A review of the trace element nutrition of deer

A Confidential Report prepared for:

DEEResearch

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Summary

- Cobalt (Co), Selenium (Se) and Iodine (I) deficiencies are serious problems requiring supplementation on only a small number of deer farms.
- Copper (Cu) deficiency is widespread and supplementation is warranted in many herds. It can be diagnosed from clinical signs such as enzootic ataxia and osteochondrosis, confirmed by pathological investigation, as well as low serum and liver Cu concentrations.
- While some knowledge and technology exists, there is widespread farmer uncertainty about the diagnosis and prevention of Cu deficiency in deer.
- The metabolism of Cu is complex. There is usually a seasonal decline in deer Cu status during winter and early spring. Age also influences Cu status. Dietary factors such as Mo, in the presence of S, may reduce Cu absorption, utilisation and storage, but little is known about these interactions in deer.
- From limited trial and clinical data the following reference ranges for serum and liver Cu concentrations are proposed to categorise the Cu status of deer: Serum Cu (µmol/L) <5 deficient, 5-8 marginal and >8 adequate; Liver Cu (µmol/kg fresh tissue) <60 deficient, 6-100 marginal and >100 adequate.
- There are inadequate data to establish reference ranges or biochemical criteria to assess the Co (vitamin B_{12}), Se or I status of deer.
- The dearth of data on trace element nutrition in deer means that further studies, including trace element supplement/deer response trials and intensive metabolic studies are needed to establish with a greater confidence the dietary requirements

and protocols to diagnose deficiency and its management in order to ensure trace element deficiencies do not limit deer production.

- There is currently insufficient research data to support the most effective veterinary and farmer decision-making about trace element nutrition management, resulting in continuation of risk to animals, and financial wastage in many circumstances.

Abbreviations used in this review:

Co Cobalt (Note: vitamin B_{12} is the biologically active form of Co)

Cu Copper

I Iodine

Se Selenium

Introduction

The first deer farms were established in New Zealand about 30 years ago and much of the early deer research was focused on immediate health problems such as tuberculosis and yersiniosis, general nutrition, management and husbandry practices. Extensive trace element studies with sheep and cattle have resulted in clarification of their trace element requirements and protocols to diagnose and prevent deficiencies. In contrast there have been relatively few studies carried out with deer, and this situation with trace elements has recently been described as an enigma in deer (Wilson, 1999). However, recent studies on Se and Cu are beginning to provide some of the much needed information to manage trace element deficiencies in deer (Grace et al 2000; Grace et al 2001), but currently little remains known about deer trace element requirements and metabolism which is pivotal to the management of deficiencies in farmed deer herds.

This review summarises information available published in the technical and scientific literature on trace element nutrition of deer with particular reference to the role of Cu, Se, Co (vitamin B_{12}), and I deficiencies on the health and performance of farmed deer in New Zealand.

The diagnosis of trace element deficiencies and the determination of dietary trace element requirements

Methods for the diagnosis of Cu, Se, Co and I deficiencies in deer vary. If the deer are deficient then the treated animals will grow significantly faster than the untreated controls managed under the same conditions while clinical signs of any deficiency will also respond to or be prevented by trace element supplementation. Scientifically valid trace element supplementation animal response trials are demanding to carry out, but are essential to provide basic information to establish tissue trace element reference ranges to diagnose trace element status of a herd or individuals (Towers et al 1984). They are also essential to determine dietary trace element requirements for deer.

Relating changes in blood and liver trace element concentrations to animal responses, or the absence or presence of clinical disease, when trace element deficient deer are supplemented in dose response trials, enables tissue reference ranges to be established so that the trace element status of deer can be readily assessed from measurement of tissue trace element concentrations. However, this approach may not provide a full understanding of the interactions between dietary components, particularly for Cu metabolism. Intensive metabolism studies are required to provide data to enable this to be done.

The minimal dietary concentration at which no response to a trace element supplement occurs is taken to be the dietary trace element requirement, that is, the daily trace element intake that is adequate to meet the deer's needs for maintenance, growth and reproduction. If the magnitude of dietary trace element interactions are

known, for example where these interfere with absorption, then these must also be taken into consideration when determining dietary trace element requirements.

To date, very few trace element supplementation deer performance response trials have been carried out and therefore there is a dearth of data on which to establish tissue trace element reference ranges and dietary trace requirements although there is more information on Cu than the other trace elements.

Copper

Introduction

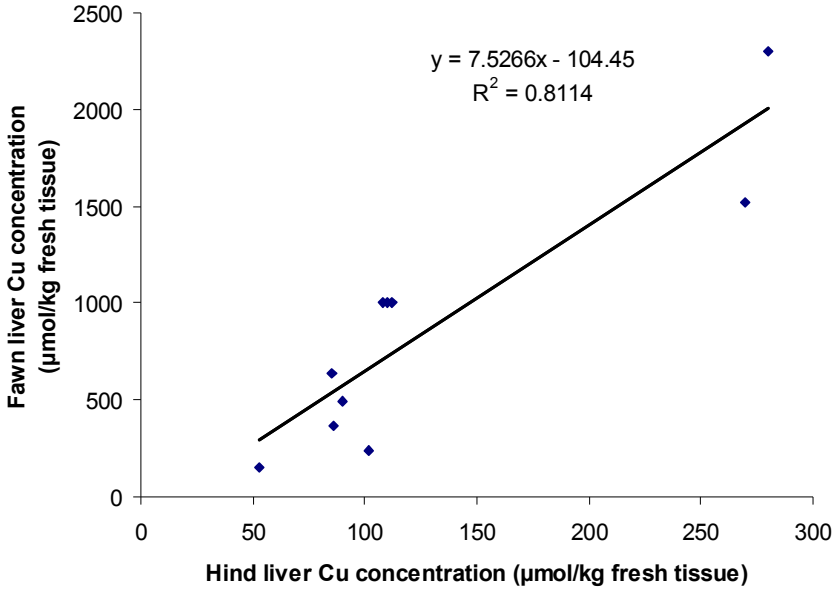
The metabolism of Cu is complex. To date it has been very difficult to obtain sufficient data on deer performance and tissue Cu responses to Cu supplementation, in relation to dietary intake, to provide the confidence to predict responses to Cu supplementation. Furthermore, clinical and research evidence suggests that dietary factors appear to influence the absorption and utilisation of Cu, but these are not yet understood.

Distribution

Cu is distributed among body tissues with the liver being an important storage organ (Booth et al 1989). Serum and liver Cu concentrations vary according to intake, age, season, origin and species (Mackintosh et al 1986; Mackintosh 1992; Wilson and Audige 1998; Grace 1999; Grace et al 2001; Tremain-Boon et al 2001, 2002). Increasing Cu intakes or Cu supplementation will increase serum Cu if concentrations are low to begin with, but not if they are high before supplementation. However, liver Cu concentrations will invariably increase after an increase in Cu dietary intake or supplementation, demonstrating its role as the primary storage organ for Cu.

Liver Cu concentrations of the foetus/neonate are much greater than those of the hind (5650 v166 µmol/kg fresh tissue) (Reid et al 1980; Grace et al 2002). Hinds with a high Cu status, that is, with high liver Cu concentrations, appear to have fawns with a high liver Cu concentrations or Cu stores as shown by limited recent data in Figure 1.

Figure 1. Effect of the liver Cu oncentrations of hinds on the liver Cu concentrations of their fawns at 3 weeks of age



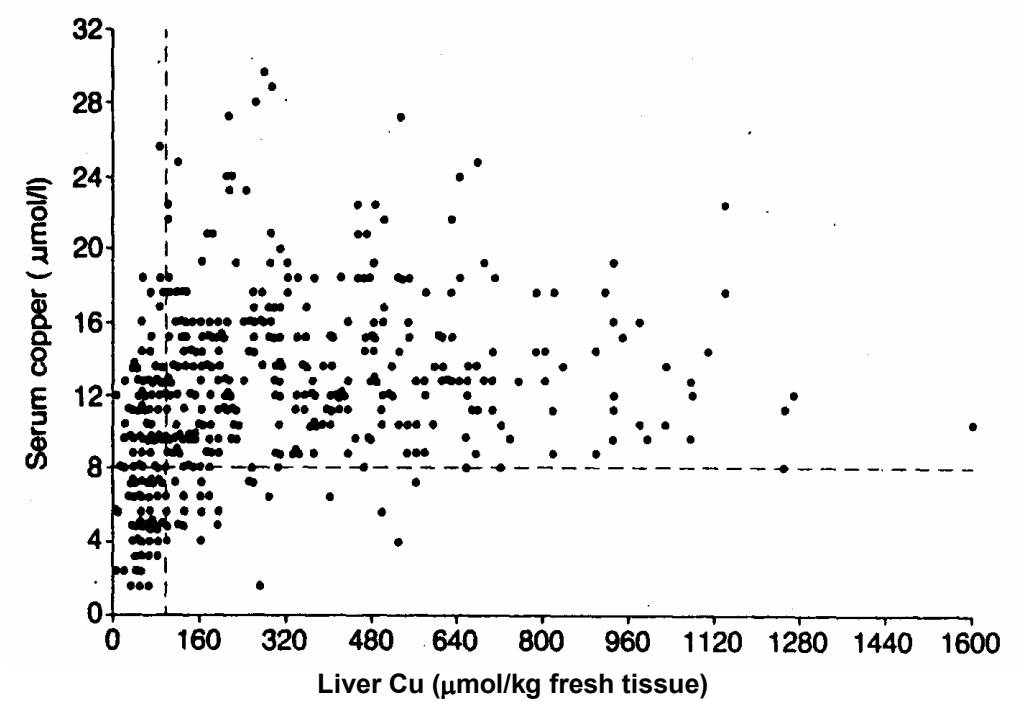
Season has a marked effect on the status with the lowest serum and liver Cu occurring in late winter/early spring (Wilson and Audigé 1998; Grace et al 2002). The factors influencing this decline in Cu status are not well understood, but may be associated with grazing management or animal metabolism (Wilson and Grace 2002).

A comparison of farmed and feral deer showed that liver Cu concentrations from samples collected in November of the feral animals were at least 3 times higher (237 v. 813 µmol/kg fresh tissue) (Tremaine-Boon et al 2001, 2002). A similar pattern was described by Reid et al (1980). This difference most likely reflects the ability of feral deer to graze, browse and/or select forages, which are higher in Cu and/or have lower concentrations of dietary factors, which can reduce Cu absorption, than the diets offered to farmed deer. Preliminary data showed that grazing chicory (mean Cu concentration 10.7 mg/kg DM) in autumn increased mean liver copper concentration to about 333 µmol/kg compared with about 160 µmol/kg in ryegrass/white clover

(mean Cu concentration 7.6 mg/kg DM) fed deer (Barry et al 2001). Further research has shown a five-fold increase in copper in late November in deer grazing chicory in autumn and spring (Wilson et al 2002, unpublished data).

There are no comparative research studies of deer species. However, clinical observations of lower liver Cu concentrations and a higher incidence of enzootic ataxia in Wapiti and Wapiti x red hybrid deer than in red deer that were grazed and managed on the same pastures suggest species differences in susceptibility to Cu deficiency (Mackintosh et al 1986; Mackintosh et al 1992). There is a relationship between liver and serum Cu concentrations, which is illustrated in Figure 2 using paired liver and serum Cu measurements from 426 deer. When liver Cu concentrations are <100 µmol/kg fresh tissue about 50% of the deer will have serum Cu concentration of <8 µmol/L. The liver acts as a store and when Cu intakes are inadequate blood Cu are maintained at the expense of the liver Cu stores. As these become more and more depleted an increasing number of deer have low Cu concentrations ranging from 2.8 to 5.0 µmol/L (Mackintosh et al 1986).

Figure 2. The relationship between serum Cu and liver Cu concentrations in deer (From Mackintosh et al 1986)



Copper is transported across the placenta and increases foetal liver Cu stores. The Cu concentrations of foetus/neonates are much greater than those of their dams. Milk Cu concentrations range from 2.8 to 3.2 µmol/L and are not influenced by Cu supplementation or intake (Grace et al 2002, unpublished data).

Functions

Copper is a vital constituent of many enzyme systems in a variety of tissues throughout the body and has an important role in many biochemical and physiological functions. These are reflected in various clinical signs and sub clinical effects of Cu deficiency.

The central nervous system disorder enzootic ataxia occurs in deer, with the youngest reported case being 5-months of age (Barlow et al 1964; Wilson et al 1979; Audige et al 1995). This contrasts with sheep, where clinical cases occur at birth, or up to 3-months of age. The syndrome is complex but it is associated with lesions in the brain and spinal cord with various nerve fibres losing their insulating layers (becoming demyelinated), thus impairing the function of the nervous system (Barlow et al 1964; Fell et al 1965).

Bone growth is a complex process during which cartilage, laid down by the chondrocytes, is progressively invaded by cells that dissolve the cartilage and develop a collagen substratum, which serves as a template for the deposition of the Ca and P. In sheep and cattle Cu-deficiency has been related to a reduction in the activity of the enzyme lysyl oxidase, responsible for the formation of cross-linkages in the collagen, the organic matrix that supports Ca and P deposition in bone, giving it strength (Rucker et al 1969). Osteochondrosis of young deer has been observed commonly in Cu deficient animals (Thompson et al 1994; Audige et al 1995). This syndrome causes arthritis, since the bone tissue under joint cartilage collapses as a result of insufficient bone organic matrix formation for calcification. Lesions can be particularly severe in the hip and hind leg joints.

Copper is important in maintaining the integrity of the immune system as a deficiency affects white blood cell (phagocyte) function and reduces antibody production (Suttle and Jones 1986). It is therefore likely that Cu deficient deer will be more susceptible to infectious diseases such as yersiniosis and tuberculosis although there has been no research into this sub clinical effect.

Ferroxidase (ceruluplasmin), a blood enzyme, oxides the ferrous iron (Fe^{2+}) to ferric iron (Fe^{3+}) allowing the mobilisation of Fe between the plasma Fe (transferrin) and the Fe stores of the tissues (ferritin). Thus, as seen in sheep and cattle, severely Cu deficient deer may show anaemia, since there could be inadequate Fe, in an available form, to synthesize haemoglobin, the oxygen carrying pigment of red blood cells. This has not been reported in deer, possibly because it has not been investigated.

Animal performance

Only 2 of 11 reported trials of Cu supplementation growth studies have resulted in a small but significant liveweight response (6-10 kg) in young deer (Ellison 1995; Wilson 1989; Killorn and Wilson 1991; Harrison et al 1992; Wilson and Grace 2001). The mean serum and liver Cu concentrations in untreated animals prior to supplementation which responded to Cu were <5 µmol/L and 40 µmol/kg fresh tissue, respectively. The greatest response of approximately 10 kg was shown by Ellison (1995), who described a mean serum Cu concentration as low as 0.9 µmol/L during the study. In most of the other 9 trials where no growth response was observed to Cu supplementation the pre-trial mean and ranges of serum and liver Cu concentrations were similar to those herds where response occurred, although concentrations were in the "marginal" range in most deer.

However, in one trial, Harrison et al (1992) demonstrated a growth response in 18-month-old stags in which tissue copper concentrations were in the "adequate" range described by Wilson and Grace (2001). This may have been a spurious result, as happens from time to time in science. These observations demonstrate the difficulty in establishing reference ranges, and in predicting responses to Cu supplementation. In

sheep and cattle trials, liveweight response to Cu supplementation have only been observed in the presence of high Mo pastures (>2.5 mg Mo/kg DM) (Phillippo 1983). Thus, priority should be given to investigation of the Cu x Mo interaction in deer to determine the role of other dietary elements in effecting growth responses.

Copper supplementation had no effect on velvet antler growth when the mean liver Cu concentration was 98 µmol/kg fresh tissue and serum Cu concentrations ranged from 6-13 µmol/L (Walker et al 1997, 2002). There are no published data on the impact of Cu supplementation on the reproductive performance of deer.

From the current information it is possible to determine from liver and serum Cu concentrations whether an animal or herd is at risk of Cu deficiency syndromes, although it is not possible to predict whether or not they will actually occur. It is currently not possible to establish tissue Cu reference ranges to accurately predict growth responses in deer (Wilson and Grace 2001), or whether a production response will occur after Cu supplementation. Further, it is currently not possible to predict the efficacy or duration of effect of Cu supplementation.

Signs of deficiency related to tissue Cu concentration

Clinical copper deficiency in deer is therefore most likely to be diagnosed from clinical disorders such as enzootic ataxia in older deer and osteochondrosis in young deer. Enzootic ataxia (Plates 1 & 2), confirmed from laboratory reports describing histopathology in 16 animals, was associated with liver Cu concentrations of <60 µmol/kg fresh tissue, except in one case where liver Cu was 86 µmol/kg fresh tissue

Plate 1. Enzootic Ataxia.
Adult hind showing incoordination of hind legs.

Plate 2. Enzootic Ataxia.
Yearling hind showing inability to stand upright.

Plate 3. Osteochrondrosis.
Weaner calf showing poor growth due to joint pain of arthritis.

Plate 4. Osteochondrosis.
Arthritic lesion on joint cartilage due to the collapse of bone underneath.

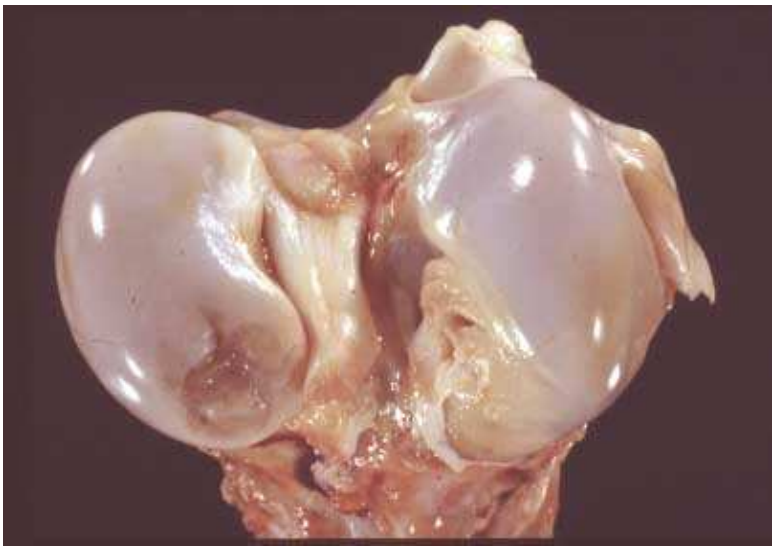


Plate 5. Osteochondrosis.
Cross-section of hip joint showing the total collapse of bone and cartilage.
Cu deficient deer on the right.

(Wilson and Grace 2001). The limited amount of serum Cu data showed concentrations of <2.7 µmol/L in affected deer and <5 µmol/L in clinically unaffected in-contact animals. Osteochondrosis (Plates 3-5), diagnosed by gross pathology, was associated with liver Cu concentrations <53 µmol/kg fresh tissue and serum Cu concentrations of <3.1 µmol/L (Thompson et al 1994; Audige et al 1995). The mean serum Cu concentrations of unaffected in-contact deer were <5.1 µmol/L. It must be cautioned that these associations are from only small numbers of animals, and supplementation history prior to investigations was largely unknown. Further studies, preferably involving field investigations where clinical signs are observed would provide data for more robust reference values.

Proposed tissue Cu reference range in deer

From the limited data available the following criteria have been proposed (Wilson and Grace 2001) to assess the Cu status of deer. For liver, Cu concentrations of ≤ 60 µmol/kg fresh tissue represent the "deficient" range wherein deer may be at risk of

clinical disease or impaired growth rate. Animals in the 60-100 µmol/kg fresh tissue range are considered "marginal" while those >100 µmol/kg fresh tissue is considered "adequate", that is, they are most unlikely to respond to Cu supplementation.

For serum Cu concentrations the reference ranges are ≤ 5 µmol/L, 5-8 µmol/L and >8 µmol/L for a "deficient", "marginal" and an "adequate" animal Cu status respectively.

Sampling protocols to assess the Cu status of deer

In contrast to Co (vitamin B_{12}) and Se where there are no marked seasonal effects on their status, and changes in serum vitamin B_{12} and blood Se largely reflect dietary intakes, the metabolism of Cu in deer is more complex. Practical considerations for diagnosis and sampling have been reviewed recently (Wilson and Grace 2001, 2002).

As there is a marked effect of season on liver and serum Cu concentrations on many deer farms (Wilson and Audige 1998; Grace et al, unpublished data) and because liver Cu stores maintain serum Cu concentrations until the former are depleted, the timing of tissue sample collection for Cu determinations is very important in the interpretation of tissue Cu concentrations and therefore the Cu status of deer herds.

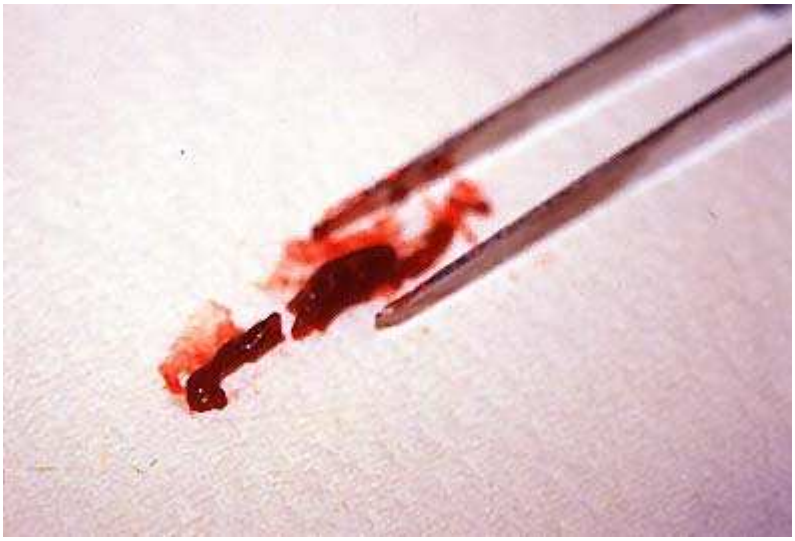
To achieve confidence that 95% or more of the Cu concentrations in the herd fall within the range measured, 16 samples are required. However, for diagnostic purposes, fewer samples are adequate (Wilson and Grace (2001) for discussion). To establish whether or not there are animals at risk of Cu deficiency in a herd attention must be given to concentrations in individual animals and not only to the herd mean. It has been noted that when the herd mean liver Cu concentration is high, the range

between animals can be very large, but when the mean is low, the range is usually small (Wilson, clinical observation).

Five to 8 liver samples / group, collected by biopsy in the live animal (Familton et al 1986; Wilson 2000) or at slaughter (Plates 6 & 7) during March/April from hinds and weaners will give a reasonable indication of Cu status, when compared to liver Cu reference ranges, in hinds before or during mating and in young deer at weaning (Grace et al 2001; Grace, unpublished data). Low serum Cu concentrations of some deer, at least 8 samples/group, at this time would likely reflect a seriously low Cu status on the farm, given that lowest Cu concentrations usually occur late winter-early spring rather than in the autumn. Immediate supplementation would be advised. However, values within the "adequate" reference range at this time provide no assurance that Cu concentrations will be sustained throughout the forthcoming winter and spring. A further sampling of liver and blood in July-September would be required to measure the Cu status of deer at a time when it is likely to be at its lowest. Hinds with a low Cu status give birth to fawns with low liver Cu stores, thereby increasing their risk of clinical Cu deficiency disease (Grace et al 2002, unpublished data).

Plate 6. A trocar is guided through the second to last intercostal space between the ribs and into the liver to remove a core of liver tissue.

Plate 7. Liver biopsy sample.



Thus, determination of the Cu status of a property in terms of deer health, and therefore the validation of the need for a Cu supplementation protocol, depends on sequential tissue sampling of deer of different ages, at different seasons. Since no two

properties are the same, this process is necessary on each individual farm rather than on a "district" basis. Further, because Cu intake is subject to so many changes in management practices such as grazing management, fertiliser application, pasture species and supplementary feeding year by year, monitoring should be repeated periodically, even on farms believed to have an adequate Cu status.

Recent data relating deer Cu status, as measured by liver Cu concentrations, to forage Cu concentrations show that diets with Cu concentrations of 10-17 mg Cu/kg DM were able maintain an adequate Cu status in deer (Wilson et al 2002). However as most pastures contain 5-8 mg Cu/kg DM the Cu status of pasture-grazed animals is frequently likely to be low or deficient, particularly in late winter/early spring.

Cu supplementation strategies

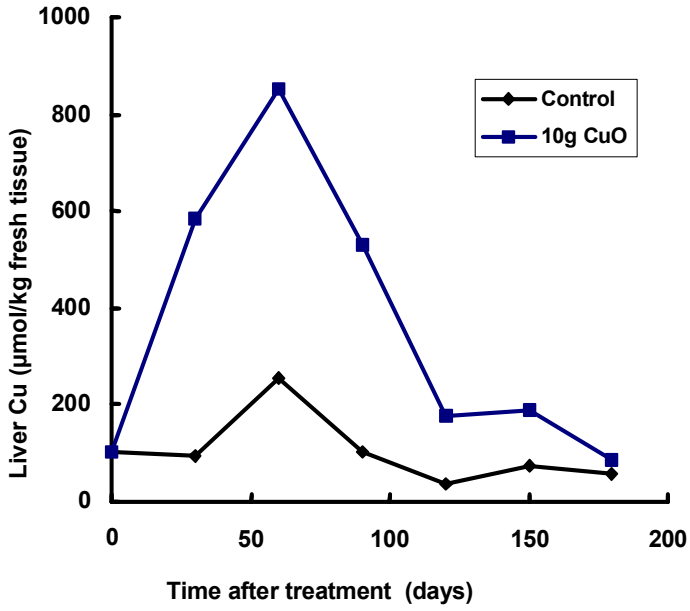
Copper supplementation options have recently been reviewed (Wilson and Grace 2002).

Copper oxide needles

The most commonly used supplement is copper oxide (CuO) wire needles (Booth et al 1989; Harrison and Familton 1992; Wilson and Audige 1998; Beatson et al 2000; Grace unpublished data 2002). There have been a few efficacy studies of this product. (Booth et al 1989, Harrison et al 1992). Data is summarised in Table 1. Recent data (Grace et al 2002, unpublished data) is presented for weaner stags (Figure 3) and pregnant hinds (Figures 4 and 5). The weaner stags were given 10 g CuO needles in March and changes in liver Cu concentrations of untreated and treated animals

monitored monthly over 6 months (Figure 3). The CuO increased and maintained adequate liver Cu concentrations for about 5 months in this class of animal.

Figure 3. Liver Cu concentrations of weaner stags either given 10g CuO needles in March or untreated



Pregnant hinds were mated in March-May and treated with 10g CuO needles in late July. They calved in November-December and their fawns were weaned in mid March. Cu status of both hinds and their fawns were monitored using serum and liver Cu concentrations. The CuO needles, given during the second trimester, increased the Cu status (i.e. liver and serum Cu concentrations) of the hinds for at least 60 days. Importantly this resulted in the substantial improvement in Cu status and liver Cu stores of their fawns from birth to weaning (Figures 4 and 5). (It should be noted that osteochondrosis of young deer has been observed previously on this property). Thus maintaining serum Cu, despite a fall in liver Cu, eliminated the risk of deficiency in the fawn, whereas offspring of hinds with low serum Cu were at risk. As the Cu

treatment had no effect on the milk Cu concentrations supplementing hinds during lactation will have no effect on the Cu status of their fawns.

Figure 4. Liver Cu concentrations of yearling hinds and their fawns either given 10g CuO needles in July, or untreated (Note: Serum Cu data in figure 5)

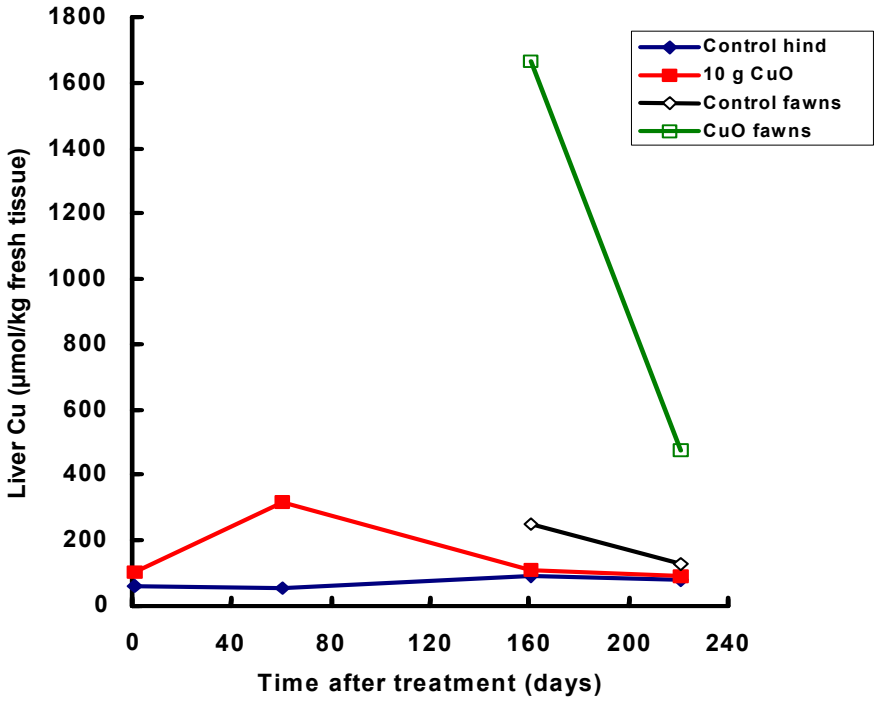
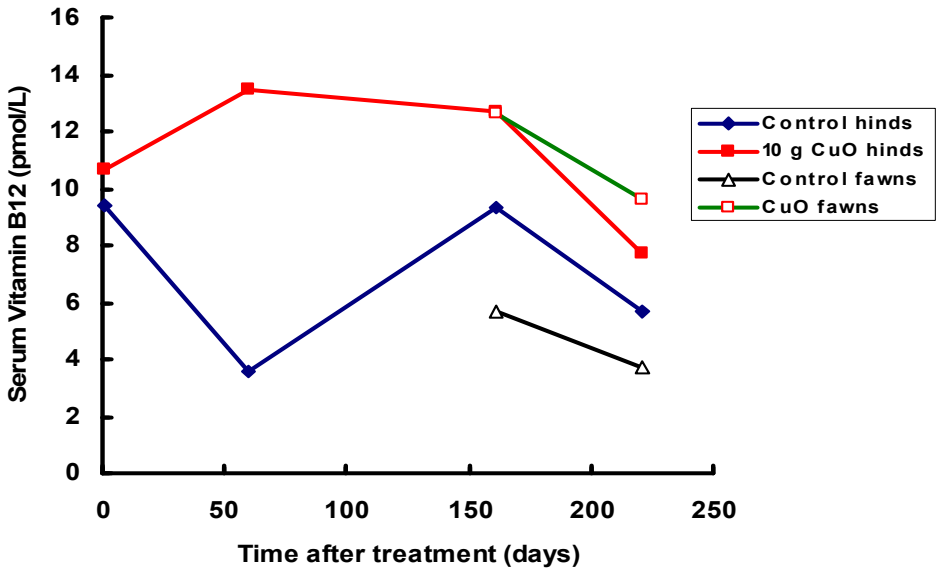


Figure 5. Serum concentrations of yearling hinds and their fawns either given 10 g CuO needles in July, or untreated (Note: Liver Cu data in figure 4)



Copper injection

An injectable Copper-Ca EDTA can be used in deer and was evaluated by Harrison et al (1989) (Table 1). The safe dose rate for the injectable Cu is up to 2 mg Cu/kg liveweight. Injected Cu is readily translocated to the liver, as the efficiency of Cu uptake is 90%. When 150 mg Cu was given to 150 kg stags the liver Cu concentrations were increased to 800 µmol/kg fresh tissue (Table 1) while efficacy of the product was estimated to be 8 to 12 weeks.

Table 1. Effect of dose rate on the efficacy of CuO needles given orally and Cu-EDTA injections expressed as increases in mean liver Cu concentration (µmol/kg fresh tissue) above untreated controls (From Grace and Wilson 2002)

Study	Treatment[⊥]	Age	Month of Treatment	No. of deer	Months after treatment						
					0.5	*1*	*2*	*3*	*4*	*5*	*6*
Booth et al 1989	10 g CuO	4 m	Mar	11		491	594	427	136	114	25
Harrison et al 1992*	5 g CuO	4 m	Mar	8			300	140		20	
	15 g CuO	4 m	Mar	8			600	280		80	
	10 g CuO	9 m	Sept	9		380		120			50
	20 g CuO	9 m	Sept	9		580		220			150
	10 g CuO	14 m	Feb	8		350	300		230		
	10 g CuO	≥ 3 yr	Feb	9		410	490	240			
	20 g CuO	≥ 3 yr	Feb	9		510	510	380			
	50 g CuO	≥ 3 yr	Feb	9		700	800	380			
	10 g CuO	≥ 3 yr	Sept	9		570		180			80
	20 g CuO	≥ 3 yr	Sept	9		700		400			180
	50 g CuO	≥ 3 yr	Sept	9		1300		400			180
Harrison et al 1989b	Cu-EDTA mean 0.28 mg/kg	3-5	NS	6	172	207**					
	Cu-EDTA mean 0.58 mg/kg	3-5	NS	6	452	316**					
	Cu-EDTA mean 1.23 mg/kg	3-5	NS	6	881	640**					

* Data extrapolated from graph, therefore approximation only. ** Actual time 5 weeks. Data converted from mg/kg DMB using DM estimate at 25%

Copper topdressing

Copper topdressing at the rate of 12 kg copper sulphate ($CuSO_4.5H_20$)/ha [3 kg Cu/ha] applied in March increased pasture Cu concentrations to 45-60 mg Cu/kg DM. When grazed, this pasture was effective in maintaining an adequate Cu status, in terms of serum and liver Cu concentrations, of weaners for at least 10 months (Grace et al 2001). It provided elevated serum and liver Cu concentrations in hinds through gestation and lactation as well as their fawns from birth to weaning (Figure 6 and 7).

Figure 6. Serum Cu concentrations of yearling hinds and their fawns after Cu topdressing (3 kg/ha) in March

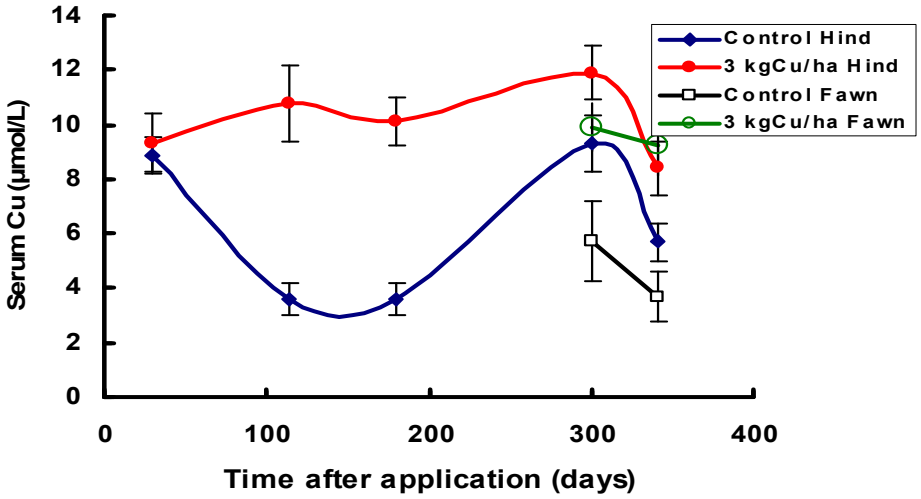
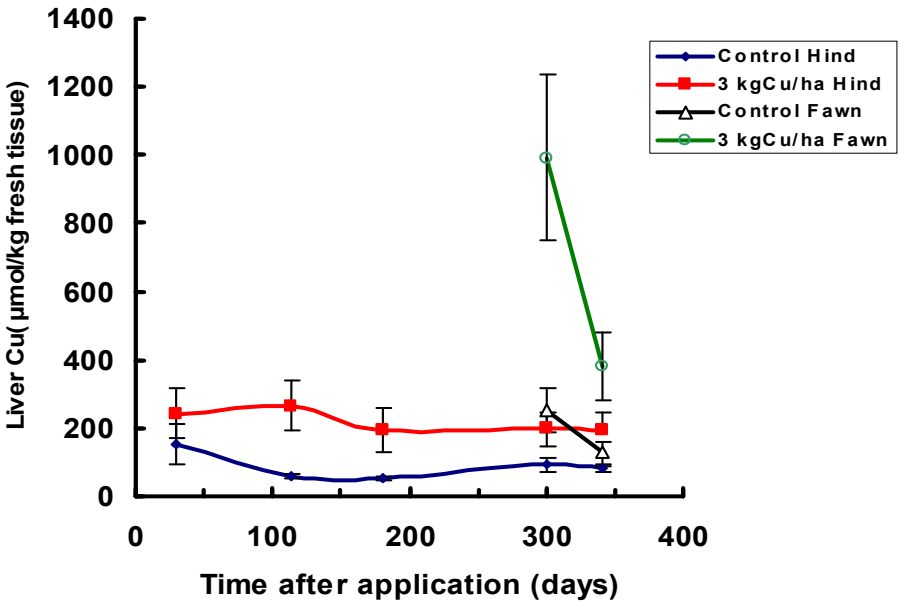


Figure 7. Liver Cu concentrations of yearling hinds and their fawns after Cu topdressing (3 kg Cu/ha) in March



The autumn application of 12 kg copper sulphate/ha [3.0 kg Cu/ha] was a very cost effective, easy approach to increase and maintain an adequate Cu status of yearling hinds during gestation and lactation and the Cu status of their fawns from birth to weaning. For this approach to be effective the Cu uptake by pasture must result in pasture concentrations being greater than 45 mg Cu/kg DM for 35 to 50 days when the deer are grazing Cu-treated pasture. This it is important to graze pastures 3-4 weeks after Cu application. The 3-4 week delay in grazing after the application allows the Cu to be washed into the soil by the rain and then to be taken up by the pasture regrowth. An application rate of 6 kg copper sulphate/ha [1.5 kg Cu/ha], the present fertiliser industry standard rate, was not effective in increasing and maintaining the Cu status of deer as pasture Cu concentrations reached only 20-25 mg Cu/kgDM. The uptake of Cu by the pasture is dependent on factors such as soil type, botanical composition, application rate and season. It is very important to monitor the changes in pasture Cu concentrations at 4-6 weekly intervals for 3-4 months to see if this

approach is suitable for a particular deer farm. Thus monitoring pasture may provide a means for predicting response in terms of deer tissue Cu concentrations. It should be noted that this research was performed on only one property, and needs to be repeated elsewhere before industry-wide recommendations can be made.

The efficacy of $CuSO_4.5H_2O$ in drinking water, added to feedstuffs, or in salt licks has not been evaluated in deer.

Thus the tissue Cu responses to Cu supplementation vary enormously, depending on a wide range of factors discussed above. Therefore, the responses to any Cu supplementation programme should be monitored by liver biopsy to ensure that the desired elevation has been achieved. In many situations, this has been shown not to be the case (Wilson and Audige 1998; Beatson et al 2000).

Copper usage by farmers

Two surveys of Cu usage by deer farmers have been reported (Wilson and Audige 1998; Beatson et al 2000) (Table 2). Several forms of Cu supplementation were used, either singly or in combination.

Table 2. Survey of farmers using Cu supplementation methods reported in South Canterbury (Beatson et al 2000) and in the lower North Island (Wilson and Audige, 1998)

Supplementation	Location	
	Canterbury	lower North Island
CuO needles alone	3	3
CuO needles & drench	1	4
CuO needles & fertilizer	1	
CuO needles & fertilizer & drench	1	
Fertilizer alone	0	1
Drench & fertilizer	0	0
Injection alone	0	0
Injection & CuO needles & feed supplement	0	0
Feed supplement alone	0	0
No Cu supplement	4 (64%)	7 (47%)

Analysis of the Cu usage patterns show little consistency within farms between years and age groups. When animal tissue Cu concentrations were observed concurrently with Cu-supplementation practice, farmers fell into the following Cu usage categories:

- Used Cu when not needed (tissue Cu concentrations were adequate)
- Did not use Cu when needed
- Used Cu when needed and achieved the desired response (ie return of tissue Cu to an adequate concentration)
- Used Cu but did not achieve the desired response
- Did not use Cu when not needed.

While these survey data are limited, it is apparent that a number of farmers are not confident with their decision making. Many farmers did not undertake Cu tissue determinations before making decisions about supplementation, and few monitored response treatment to assure the desired response.

Thus, despite the existence of basic knowledge sufficient to diagnose the need for Cu supplementation and to monitor treatment response, few farmers apply that knowledge. This suggests that there is a significant opportunity for farmers to improve the cost-effectiveness of their approach to mineral supplementation. It is the authors' proposition that every deer farmer should establish the trace element status (at least Cu, Se and vitamin B_{12}) of their deer herds in terms of blood, liver and pasture data and seek the expertise to interpret data and respond accordingly.

Cobalt

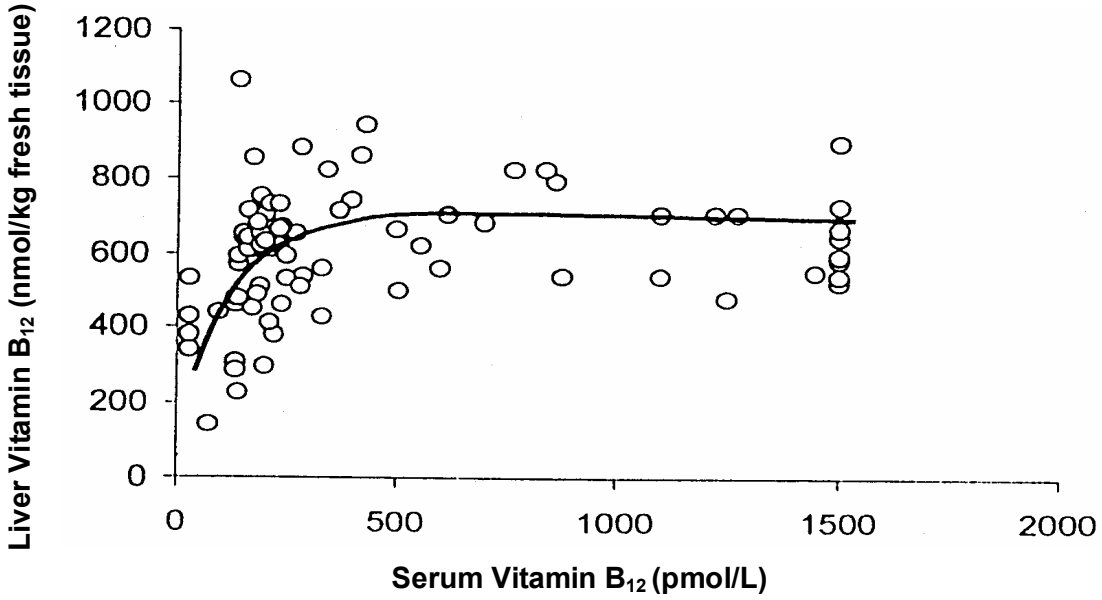
Introduction

Anecdotal evidence suggests that a number of deer farmers supplement their deer with vitamin B_{12}, or use Co in fertilizer, in the belief that it improved or protects the health and productivity of their deer. However there have been no growth responses to Co/vitamin B_{12} supplementation or clinical signs of Co deficiency reported in deer. Indeed, Clark et al (1986) failed to show a growth response even when blood vitamin B_{12} concentrations were in the range at which a growth response occurs in sheep. Note vitamin B_{12}, which contains Co, is a cofactor for enzymes that have a role in energy and protein metabolism and therefore tissue vitamin B_{12} concentrations are used to evaluate the cobalt status in grazing animals.

Distribution

Cobalt is found in all tissues, especially the liver. However, it is the distribution of vitamin B_{12} which is important when considering the metabolism of Co and its effect on the health and performance of deer. Tissue vitamin B_{12} concentrations are directly related to Co intakes. Serum and liver values can range from 50 to 1500 pmol/L and 150 to 1100 nmol/kg fresh tissue, respectively.

Figure 8. The relationship between serum vitamin B_{12} and liver vitamin B_{12} concentrations in deer (From Beatson et al 1999)



There is a non-linear relationship between liver and serum vitamin B_{12} (Beatson et al 1999), meaning that serum concentrations reach a maximum, but liver vitamin B_{12} can reach higher levels without further increases in serum concentrations (Figure 8). Thus, it appears that the liver acts as a store or reservoir for vitamin B_{12}. Vitamin B_{12} readily crosses the placenta and is secreted into milk. Deer milk can contain 3600 pmol vitamin B_{12}/L.

Function

Vitamin B_{12} is essential in the biochemical pathway for producing glucose, the main energy source for animals. The ingested Co is used by the rumen microorganism for the synthesis of vitamin B_{12}, part of which is then absorbed and stored in the liver (Somers and Gawthorne 1969). Vitamin B_{12} is a co-factor for several enzymes namely methylmalonyl, coenzyme A mutase and methionine synthase. Much of the glucose required by deer is derived by gluconeogenesis from propionic acid which is one of

the major volatile fatty acids produced during gut fermentation (Rice et al 1989). The methylmalonyl coenzyme-A mutase converts propionic acid to methylmalonic acid which is then converted to glucose via the tricarboxylic acid and glycolytic pathway. Methionine synthase converts homocysteine to methionine in essential amino acid (Kennedy et al 1992). Cobalt is therefore important in the energy and protein metabolism of deer.

Animal performance

Clinical evidence suggests that Co deficiency on deer farms is uncommon, if indeed it has occurred on New Zealand deer farms. Audige (1995) demonstrated no statistical relationship between blood serum, vitamin B_{12} and growth of young deer from data of a longitudinal study of deer production. Many herds studied had mean vitamin B_{12} in the "responsive" range for sheep (<180 pmol/L). No responses to supplementation have been demonstrated. Despite this, vitamin B_{12} supplementation of deer is not uncommon.

Two studies of the growth rates of vitamin B_{12} treated (injected with 2 mg vitamin B_{12} at 4-6 weekly intervals) and untreated deer have been reported. Growth rates of thirty 8-9-month-old red deer hinds (Trial 1) and 40 10-14-month red deer hinds (Trial 2) grazing pastures containing 0.04 to 0.1 mg Co/kg DM for at least 4 months were recorded along with changes in serum vitamin B_{12} and pasture Co concentrations (Clark et al 1986). No significant growth responses (Trial 1 184 v. 187 g/day; Trial 2 135 v. 146 g/day) to vitamin B_{12} supplementation were observed. The lowest serum vitamin B_{12} concentrations were 83 and 119 pmol/L for Trials 1 and 2, respectively. The treated deer had significantly higher serum vitamin B_{12} concentrations which

were at least double (i.e. 150-400 pmol/L) those of the untreated controls at the end of the study. No data for liver vitamin B_{12} concentrations were reported.

Thus, young red deer hinds appear to be less susceptible to Co deficiency than lambs. While the limited data make it impossible to determine tissue vitamin B_{12} reference ranges for deer, it can be noted that deer with serum vitamin B_{12} concentration of >120 pmol/L and grazing pastures containing 0.05-0.08 mg Co/kg DM are unlikely to respond to vitamin B_{12} supplementation.

Clinical signs of deficiency

No clinical signs of Co deficiency have been reported in deer. In cattle and sheep, chronic loss of condition and failure to thrive despite the apparently ample feed supplies, initially known as "Bush sickness", was the classical clinical syndrome of Co deficiency.

Tissue vitamin B_{12} reference ranges

Further trials investigating growth responses to vitamin B_{12} supplementation in deer grazing very low Co pastures (i.e. <0.04 mg Co/kg DM) are needed in order to establish reliable tissue vitamin B_{12} reference ranges.

Effect of diets of varying Co content on tissue vitamin B_{12} concentrations in deer

A pilot trial (Barry et al 2001) showed that deer grazing chicory had higher liver vitamin B_{12} concentrations than those grazing perennial ryegrass/white clover (PRG/WC) pastures. A subsequent study (Wilson et al 2002) has shown mean liver vitamin B_{12} concentrations of 495 and 490 nmol/kg fresh tissue in deer grazing

chicory in autumn and spring, respectively, compared with 392 and 446 nmol/kg fresh tissue in those grazing PRG/WC. The chicory and PRG had mean Co concentrations of 0.8 and 0.14 mg/kg DM respectively during the autumn grazing period.

The mean liver vitamin B_{12} concentrations of feral red deer was significantly higher than farmed deer (805 and 442 nmol/kg fresh tissue respectively) (Tremaine-Boon et al 2001, 2002). The feral deer were able to browse and graze on plants that provided a greater Co intake when compared to farmed deer grazing pastures.

Supplementation strategy

No supplementation strategy can be recommended. Subcutaneous injections of water soluble or microencapsulated vitamin B_{12} (0.12-0.24 mg/kg liveweight) [SMARTShot B_{12}^{TM}, Stockguard] are likely to increase the vitamin B_{12} status of deer but there are no published data are available for the microencapsulated vitamin B_{12}.

Selenium

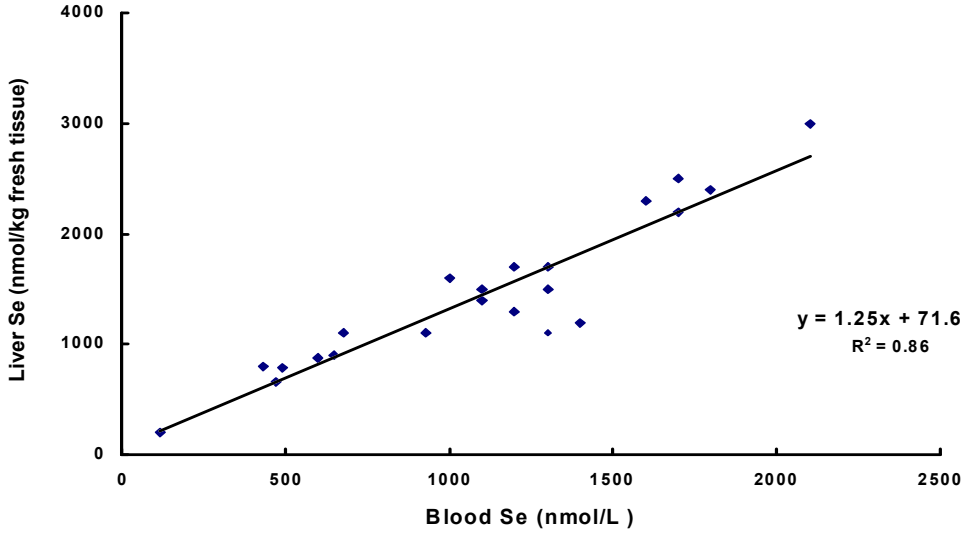
Introduction

Selenium supplementation on deer farms is common (Audige 1995). No growth responses to Se supplementation have been observed but clinical signs, namely white muscle disease in young deer, have been and continue to be reported in deer.

Distribution

High Se concentrations are associated with the liver while a large proportion of the body Se (i.e. about 40-50%) is associated with muscle. Tissue Se concentrations reflect dietary Se intakes. In animals grazing pastures containing 20 to 60 μg Se/kg DM, blood Se can vary from 120 to 400 nmol/L while liver Se range from 100 to 800 nmol/kg fresh tissue (Mackintosh et al 1989). In contrast to Cu and vitamin $B_{12,}$ there is a highly significant linear relationship between blood Se and liver Se concentrations (Figure 9) confirming that Se is stored not only in the liver but also in other tissues, including the blood, as the selenoamino acids when Se intakes are more than adequate to meet Se requirements. Blood Se concentrations directly reflect liver Se concentrations (Grace et al 2000).

Figure 9. The relationship between blood Se and liver Se concentrations in red deer



Thus, assessment of Se status of deer can be achieved either by measuring Se concentrations in liver or blood. Selenium crosses the placenta and is secreted into milk. Milk Se concentrations are related to Se intakes and can be as low as 40 nmol/L in hinds grazing pastures containing <0.03 mg/kg DM (Grace et al 2002, unpublished).

Function

Selenium has an important role as an antioxidant in maintaining the integrity of the cell as the selenoenzyme, glutathione peroxidase, which is able to destroy toxic oxygen metabolites such as superoxide and hydroxy radicals that would damage the lipoprotein membrane structures of cells (McMurray and Rice 1982). Selenium is present as the selenoamino acids selenocysteine and selenomethionine that are incorporated into various proteins. Selenium also maintains the integrity of the immune system (Finch and Turner 1986).

Animal performance

Two selenium response studies have been reported. In the first, 38 red deer weaned calves 3 to 4 months of age grazed pastures containing 30 to 57 µg Se/kg DM. Animals received either 6 oral doses of sodium selenate (Na_2Se0_4 dose 0.1 mg Se/kg liveweight) or 1 ml injection of "Deposel" (Novartis NZ Ltd.) (50 mg Se as barium selenate, $BaSe0_4$) and had similar growth rates to untreated controls (93, 103 and 101 g/day respectively) over 12 months. The mean calculated blood Se concentrations were 400, 800 and 1200 nmol/L for control, Na_2Se0_4 and $BaSe0_4$ treated deer, respectively. A repeat trial the following year using 1 ml "Deposel" (50 mg Se as $BaSeO_4$) and untreated control with 32 weaned red deer calves also found no differences in growth rates (116 v. 117 g/day) (Mackintosh et al 1989).

In a second study, 36 stags aged 4-5 months, grazed pasture containing 10 to 30 µg Se/kg DM. No growth response to Se supplementation was observed when groups of animals (n=16) were treated with 0.5, 1 and 2 ml "Deposel multidose" (Norvatis NZ Ltd) to provide 25, 50 and 100 mg Se, were compared with untreated control animals (n=17) over a 9 month period. The mean blood Se concentrations of untreated deer at days 1, 30, 141 and 377 were 120, 227, 97 and 228, respectively, while those of treated animals increased to >750 nmol/L at day 60 and remained at this level for the duration of the trial (Figure 10). The daily growth rates were 179 and 174 g/day for treated (all dose rates combined) and untreated deer, respectively (Grace et al 2000).

Clinical signs

Young deer, usually less than 6 months of age, affected with white muscle disease have been found to have liver Se concentrations ranging from 73 to 440 nmol/kg fresh tissue, while healthy in contact animals had blood Se concentrations ranging from 46-74 nmol/L (Wilson and Grace 2001). Diagnosis of white muscle disease is confirmed by gross histopathology of skeletal and heart muscle, where areas of pale damaged muscle can be seen.

Tissue Se reference ranges

While the data to establish tissue reference ranges for Se is very limited, based on data of Grace et al (2000) growth responses to Se supplementation are unlikely in deer with blood Se concentrations of >120 nmol/L or in animals grazing pastures containing 20 to 30 µg Se/kg DM. Further Se supplementation growth-response trials in deer with blood Se <100 nmol/L are needed to establish tissue Se reference ranges. In contrast, growth responses occur in sheep and cattle at blood Se concentrations of <250 nmol/L in other words deer are less sensitive to Se deficiency.

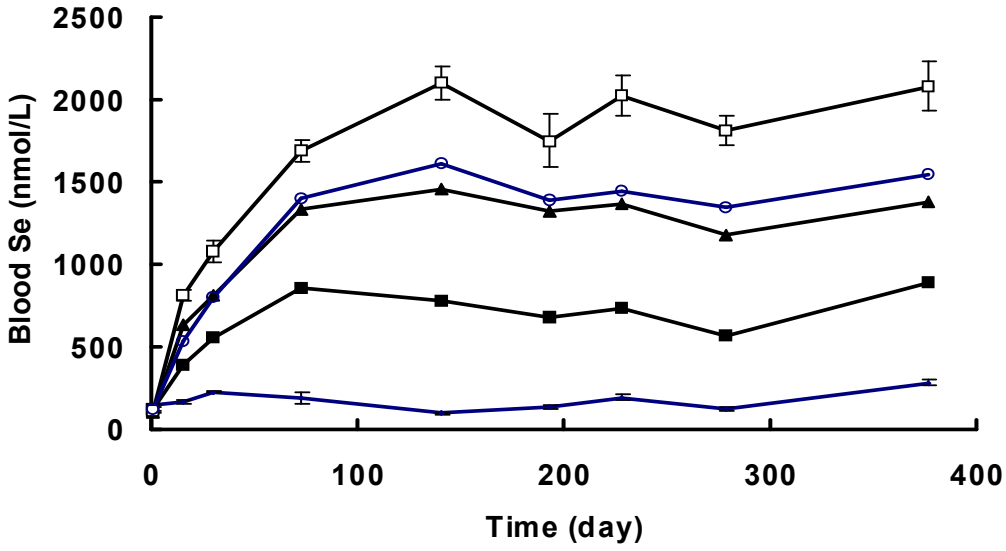
Sampling protocol to assess Se status

Early autumn is the best time to assess the Se status of deer as hinds are about to be mated and young deer have been weaned. Since between–animal variation in selenium concentrations is small, 4-5 blood samples are sufficient. There appears to be no age difference in Se concentrations (Audige 1995). Although the blood Se reference range is based on very limited data concentrations of >120 nmolSe/L are provisionally regarded as being an adequate Se status.

Selenium supplementation strategies

The injectable $BaSe0_4$ product (Deposel multidose, Norvatis) was very effective in increasing and maintaining blood Se concentrations for at least a year (Figure 10) (Grace et al 2000). Application of Se prills, at a rate of 1 kg/ha to provide 10g Se/ha, to pasture is common, and appears effective. Selenium may also be administered orally, at a dose rate of 0.1 mg Se/kg liveweight, in anthelmintic drenches, and by selenised vaccines. None of the above products are licensed for use in deer

Figure 10. The effect of various doses of a BaSeO4 formulation on mean blood Se concentrations in red deer. Control (♦), 0.5 mgSe/kg LW (■), 1.0 mgSe/kg LW (▲ and ○), and 2.0 mgSe/kg LW (□)



Iodine

Introduction

Iodine deficiency in newborn and neonatal deer has been diagnosed clinically on a number of deer farms (Wilson et al 2002), and there are two Animal Health Laboratory reports confirmed goitre.

Distribution

About 80% of the total body I is associated with the thyroid gland and the remaining 20% is distributed in the other tissues (Grace 1994). Iodine intakes and the presence of plant goitrogens, which interfere with I metabolism, influence the I content of tissues (Newton et al 1974).

Metabolism

Iodine deficiency can arise due to a low dietary I intake or as a result of thiocyanates which are released during the chewing and digestion of some brassicas and white clover. Thiocynates interfere with the uptake of I by the thyroid gland and its goitrogenic activity can be abolished by increasing the I intake. Thiouracil is another goitrogen that acts by blocking the synthesis of thyroxine but is not present in New Zealand plants.

Function

Iodine is readily absorbed and rapidly taken up by the thyroid gland for the synthesis of the thyroid hormones T_4 and T_3. The thyroid hormones control the basal metabolic rate and are important for growth and cell differentiation of tissues (Grodsky 1979).

Iodine can cross the placenta and is readily secreted into milk. The foetal demand for I is high.

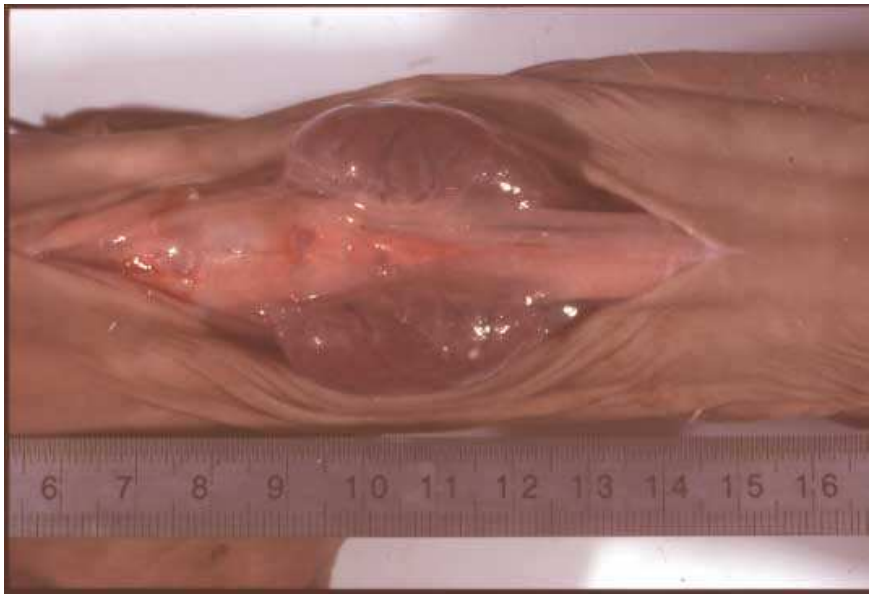
Animal performance

No growth responses to I supplementation in deer have been reported. There is no data on the role of iodine in reproductive losses in deer.

Clinical signs of deficiency

There have been reports of goitre in fawns (Plate 8) (Wilson et al 2002). This can arise when hinds are fed low I diets or low I diets containing goitrogens, such as found in clover and brassica crops, during mid to late gestation. A USA study with white-tailed deer concluded that diet containing 0.26 mg I/kg DM provided an adequate intake for these deer. Low thyroxine concentrations reduce metabolic rate, and therefore heat production and physical vigour. Iodine deficient newborn deer may fail to survive. Lack of hair may be seen. Abortion has been observed with goitre of the foetus in fallow deer (Wilson et al 2002). One I supplementation response trial involving 2600 hinds on 4 properties showed no difference in calf survival rates between treated and control groups (Wilson et al 2002).

Plate 8. Enlargement of the thyroid gland of a late term foetus aborted because of I deficiency



Criteria to assess I status

At present there are no suitable tissue I reference ranges or biochemical criteria to assess the I status of deer.

As an example in sheep a thyroid (g): liveweight (kg) ratio of >0.4 in lambs is indicative of an I deficient flock.

Iodine supplementation strategies

An intramuscular injection of an iodised oil (4-8 mg I/kg liveweight) [Flexidine, Bomac Laboratories] is effective in increasing the serum I concentrations in deer for at least 6 months. Oral potassium iodide (400 mg/head) has been used effectively in hinds during the last half of pregnancy to prevent goitre in fawns.

Acknowledgements

The coloured photographs were provided by Peter Wilson, Massey University.

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Popular summary for The Deer Farmer

Trace element nutrition of deer

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Copper (Cu), selenium (Se), cobalt (Co) and iodine (I) are the important trace elements for farm animals, including deer. Adequate trace element nutrition is important to ensure good health and animal performance. While there has been a dearth of information on trace element nutrition in deer, recent studies on Cu have provided new information on Cu requirements, factors influencing Cu status and the prevention of Cu deficiency.

Copper has an important role in the biochemistry and physiology of animals. It is essential for nerve function, bone growth and development, maintaining the integrity of the immune system and the metabolism of Fe iron. While Cu is found in all tissues it is stored in the liver from whence it maintains blood Cu concentrations of 8-20 µmol/L for normal tissue function provided intakes are adequate. If Cu intakes are low liver Cu stores become depleted and blood Cu concentrations can decrease to 3-5 µmol/L, or less, and disease and poor growth may result. Changes in liver and serum Cu concentrations can be used to assess the Cu status of deer and to diagnose Cu deficiency.

During pregnancy Cu is transferred across the placenta and stored in the liver of the foetus. The liver Cu concentration of the foetus/neonate can be 10-30 times greater than that of its dam (e.g. 5650 v 166 µmol/kg fresh tissue). The Cu concentration of milk is low (i.e. 2.8-3.2 µmol/L) and is not increased by Cu supplementation or increased dietary Cu intake so supplementation should be given to hinds earlier in pregnancy to ensure an adequate Cu status in their calves.

On most deer farms there is a marked seasonal change in the Cu status of deer as liver and serum Cu concentrations are high in the autumn and are at their lowest in late

winter/early spring. As the Cu status of the hind markedly influences the Cu status of the foetus the impact of the seasonal decline in Cu status means that new born and young deer could be at a greater risk of being Cu deficient.

There have been very few trials where a significant growth response to Cu supplementation has been reported. Copper deficiency in deer has been diagnosed from clinical signs such as enzootic ataxia (a nerve disorder) and osteochondrosis (a bone disorder). Deer with liver and serum Cu concentrations being <60 µmol/kg fresh tissue and <5µmol/L, respectively, are considered to be Cu deficient that is they at risk of showing signs of Cu deficiency such as nerve and bone disorders which then impact on movement, feed intakes and animal performance. Liver and serum Cu concentrations >100 µmol/kg fresh tissue and >8 µmol/L, respectively, reflect an adequate Cu status.

Copper supplementation as CuO needles given to pregnant hinds in late autumn/early winter will improve their Cu status and more importantly the Cu status of their offspring. Likewise, grazing hinds on pasture 4 weeks after it has been topdressed with 12 kg copper sulphate/ha will also maintain an adequate Cu status of hinds and their fawns. Feeding forages such as chicory with Cu concentrations >11 mg/kg DM will improve the Cu status of deer. Copper–EDTA injection is licensed for use in deer, but tissue reactions at the injection site are common.

Selenium deficiency occurs on a few deer farms. Signs are of white muscle disease causing stiffness and muscle pain, and are often fatal. Selenium functions as an antioxidant and maintains the integrity of cells and the immune system. Cobalt deficiency has not been reported in deer but as a constituent in Vitamin B_{12} it is important in the energy and protein metabolism of deer. Iodine is needed for the formation of thyroxine which controls basal metabolism and cell differentiation.

No growth responses to Se, Co and I supplementation in deer have been observed and there are no tissue reference ranges to assess the Se, Co (Vitamin B_{12}) and I status of deer. Selenium and I deficiency in deer are diagnosed from clinical signs namely white muscle disease in the case of Se and goitre in the case of I.

Plate 1. Enzootic Ataxia.
Yearling hind showing inability to stand upright.

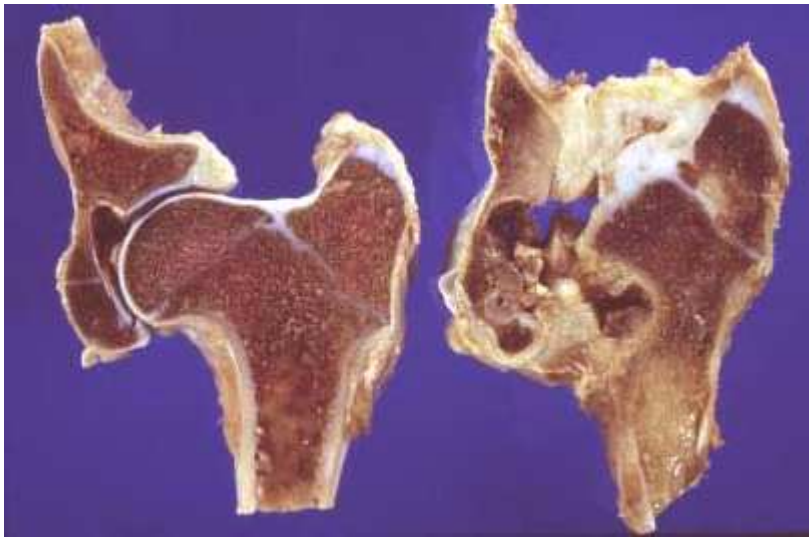

Plate 2. Osteochondrosis.
Cross-section of hip joint showing the total collapse of bone and cartilage.
Cu deficient deer on the right.

Comments on current research and funding on trace element nutrition of deer

1 New Zealand Research

In many aspects of trace element nutrition of deer New Zealand is leading the way for farmed deer but there is a need to:

- Establish more robust trace element tissue reference ranges based on supplementation/response trials and clinical signs to assess trace element status
- Establish and evaluate the best protocols to prevent trace element deficiencies through fundamental research of trace element metabolism in deer, and a range of methods for maintaining tissue trace element adequacy.
- Develop a culture amongst deer farmers to use a scientific approach to resolve trace element deficiency problems

2 Overseas Research

Presently to the authors' knowledge there is no overseas research activity to compliment the N Z trace element research programme

3 Linkages to FoRST and other funding

- There is no FoRST funding for trace element research in deer
- FertResearch has and is continuing to fund Cu research in deer
- Norvatis has funded Se trials on the development of a long acting injectable Se (Deposel multidose)
- Bomac Laboratories Ltd funded I research and the use of iodised oil (Flexidine) to increase the I status of deer and its impact on reproduction and novel copper supplement products
- Massey University has funded some trace element research from internal sources and revenue generated by researches

- Wrightson Nutrition has contributed to evaluation of forages and their effect on animal trace element status
- The New Zealand Game Industry Board Research Trust contributed to overheads in a pilot trial of forage effects
- The Manawatu Branch of the NZDFA contributed toward forage evaluations.

4 Potential funders

- Livestock industry consortium (Dairy/Meat/Wool/Deer) may fund animal production trace element supplementation projects
- Animal health companies developing new animal remedies, particularly Cu products
- Seed/Nutrition companies for various forage effects.

5 Deer trace element nutrition research capabilities

AgResearch Grasslands

- Dr Neville Grace (BAgSc, MAgSc, PhD)
  Scientist – Animal Nutrition especially trace elements
- Other scientists with interests in nutrition and biochemistry
- Analytical laboratories
- Aorangi deer farm -16 hectares
- Flock House deer farm – 40 hectares

AgResearch Invermay

- Dr Colin Mackintosh (BVSc, PhD)
  Veterinary scientist – deer health
- Invermay deer farm - 122 hectares wintering 1100 deer; indoor pen feeding for 80-100 deer; isolation building for 8-12 deer

Massey University

- Professor Peter Wilson (BVSc, PhD, MACVSc)
  Veterinarian, deer health and diseases, production and welfare
- Other staff with expertise in veterinary and animal sciences
- Post graduate students
- Research/diagnostic laboratories
- Massey University Deer Research Unit – 24 hectares with 100 hinds and replacements. Special facilities for digestion/metabolism studies including 12 hand-reared deer with rumen, abomasal and oesophageal fistula
- Veterinary clinic with full surgical facilities and surgical expertise

Lincoln University

- Dr Alistair Nicol (PhD) and Professor Andrew Sykes (PhD, DSc)
- Animal nutrition including trace elements
- Deer farm – 20 hectares

Diagnostic Laboratories

Determination of tissue trace elements and pathology

- Alpha Scientific
- Gribbles Veterinary Pathology
- LABNET Invermay

Research projects and priorities

A number of key areas of deer trace element nutrition need to be considered for further research and funding

(1) Copper

(1.1) Copper x molybdenium interaction

Increasing Mo intakes decrease Cu absorption and utilisation in cattle and sheep. The importance and magnitude of the Cu x Mo interaction in deer is not known.

A study funded by FertResearch and DEEResearch is planned to start in February 2003.

(1.2) Dietary Cu requirements

A knowledge of dietary Cu requirements enable pastures and forages to be readily evaluated, from their Cu concentrations, in terms of whether they will provide an adequate Cu intake for deer. As Cu nutrition is complex this assessment needs to be made bearing in mind that dietary factors such as increasing Mo intakes increase dietary Cu requirements.

Recent studies have shown that as forages containing 10-17 mg Cu/kgDM and <0.5 mgMo/kg DM will maintain adequate liver Cu stores in late winter and meet the Cu needs of grazing deer. These studies need to be extended and funded to provide information to assist deer farmers to improve the Cu status of their deer by the use of high Cu forages such as chicory.

It is necessary to conduct some research on commercial deer farms to verify relevance. Establishing a profile of trace elements involving diet and animal tissues, from all age groups on a number of farms, under various grazing and management strategies would provide significant information about comer needs and their management.

(1.3) Establish more robust trace element tissue reference ranges

Trace element tissue reference ranges are paramount in order to assess the trace status of deer and to diagnose trace element deficiencies.

A proposed set of tissue trace element reference ranges for Cu in deer have been published (Wilson and Grace 2002).

However the present database is limited and needs to be extended by setting up a protocol and funding where by deer veterinarians and animal health laboratories are able to and are strongly encouraged to follow up cases where trace element deficiencies are diagnosed by gross pathology or histopathology. These reports must include trace element analyses of the liver and blood if possible of affected and in-contact deer. Pasture trace element concentrations of the areas grazed by affected herds should also be determined.

A central database needs to be established to collate all the information.

Deer farmers should monitor the trace element status of their herds as part of an animal health programme.

The following is a shortlist of recommended research topics:

- Copper
    - Establishing the effect of dietary interactions such as Fe, S and Mo on tissue Cu concentrations
    - Profiling trace elements on commercial deer farms
    - Using data, model Cu to provide for more robust and cost-effective management of this element
    - Evaluate different forage species for Cu (and other trace elements)
    - Validate data on Cu topdressing of pastures on commercial farms
    - Evaluate novel and alternative supplements (commercial products such as pour-on Cu animal remedies).

- Selenium
    - Establish more robust tissue reference ranges by growth response trials and relationship between disease and tissue Se concentrations
    - Evaluate relationship between dietary Se and animal tissue Se, that is dietary Se requirements
    - Evaluate alternative Se supplementation methods (for example pour-on products)

- Cobalt
    - Establish tissue reference ranges by Vitamin B_{12} supplementation growth response trials
    - Evaluate dietary Co requirements

- Iodine
    - Evaluate the role of I in reproductive loss in herds in terms of foetal loses and high perinatal mortalities

A bibliography of relevant publications

Grace ND. *Managing trace element deficiencies*. Pp 70. New Zealand Agriculture Research Institute Ltd, Palmerston North, 1994

Wilson PR, Grace ND. A review of tissue reference values used to assess the trace element status of farmed red deer (Cervus elaphus). *New Zealand Veterinary Journal* 49, 126-32, 2000

Grace ND, Wilson PR. Trace element metabolism, dietary requirements, diagnosis and prevention of deficiencies in deer. *New Zealand Veterinary Journal*, submitted