum intakes, age of deer, dose given etc At least in the initial stages, it is insufficient to rely on the manufacturers recommendations These are very much average expectations and in many circumstances the supplements do not behave to the average For example in one trial, 10 g of COWP had to be given every 2 months to weaner deer before normocupraemia was established[5]

As a recommendation, the supplemented animals should be sampled approximately at the half way stage of expected efficacy For example, for COWP if the expected efficacy is 4 months, then samples should be collected at 2 months Livers collected from animals sent for slaughter, may be a valuable way of determining efficacy of supplements if they are being slaughtered at an appropriate time For example, if supplements are given in September and some of the group are being slaughtered over the next 6 months, then some livers should be collected and analysed from several of the groups (e g those slaughtered in November, January, March)

It is also recommended to collect some more pasture samples, representative of soil types, on at least two occasions (December, April) This will provide information on the effect of season on pasture levels which is valuable for determining how seasonal the copper deficiency is likely to

be It may be that supplements are only needed for a 3-4 month period in only one group of deer over the winter, spring period

Selenium

As the only selenium responsive condition so far described in deer is white muscle disease the initial test should probably be done on late pregnant hinds An alternative, depending on the time of year, would be to bleed a proportion of the weaners in the autumn. In cattle, this is the time of year when selenium responsive illthrift is most likely As with copper, obtaining pasture samples from representative soil types is also highly recommended at this time If pasture selenium levels are >0 03ppm and blood selenium levels considerably higher than 250nmol/l, then selenium deficiency is unlikely on this farm If levels are lower than these, then it would seem prudent to advise some form of supplementation at dose rates and intervals similar to those recommended for sheep and cattle Although the effect of selenium supplements on blood selenium levels is probably reasonably predictable it is still advisable to check by bleeding some deer at periodic intervals (whilst on the farm doing something else)

Cobalt

The best way to determine if cobalt deficiency is likely to be a factor in deer is to bleed, or take liver samples from lambs grazing the same soil type This should be done between December and February and may be from livers of fat lambs sent to the works during this period Adequate levels in lambs at this time would make cobalt deficiency in deer most unlikely Deer, almost certainly have lower cobalt requirements than lambs and the summer is the time when pasture cobalt levels are at their lowest If levels are marginal to low in the lambs or there are no sheep on the farm, then serum samples should be collected from a group of young growing deer (one year olds at this time of year) Pasture should also be collected for cobalt measurements as for selenium and copper If serum B12 levels are >120pmol/l and pasture cobalt levels are >0 08ppm, then cobalt deficiency in the deer is most unlikely If serum B12 levels are <120pmol/l or pasture cobalt levels are <0 08ppm then the farmer should be encouraged to perform a vitamin B12 response trial This is probably best started in the spring with some rising one year hinds or stags and continued over the summer There should be at least 20 deer in the supplemmented group and 20 in the unsupplemented group and all other growth limiting factors eliminated (worms, other mineral deficiencies, level of nutrition etc)

On farms where lambs have been diagnosed as deficient there is obviously a need to correct this deficiency If the area being grazed by the deer is relatively small and/or some of the deer fenced land is periodically grazed by lambs, then it seems reasonable to apply cobalt fertiliser to the deer area Applications of cobalt fertiliser, sufficent to correct a deficiency in lambs almost certainly will eliminate any potential deficiency in the deer and therefore there will be little need to test the animals

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