

The copper nutrition of grazing deer

N. D. GRACE¹, P.R. WILSON² and A.M. NICOL³

¹AgResearch Limited, Grasslands, Private Bag 11008, Palmerston North

²Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Private Bag 11222, Palmerston North

³Animal and Food Sciences Division, Lincoln University, P O Box 94, Canterbury
neville.grace@agresearch.co.nz

Introduction

The first deer farms were established in New Zealand about 30 years ago and much of the early deer research was focussed on general nutrition, infectious diseases, management and husbandry practices, while little attention was given to trace elements. Limited trace element studies have identified copper (Cu) as the most important trace element for deer in terms of observed clinical signs of deficiency, namely enzootic ataxia and osteochondrosis, and their possible impact on animal performance (Wilson *et al.* 1979; Thompson *et al.* 1994). To ensure that the Cu status of deer is adequate, and to determine if a Cu supplementation strategy is necessary, it is important to understand the distribution, function and metabolism of Cu, as well as dietary Cu requirements and criteria to diagnosis Cu deficiency.

Keywords: *Cervus elaphus*, copper, deficiency, dietary requirements, fertiliser, forage, red deer, supplementation strategies

Distribution

Copper 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.* 1986a; Mackintosh 1992; Wilson & Audigé 1998; Grace 1999; Grace *et al.* 2001; Tremain-Boon *et al.* 2001, 2002). Increasing Cu intake or Cu supplementation will increase serum Cu if the concentrations are low (eg <8 µmol/l) to begin with, but will not have any influence if they are already high. However, liver Cu concentrations will increase after an increase in Cu dietary intake or supplementation when this is greater than the daily Cu requirements, demonstrating its role as the primary storage organ for Cu.

Figure 1 Effect of liver Cu concentration of hinds on the liver Cu concentration of their fawns at 3 weeks of age. (Grace & Wilson unpublished data).

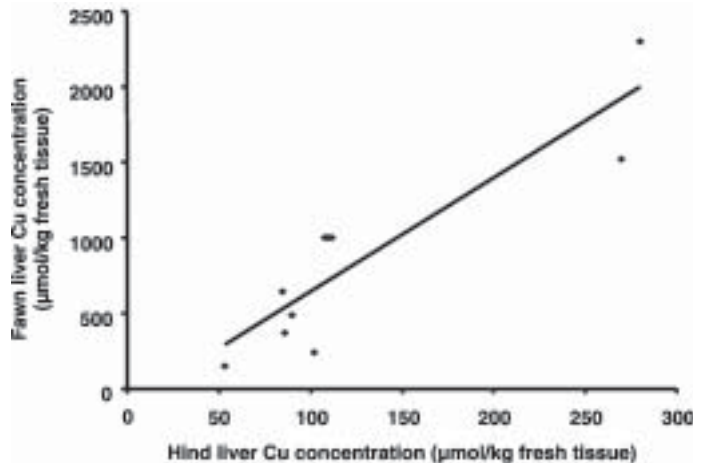
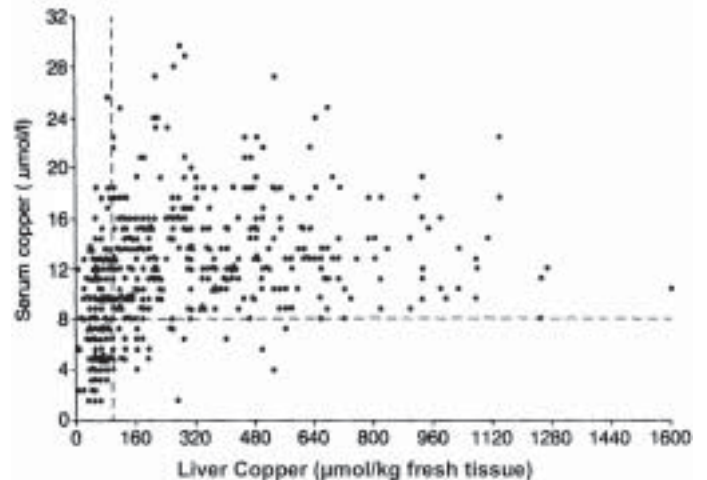


Figure 2 The relationship between serum Cu and liver Cu concentrations in deer. (Mackintosh *et al.* 1986b.).



Copper is transported across the placenta. Liver Cu concentrations of the foetus/neonate are much greater than those of the hind (5650 v 166 µmol/kg fresh tissue) (Reid *et al.* 1980). Hinds with a high Cu status, that is, a high liver Cu concentration, will give birth to fawns with a high liver Cu concentration or Cu store (Figure 1). Milk Cu concentrations range from 2.8 to 3.2 µmol/L

and are not influenced by Cu supplementation or intake (Grace & Wilson unpublished data).

Season has a marked effect on the status, with the lowest serum and liver Cu occurring in late winter/early spring (Wilson & Audigé 1998; Grace *et al.* 2001). The factors influencing this decline in Cu status are not well understood. Increasing intakes of Mo, Fe and soil have been suggested, or it may be associated with grazing management or changes in deer Cu metabolism, but to date there is a dearth of data on the magnitude of the impact of these factors on Cu nutrition in deer (Wilson & Grace 2002).

There is a relationship between liver and serum Cu concentrations, which is illustrated in Figure 2. 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 concentrations are maintained at the expense of the liver Cu stores. As these stores 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.* 1986b).

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.

A central nervous system disorder, enzootic ataxia, occurs in deer and it is similar to the condition described in lambs. 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). It has been suggested that Cu deficiency decreases the activity of several enzymes, including cytochrome oxidase, reducing the amount of phospholipids synthesised which are important components of the myelin sheath.

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 sub-stratum, which serves as a template for the deposition of calcium (Ca) and phosphorus (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).

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 & Jones 1986). It is therefore possible that Cu deficient deer will be more susceptible to infectious diseases such as yersiniosis and tuberculosis although there has been no research into this effect.

Ferroxidase (ceruloplasmin), a blood enzyme, oxidises the ferrous iron (Fe²⁺) to ferric iron (Fe³⁺) allowing the mobilisation of iron (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 12 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 *et al.* 1991; Harrison & Familton 1992; Wilson & Grace 2001; Nicol *et al.* 2002). 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 10 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 of herds where response occurred, although concentrations were in the “marginal” range in most deer. It seems that in deer, for a liveweight gain response to Cu supplementation to occur, their mean serum Cu concentrations must be very low, (<1.0 µmol/L) and remain so for at least several months.

These observations demonstrate the difficulty of obtaining good Cu supplementation animal response data to establish reference ranges, and in predicting responses to Cu supplementation in deer. In sheep and cattle trials, liveweight responses to Cu supplementation have been observed where the absorption and utilisation of Cu has been reduced by increased Mo intakes (ie pastures containing >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 may be at risk of Cu deficiency syndromes, although it is not possible to predict whether or not they will actually occur. Further, it is currently not possible to establish tissue Cu reference ranges to accurately predict whether a production response will occur to Cu supplementation (Wilson & Grace 2001).

Signs of deficiency related to tissue Cu concentration

Clinical Cu deficiency in deer is therefore most likely to be diagnosed from clinical disorders such as enzootic ataxia and osteochondrosis. Enzootic ataxia occurs most commonly in yearling deer, the youngest reported case being 5 months of age (Barlow *et al.* 1964; Wilson *et al.* 1979; Audigé *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 nervous system, which then result in a lack of co-ordination of the hind quarters. This condition is associated with mean liver Cu concentrations of < 60 µmol/kg fresh tissue, except in one case where liver Cu was 86 µmol/kg fresh tissue (Wilson & Grace 2001). The limited amount of serum Cu data showed mean concentrations of 1.5 µmol/L in affected deer, and 2.5 µmol/L in clinically unaffected in-contact animals (Wilson & Grace 2001).

Osteochondrosis of young deer has been observed in Cu-deficient animals (Thompson *et al.* 1994; Audigé *et al.* 1995). This syndrome causes arthritis, since the bone tissue under the 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 and cause a “bunny-hopping” gait. Osteochondrosis is associated with mean liver Cu concentrations of < 42 µmol/kg fresh tissue and mean serum Cu concentrations of < 3.0 µmol/L (Thompson *et al.* 1994; Audigé *et al.* 1995). The mean serum Cu concentrations of unaffected in-contact deer were < 3.3 µmol/L.

It must be cautioned that these associations are from only small numbers of animals, and supplementation history prior to the investigations was largely unknown. Further studies, preferably involving field investigations where clinical signs are observed, would provide data for more robust reference values.

Tissue Cu reference range to assess Cu status in deer

The association of tissue Cu concentrations with clinical signs of Cu deficiency enables tissue Cu reference ranges to be established as aids to diagnose Cu

deficiency. From the limited data available the following criteria have been proposed (Wilson & 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 are 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 “deficient”, “marginal” and “adequate” animal Cu status, respectively.

Sampling protocols to assess the Cu status of deer

Practical considerations for diagnosis and tissue sampling protocols to assess Cu status of deer have been reviewed recently (Wilson & Grace 2001, 2002).

As there is a marked effect of season on liver and serum Cu concentrations on many deer farms (Wilson & Audigé 1998; Grace & Wilson unpublished), 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, therefore, the Cu status of deer herds.

Five to 8 liver samples/group, collected by biopsy in the live animal (Familton 1985; Wilson 2000) or at slaughter 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). Low serum Cu concentrations (eg < 5 µmol/L) of some deer at weaning time, at least 8 samples/group, would likely reflect a seriously low Cu status on the farm, given values are likely to fall over winter. 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 or blood in July/September would be required to determine 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 Cu stores, thereby increasing their risk of clinical Cu deficiency (Grace & Wilson unpublished data). A recovery in the Cu status of deer usually occurs in late spring or early summer.

Dietary Cu content and Cu status of deer

The Cu content of forages and pastures grazed by deer, assuming adequate DM intake and a limited effect from dietary constituents, such as Mo and Fe, has an impact

on their Cu status. A comparison of the liver Cu concentrations in May of deer grazing chicory, containing 10.5 mg Cu/kg DM, and ryegrass/white clover pasture, containing 7.6 mg Cu/kg DM, showed animals on chicory had greater liver Cu stores (461 v 175 $\mu\text{mol/kg}$ fresh tissue) (Barry *et al.* 2001). In another study, with deer on a marginally Cu deficient farm where pasture Mo concentrations were low (0.25 mg/kg DM), young animals grazing chicory during autumn and spring had about a 5 fold increase in liver Cu concentrations (93 v 500 $\mu\text{mol/kg}$ fresh tissue) in early summer, when compared with deer grazing a ryegrass/white clover pasture (Wilson *et al.* unpublished). Further, at the end of winter period only 6 of the 23 deer on the chicory had liver Cu concentrations <100 $\mu\text{mol/kg}$ fresh tissue, while in the case of the deer on the ryegrass/white clover pasture, 17 of the 22 had liver Cu concentrations <100 $\mu\text{mol/kg}$ fresh tissue, that is, in the “marginal” Cu deficient range (Wilson *et al.* unpublished data).

Cu supplementation strategies

If the Cu status of a herd is inadequate, and/or clinical signs of Cu deficiency have been observed, then a Cu supplementation strategy should be implemented. The various Cu supplementation options have recently been reviewed (Wilson & Grace 2002).

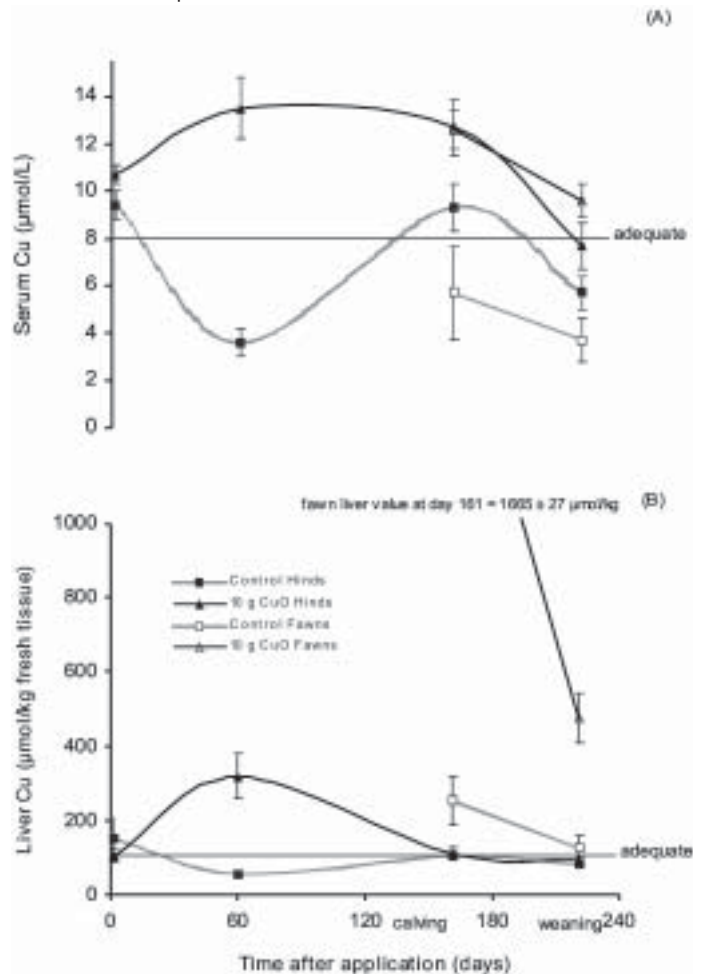
Copper oxide needles

The most commonly used supplement is CuO wire needles (Booth *et al.* 1989; Harrison & Familton 1992; Wilson & Audigé 1998; Beatson *et al.* 2000; Grace & Wilson unpublished data). There have been a few efficacy studies of this product in deer. Recent data are presented for weaner stags and pregnant hinds. When weaner stags were given 10 g CuO needles in March, and changes in liver Cu concentrations of untreated and treated animals were monitored monthly over 6 months, the CuO increased and maintained adequate liver Cu concentrations for about 5 months. A peak liver Cu concentration of 850 $\mu\text{mol/kg}$ fresh tissue being reached at 2 months after dosing.

Pregnant hinds were mated in March–May and treated with 10 g CuO needles in late July (Grace & Wilson unpublished data). 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 (Figure 3). (It should be noted that osteochondrosis of young deer had been observed previously on this property). Thus maintaining serum Cu in pregnant hinds 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

Figure 3 Effect of CuO needles given in July on the (A) serum Cu and (B) liver Cu concentrations of yearling hinds and their fawns. (n=11) (Grace & Wilson unpublished data). Deer with serum and liver Cu concentrations of > 8 $\mu\text{mol/L}$ and > 100 $\mu\text{mol/kg}$ fresh tissue are considered to have an adequate Cu status.



concentrations, supplementing hinds during lactation is unlikely to effect the Cu status of their fawns.

Copper injection

An injectable Copper Ca EDTA can be used in deer and was evaluated by Harrison *et al.* (1989). The safe dose rate for 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 $\mu\text{mol/kg}$ fresh tissue and efficacy of the product was estimated to be 8 to 12 weeks.

Copper topdressing

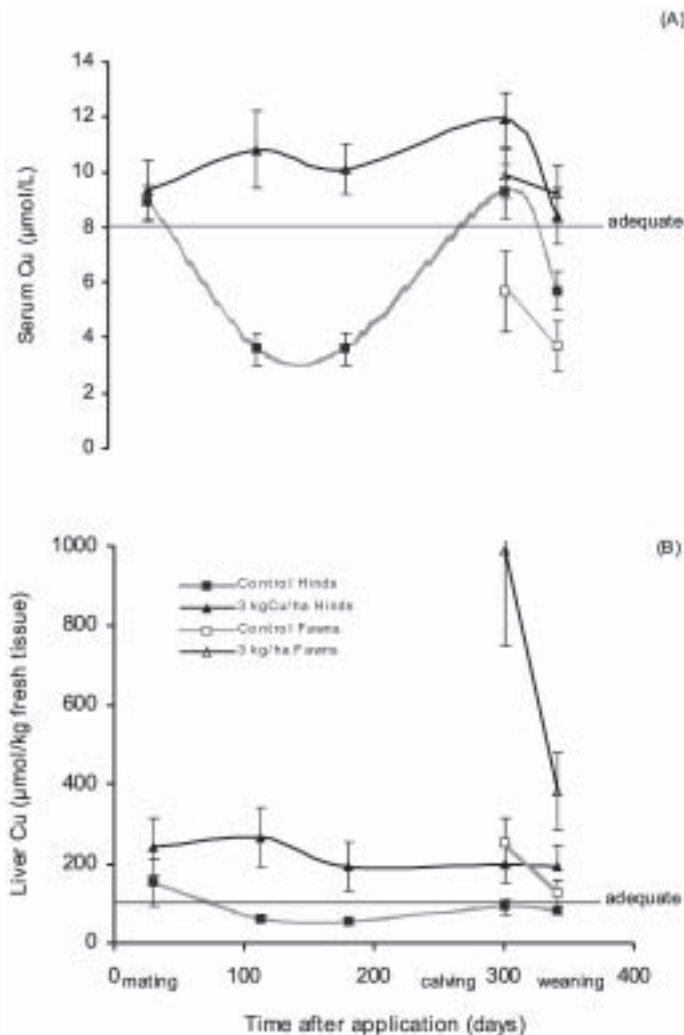
Copper topdressing, applied in March at the rate of 12

kg copper sulphate ($\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$)/ha [3 kg Cu/ha], increased pasture Cu concentrations to 45-60 mg Cu/kg DM. The pasture Mo concentrations varied from 0.5 to 1.3 mg/kgDM. 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 also provided elevated serum and liver Cu concentrations in hinds through gestation and lactation, as well as in their fawns from birth to weaning (Figure 4). The serum and liver Cu concentrations of the untreated hinds were $<4\mu\text{mol/L}$ and $<70\mu\text{mol/kg}$ fresh tissue, respectively.

The autumn application of 12 kg copper sulphate/ha (3.0 kg Cu/ha) was a very cost effective and easy approach to increase and maintain a higher 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 Cu concentrations being greater than 45 mg Cu/kg DM for 50 to 100 days when the deer graze the pasture. It is important not to graze pastures until 3-4 weeks after Cu application. This delay in grazing after application allows the Cu to be washed into the soil by the rain (10-20 mm) 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/kg DM.

The uptake of Cu by 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 whether this approach is suitable to increase the herbage Cu concentrations on a particular deer farm. Monitoring pasture may also provide a means for predicting a response in deer tissue Cu concentrations to Cu topdressing. It should be noted that this research was performed on only one property, and needs to be repeated before industry wide recommendations can be made. Further it should be noted that as sheep

Figure 4 Effect of Cu topdressing (3kg/ha) pasture in March on the (A) serum Cu and (B) liver Cu concentrations of yearling hinds and their fawns. (n=11) (Grace & Wilson unpublished). Deer with serum and liver Cu concentrations of $> 8\mu\text{mol/L}$ and $> 100\mu\text{mol/kg}$ fresh tissue are considered to have an adequate Cu status.



are most sensitive to Cu toxicity they could be put at risk if they graze pastures topdressed with 12 kg copper sulphate/ha (Grace *et al.* 1998).

Feeding high Cu forages

Grazing deer on forages such as chicory and red clover containing at least 11 mg Cu/kg DM in the autumn will ensure that they will have increased liver Cu stores in preparation for the winter. Likewise, feeding high Cu supplements such as red clover hay during the winter will also maintain an adequate Cu intake, even during the late winter and early spring.

Comments on Cu supplementation

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

Where no clinical signs of Cu deficiency have been observed, but the Cu status of a proportion of the herd at certain times of the year are in the “deficient” or “marginal” range, decisions on Cu supplementation may need to be made on grounds of insurance. Copper supplementation under these situations will raise the Cu status of the herd and remove the animals in the ‘at risk’ category, but on current evidence production responses are unlikely to be observed and the cost of Cu supplementation will not be recovered.

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