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Major trace elements limiting livestock performance in New Zealand

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Key Points

- Many New Zealand soils are naturally deficient in cobalt and/or selenium and/or copper, or are naturally high in molybdenum. Livestock grazing pasture grown on such soils may be deficient in one or more of these trace elements.
- In the 1940s and 1950s, New Zealand researchers were at the forefront of research to define the cause of trace-element related ill-thrift and clinical diseases like white muscle disease, peat scours and enzootic ataxia.
- New Zealanders have devised production-related reference ranges for blood and liver copper, vitamin B_{12} and selenium that are used for the diagnosis and prevention of deficiencies.
- A range of supplementation procedures has been devised, from topdressing or spraying pasture to direct animal supplementation, to suit the range of livestock management systems found in New Zealand.
- Trace-element monitoring programmes are now a routine procedure for farmers grazing cattle, sheep, and deer on trace-element deficient land.
- Copper deficiency is the main trace-element deficiency diagnosed in deer.

History

Shortly after domestic livestock were introduced to New Zealand, it was realised that in some areas stock did not thrive, or suffered particular diseases not present in other areas. Large parts of New Zealand were subsequently discovered to be trace-element deficient and these deficiencies have been a significant part of the New Zealand agricultural scene ever since. This stimulated considerable ground-breaking research by New Zealand soil scientists, agronomists, animal scientists and veterinarians. As a result of this research we now have a clear understanding of where deficiencies occur, how they present, how they limit animal performance, and how they can be diagnosed and prevented. The main trace-element deficiencies of livestock in New Zealand are cobalt, selenium and copper. This paper is a commentary of trace element research in farmed livestock from a New Zealand perspective.

Cobalt

Cobalt deficiency largely manifests as ill-thrift, particularly in young stock. As early as 1893, various names were coined for this disease in New Zealand such as “Mairoa dopiness”, “bush sickness” and “Morton Mains disease” (Andrews 1961). However, it was not until the mid 1930s, following the discovery by Australian scientists that cobalt in limonite was effective in curing similar diseases in that country, that cobalt was found to be the cause of these wasting diseases in New Zealand (Underwood and Filmer 1935).

Selenium

In 1957, selenium was discovered to be an essential trace element for animals and within a couple of years, New Zealand workers documented a wide range of clinical and subclinical conditions in sheep and cattle that responded to or were prevented by selenium supplementation. They were the first to determine that white muscle disease in New Zealand was selenium rather than vitamin-E responsive and they also described and defined a selenium responsive ill-thrift syndrome in lambs, hoggets, adult ewes and cattle of all ages, and a selenium responsive infertility in ewes (Hartley and Grant 1961, later reviewed by Andrews et al 1968). These selenium responsive conditions were found to be widespread on pumice soils in the North Island and sandy soils in both islands and, subsequently, maps have been published classifying soil types as severely, mildly or not affected (Andrews et al 1968). These early workers also developed supplementation strategies to prevent deficiencies and many of these strategies are still used extensively today (Andrews et al 1968).

Copper

It was in the 1940s, a few years after copper deficiency was first reported as a problem in grazing animals overseas, that the copper responsive disorders such as enzootic ataxia or “swayback” in lambs, osteoporosis of lambs, and ill-thrift and peat scours in cattle were reported in New Zealand (Cunningham 1950). Like cobalt and selenium, the major areas of deficiencies were soon identified and published and the causative role of molybdenum in peat scours was defined (Cunningham 1950). Work published in 1946 described how these conditions could be prevented by topdressing copper sulphate onto pasture in the autumn (Cunningham and Perrin 1946).

The effect of deficiencies on productivity

An insight into the severity of trace elements on farm animal performance is graphically displayed in early papers describing their deficiencies in New Zealand.

Cobalt

In severely deficient areas, ill-thrift was so marked that calves and lambs died (Andrews 1961). In those that survived, growth rates were markedly depressed and often zero over summer months compared with gains of approximately 1.5 lbs/week (97 g/day) for cobalt supplemented lambs (Andrews and Anderson 1955; Andrews and Stephenson 1966). Today, cobalt deficiency is mainly confined to lambs, because severely deficient areas, where cobalt deficiency was seen in adult sheep and cattle as well as in lambs, have had cobalt fertiliser applied for many decades. Cobalt responsive ill-thrift in lambs is still seen where cobalt fertiliser applications or vitamin B_{12} injections to lambs are haphazard. Liveweight gains to supplementary vitamin B_{12} injections of up to 180 g/day have been reported as recently as 1985 (Clark et al 1985a). Another manifestation of cobalt deficiency is ovine white liver disease, which has been reported from a number of areas of New Zealand including the north and east of the North Island, and in south Canterbury (as reviewed by Sutherland et al 1979).

Selenium

In the 1950s, selenium deficiency was found to cause early embryonic mortality in ewes (reviewed by Andrews et al 1968). On severely deficient farms, selenium administration before mating

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DM Dry matter

increased lambing percentages from 25 to 90% and from 80 to 120%, and the percentage of dry-dry ewes decreased markedly. On some of the farms there were also cases of white muscle disease and selenium responsive ill-thrift. Morbidity rates for white muscle disease of up to 25% and mortality rates of 15% were reported. In outbreaks precipitated by droving, morbidity rates approached 100% and more than 50% of affected animals died (Hartley and Dodd 1957). Selenium responsive ill-thrift was also demonstrated to be a very significant production limiting deficiency in growing sheep. Over a 20–28 week period, severe selenium responsive ill-thrift was associated with mortality rates of 25–45%, and mean liveweights of survivors were depressed by 6.8–11.4 kg compared with supplemented controls (as reviewed by Sheppard et al 1984). Selenium deficiency in cattle mainly presents as ill-thrift. Growth rate responses to selenium supplements of 50–280 g/day have been recorded, compared with unsupplemented calves (Fraser and Wright 1986). A severe clinical syndrome of ill-thrift, diarrhoea and some mortalities has been described in the autumn and winter months in both beef and dairy calves (Hartley and Grant 1961). In contrast, nutritional myopathy occurred at a very low rate in both beef and dairy cattle grazing severely selenium deficient soils (Hartley and Grant 1961). In adult cows grazing severely selenium deficient pasture, supplementation increased milk production and fertility (Hupkens van der Elst and Watkinson 1980; Fraser et al 1987; Tasker et al 1987).

Copper

Copper deficiency was described as 'simple' if stock were showing signs of deficiency whilst grazing pasture with less than 5 mg/kg dry matter (DM) of copper and less than 3 mg/kg DM of molybdenum. 'Complicated' copper deficiency occurred when animals grazed pasture with a high molybdenum content (greater than 3 mg/kg DM) (Cunningham et al 1956). These authors found, from an extensive survey of copper and molybdenum herbage levels, that 'simple' copper deficiency was uncommon, whereas approximately 2.1 million hectares of the North Island and smaller areas of the South Island produced pastures that had molybdenum concentrations sufficient to induce 'complicated' copper deficiency.

Most of the early work describing copper deficiency in cattle and sheep in New Zealand was descriptive and only a limited number of controlled trials quantifying effects were published. For 'simple' copper deficiency, Cunningham (1950) described adult cattle as anaemic, unthrifty and poorly grown, their reproductive capacity was reduced, and the coat colour of some red animals changed to a bleached 'yellowish' red. The growth of calves was slower than normal, their bones fractured easily, and some cases of ataxia were observed. For peat scours or 'complicated' deficiency he stated: "calves are difficult or impossible to rear because of unthriftiness, debilitating spring scouring, increased susceptibility to internal parasites, and proneness to fracture of bones. Adult cattle are anaemic and debilitated, the coat is rough and staring and the colour of the black animals becomes tinged with dull red. The animals scour profusely and persistently on flush pastures of spring and become extremely debilitated and produce very much less meat or milk." Six young cattle over a 9-month period grazing a low copper and high molybdenum pasture gained 3 lbs (1.36 kg) whereas another six animals supplemented with 20 mg oral copper three times a week over the same period gained 111 lbs (50.3 kg) (Cunningham 1950).

Most of the published trial work in cattle on the effect of copper on growth is from overseas. In Northland, New Zealand, copper deficiency was found to be the cause of a Heinz-body haemolytic anaemia in the postpartum period. It often affected large numbers of cows in a herd, and some cows died. Following the widespread introduction of copper supplementation and reduced applications of molybdenum in fertiliser, this debilitating condition is seldom seen today (Smith 1973).

Development of biochemical criteria to determine deficiency status

Initial diagnoses of trace-element deficiency were based on comparing production parameters between treated and untreated animals (controlled trials), or on reduced prevalence of clinical disease following supplementation. As a result of these techniques and the discovery that certain trace element deficiencies were closely linked to soil type, areas where trace-element deficiencies were common were defined. However, such methods of diagnosis are time consuming and expensive for widescale screening of farms and determining adequacy of supplementation. Methodologies, therefore, were developed for measuring the minute levels of trace elements in blood and tissue samples. By correlating the concentration of trace elements to the magnitude of response in a controlled trial, production-related reference ranges were derived. These were expressed as 'deficient', 'marginal' or 'adequate'. If the mean of a group of samples fell within the deficient range, then the probability of a production response to supplementation was high and the magnitude of the response would be significant in most cases. If the trace-element levels were marginal, the herd or flock may or may not respond to additional supplementation and if it did, the magnitude of the response would be small. If the levels were in the adequate range, then a response to supplementation was unlikely (Fraser 1982).

Cobalt

Using a combination of trial results, liver and pasture cobalt testing, and soil maps, a list of soil types known and suspected of being cobalt deficient was published and these were categorised as severe, moderate or marginal. In total, of the land considered suitable for farming, 1,061,000 hectares in the North Island and 918,000 hectares in the South Island were defined as cobalt deficient (Andrews 1961). Additional soil types were later classified as deficient for lambs (Clark et al 1985b). In 1946, Wallaceville workers derived a liver cobalt reference range for lambs (Andrews et al 1958) that was subsequently upgraded to a vitamin-B_{12} reference range (Andrews et al 1959). In 1966, a reference range for vitamin B_{12} in serum was published but serum was not considered as reliable as liver for diagnosing vitamin-B_{12} deficiency (Andrews and Stephenson 1966). The method used for measuring vitamin-B_{12} in serum and liver was a time-consuming microbiological assay. Laboratories later switched to the faster, more automated radioassay method following Millar's work, which demonstrated that some of these assays correlated well with the microbiological assay (Millar and Penrose 1980). The vitamin-B_{12} reference ranges for liver and serum were further refined by Clark et al (1989) by integrating the results of 64 published and unpublished New Zealand trials on lambs, and by expressing the data in the form of reference curves. These reference curves were used to extrapolate expected liveweight response to supplementation from particular levels of vitamin-B_{12} in liver or serum, as well as the probability that the economic value of the response would be higher than the cost of correcting the deficiency. They also defined that the relationship between the responses and liver or serum vitamin-B_{12} levels varied between seasons. In spring and summer, lambs were more responsive to vitamin-B_{12} supplementation at a particular B_{12} level within the responsive range than they were in autumn (Clark et al 1989). Factors such as prolonged yarding, haemolysis of blood samples, and liver damage from sporidesmin toxicity were also discovered to be factors that affected serum vitamin-B_{12} levels (Millar et al 1986; Clark et al 1988). New Zealand data on the effect of cobalt deficiency in sheep older than 1 year are sparse,

and summarised by Clark (1998). From these data, and observations from other countries, tentative reference ranges have been suggested for liver and serum vitamin B_{12} levels for older sheep (Clark 1998).

Despite widespread knowledge about areas where cobalt deficiency occurs, and good methods now available to prevent deficiencies, low levels of serum and liver vitamin-B_{12} are still reported by diagnostic laboratories (Clark 1998). Approaches to the monitoring of sheep in susceptible areas have been devised and published (Clark 1998).

Selenium

In the early New Zealand work that described the range and extent of selenium deficiencies, some preliminary liver and kidney selenium reference ranges were published for lambs with nutritional myopathy (Hartley and Grant 1961). These authors defined that farms with selenium responsive ill-thrift had pasture selenium levels <0.03 mg/kg DM and this cutoff value is still widely accepted today. The analyses were possible due to the development in New Zealand of fluorometric methods for determining micro amounts of selenium in plant and animal tissues (Watkinson 1960). It was discovered that the selenium concentration of soils, pastures grown on these soils, and blood samples from animals that grazed these pastures, correlated well. As a result of this information, maps were devised outlining the extent of selenium deficient areas (Andrews et al 1968). The relationship between the degree of selenium-responsive ill-thrift and infertility, and blood selenium levels in sheep was further defined (Sheppard et al 1984) and later reviewed (Ellison 1992).

Few trials on cattle correlating growth response to supplementation with blood selenium levels have been published. However, numerous trials have been conducted and these, together with published trials, were reviewed by Fraser and Wright (1986). Of the trials reviewed, 22/96 contained sufficient data to be included in a plot of growth response to supplementation vs blood selenium concentration. A negative relationship was found between blood selenium levels and magnitude of growth response, but the data varied considerably, particularly with time of year. The greatest growth responses occurred in trials performed in the autumn, an observation mentioned 25 years earlier (Hartley and Grant 1961). As a result of such work, selenium reference ranges for growth of young cattle were established.

Publications examining relationships between milk production responses and blood selenium levels in cattle were reviewed by Ellison (1992). Nineteen dairy herds, most with low blood selenium levels, were evaluated and the data indicated that economically significant milk production responses to selenium supplementation did occur but only if blood selenium levels were <150 nmol/l.

There have been a few trials published examining the effect of selenium supplementation on reproductive performance of dairy and beef cattle (again reviewed by Ellison 1992). In most trials, no effect was found. In one trial involving two herds, in which the average blood selenium levels of control cows were 115 and 130 nmol/l, there was a significant improvement in conception and submission rates in supplemented animals (Tasker et al 1987).

Studies in both sheep and cattle demonstrated that levels of activity of the selenium-containing enzyme, glutathione peroxidase, correlated well with concentrations of selenium in whole blood when selenium intake was constant (Sheppard and Millar 1981; Thompson et al 1981). The concentration of selenium in serum was a reliable indicator of recent selenium intake (Thompson et al 1981). Glutathione peroxidase activity in whole blood is now a common assay for estimating the selenium status of cattle and sheep, whereas serum selenium concentrations are widely used for cattle.

Copper

The reference ranges relating growth responses to serum and liver copper levels are mainly based on published data from other countries (reviewed by Ellison 1992), because of a dearth of published New Zealand data. There is even less published work examining relationships between milk production and fertility responses to copper supplementation. Cunningham (1950) documented that preventing peat scours improved cow condition, milk production and fertility. Recent work on clay soils near Morrinsville demonstrated a positive effect of copper supplementation on milk production in herds that were severely copper depleted (reviewed by Ellison 1992).

In sheep, there are also few published trials relating copper responses to biochemical levels, and in New Zealand there is one report in which response to copper supplementation was related to copper levels in liver and blood (reviewed by Ellison 1992). Most of the data available relate to levels required to prevent clinical copper deficiencies like enzootic ataxia and osteoporosis.

Development of supplementation procedures

At the same time as deficiencies were being discovered, trials were conducted to define appropriate and safe supplementation procedures. For cobalt, copper and selenium, both direct and indirect (via fertiliser) methods of animal supplementation were examined. Many of the systems devised then are still widely used today and have proven to be both practical and cost-effective. Other products and systems have since been developed in New Zealand and overseas.

Cobalt

Topdressing/spraying

Considerable trial work was undertaken to determine the effectiveness of topdressing with cobalt for preventing cobalt deficiency in stock. On pumice soils, annual topdressing at 5 oz of cobalt sulphate per acre (350 g/ha) prevented severe cobalt deficiency in all classes of stock (Andrews 1961) and this, applied with superphosphate fertiliser during the autumn, became the standard method of prevention. However, the length of time pasture cobalt levels remained elevated varied from 3–9 months, thus topdressing in autumn would not guarantee maximum growth rates in spring-born lambs the following summer (Andrews and Anderson 1955). More recent work indicated that cobalt sulphate must be applied at 350 g/ha for 7–10 years on pumice soils and possibly longer on some other soil types before pasture cobalt levels consistently remained in the adequate range (reviewed by Clark 1995). Once that occurred, a reduced annual rate of application of 175 g/ha would maintain adequate levels. Where pasture cobalt concentrations were below the adequate range, reduced application rates of cobalt sulphate increased pasture cobalt levels for only 8–12 weeks on pumice soils and 6 weeks on yellow-brown loam soils in Southland (reviewed by Clark 1995). Soil types with a high manganese content, such as Southland yellow-brown earth, are unique in that they bind soil cobalt and make it unavailable to plants, particularly in dry summers (Metherell 1989). Consequently, cobalt applied as fertiliser is less effective at preventing cobalt deficiency on these soils.

Strategies have been developed to provide the most practical and cost-effective way of preventing cobalt deficiency. Where cobalt deficiency is a problem, annual topdressing is the recommended procedure and failure to do so will further deplete soil cobalt levels, making deficiencies even more severe. Direct supplementation of lambs with vitamin B_{12} is more cost-effective in marginal areas where cobalt deficiency occurs infrequently and only in lambs (Clark 1995).

Intraruminal bullets
Intraruminal cobalt bullets were also developed soon after the initial diagnosis of cobalt deficiency. The early bullets were often regurgitated or were rendered ineffective due to coating with calcium phosphate (Andrews 1961), problems which have not been totally alleviated (Milllar et al 1984). Additionally, bullets were not as cost-effective as topdressing with cobalt, particularly where stocking rates were moderate to high (Andrews 1961).

Oral drenching
The first diagnostic test to confirm cobalt deficiency as a cause of ill-thrift was to drench lambs weekly with cobalt sulphate and compare their growth with that of unsupplemented controls (Andrews 1961). It was found that a weekly drench with cobalt at 7 mg per lamb maintained good growth rates, whereas monthly drenching with 300 mg resulted in greatly reduced death rates but suboptimal growth rates (Andrews et al 1966). Because weekly drenching is not practical and monthly drenching is not totally effective, vitamin-B_{12} injections are now used as the preferred means of treating individual animals.

Vitamin-B_{12} injections
Where individual animal supplementation is more desirable than cobalt fertiliser application, subcutaneous injections of water-soluble vitamin B_{12} are now used extensively in cattle and sheep as a means of increasing cobalt status. In lambs grazing pasture low in cobalt (0.03–0.04 mg/kg DM), monthly injections of 2 mg vitamin B_{12} were required to maintain liver levels in the adequate range (Clark 1995). The dose rate can be reduced to 1 mg and the interval between injections extended to 6–12 weeks where deficiencies were less severe (Clark 1995).

More recently, New Zealand scientists have developed a long-acting injectable vitamin-B_{12} formulation by incorporating the vitamin into biodegradable polymers of lactide/glycolide (Grace and Lewis 1999). For at least 250 days, this product maintains the vitamin-B_{12} status in the adequate range for lambs and ewes grazing pasture low in cobalt.

Selenium

Individual animal treatment
In a ground-breaking paper by Hartley and Grant (1961) there is a detailed report of two trials in the North Island examining the efficacy of different dose rates and frequencies of oral and subcutaneous administration of sodium selenite on two severely selenium deficient properties. Five mg of selenium given 3-monthly, from tailing, was as effective as monthly dosing at maintaining growth rates, and there was no significant difference between oral and subcutaneous routes on animal performance. Preliminary results in growing cattle suggested 20 mg given every 4 months was probably sufficient to maintain optimal growth rates. Subsequently, it was found that adding various selenium salts to an oily base and subcutaneously injecting a single 5 mg dose of selenium formulated in this way was as effective as monthly injections with aqueous sodium selenite at 5 mg/month for 7–10 months (Hartley 1966). Subsequent New Zealand studies in both sheep and cattle confirmed that 0.1 mg Se/kg liveweight given as sodium selenate or selenite to deficient animals was effective in increasing blood selenium levels to within the normal range (Meads et al 1980; Thompson et al 1981). Frequency of administration depended on the degree of deficiency but monthly supplementation resulted in a progressive rise in blood selenium concentrations. Anthelmintics and vaccines now frequently contain selenium allowing improvement of an animal's selenium status whilst protecting against parasitism or infectious diseases.

Various slow-release selenium devices have been evaluated under New Zealand conditions. These include intraruminal devices such as selenium/iron pellets, selenium glass boluses, osmotic selenium pumps, selenium in slow release anthelmintic capsules, and depot selenium injections of barium selenate, which are effective at providing an adequate level of selenium supplementation for 200–360 days in sheep and cattle (reviewed by Grace 1994). Their application is particularly useful in situations where stock are not regularly handled.

Selenium applied to pasture as a fertiliser
In 1961, it was demonstrated that a single topdressing of selenium salts on sand and pumice soils increased pasture selenium levels sufficiently to prevent selenium responsive unthriftiness for at least 7 months (Hartley 1961, 1966). Work continued through the 1970s (Andrews et al 1968; Grant 1969; Hupkens van der Elst and Watkinson 1972, 1977), until it was finally approved by the New Zealand Animal Remedies Board as a legitimate means of supplementing stock with selenium (Easingwood 1981), and New Zealand was the first country to approve such a procedure. This followed detailed studies measuring the effect of selenium applied to pasture on the environment, wildlife, domestic animals, water supplies, and meat and offal from grazing animals (Watkinson 1983). At the recommended application rate of 10 g Se/ha, the selenium content of pasture increases for approximately 7 months (Watkinson 1983). However, in most cases, stock grazing such pasture will have adequate blood selenium levels for 12 months. Annual application of selenium to pasture in prill form applied with phosphate fertilisers has now become widespread in New Zealand. Formulations that release selenium to plants more slowly and maintain adequate pasture selenium levels for at least 12 months have now been developed (Watkinson 1992). Today, protocols for supplementing animals grazing selenium deficient soil types are well established (Grace 1994).

Copper

Individual animal treatment
Cunningham (1950) demonstrated that a drench of 3.5 g of copper sulphate every week prevented peat scours, and ataxia in lambs was prevented by either a weekly dose of 1.5 g to ewes during the last 7 weeks of gestation, or 35 mg administered to lambs twice a week from birth. However, a practical approach was required for treating beef cattle and, as a result of a number of trials at Wallaceville, an injectable product containing 400 mg of copper glycinate was developed which would increase liver copper levels to within the normal range by 2–4 weeks after injection (Cunningham 1957). Copper glycinate injections are still used today, as are regular drenches containing soluble copper salts. The latter have particular application in dairy cattle, either as part of a mineral mix drenched to cows once or twice daily during milking, or dispensed into the drinking water supply using a metered device (Dewes et al 1990).

Gelatin capsules containing copper-oxide wire particles are also a common means of copper supplementation. The particles become trapped within the abomasal folds and copper is slowly released in the acidic environment. Much of this work was conducted in Australia (Langlands et al 1986) and other countries; further studies have found that this method of supplementation is highly effective in preventing both primary and induced copper deficiency in cattle, sheep and deer in New Zealand (reviewed by Wakelin 1992). The capsules are commonly administered at 0.1 g/kg liveweight and between 1–2% of the elemental copper in the needles becomes stored in the liver; maximum liver copper levels are attained about 6 weeks after administration.

Topdressing pastures
Cunningham (1950) demonstrated that annual applications of 5 lbs/acre (5.6 kg/ha) copper sulphate with fertiliser prevented clinical copper deficiency in both sheep and cattle. Fertilisers containing copper sulphate applied at 2–4 kg/ha increased herb-

age copper concentrations from 5 to 12 mg/kg DM within 4 weeks, soon after which it decreased to 8 mg/kg DM, followed by a more gradual decline over 9–10 months (Cunningham and Perrin 1946; Sherrell and Rawnsley 1982). The effectiveness of topdressing with copper sulphate at preventing copper deficiency in grazing livestock is dependent on pasture molybdenum levels. Ryegrass-clover pastures restrict the uptake of copper into the leaves at concentrations above 8–12 mg/kg DM. Therefore, in pastures high in molybdenum where copper requirements are higher than this, topdressing with copper may not meet the total needs of grazing animals (Sherrell and Rawnsley 1982, reviewed by O'Connor 1992).

Trace elements in deer

Since the commencement of deer farming in the early 1970s there has been much interest in the mineral requirements of deer. The most common trace-element deficiency in deer is copper. Enzootic ataxia, osteochondritis, and osteoporosis with limb fractures are all recognised clinical entities of copper deficiency in deer and, in severe depletion, growth may also be impaired. Reference ranges for serum and liver copper have been reviewed by Ellison (1995) and biochemical reference criteria for assessing the copper, selenium, vitamin B_{12} and iodine status of farmed red deer have been reviewed by Wilson and Grace (2001).

Occasional cases of white muscle disease in 1–6-month-old deer have been reported, and they have had low blood and/or liver selenium levels relative to reference ranges for cattle and sheep. As yet, no selenium responsive ill-thrift or infertility syndrome has been reported in deer (reviewed by Ellison 1995).

No growth responses to supplementation with vitamin B_{12} have been demonstrated in deer, even when grazing pasture containing 0.04–0.05 mg cobalt/kg DM, which is 50% of the cobalt requirement for lambs (Clark et al 1986). Other studies on trace elements in deer are briefly reviewed in this issue by Wilson (2002).

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