Section

5

5. Sustainable use of trace elements and the mineral nutrition of the grazing animal

Key Learning Objectives

After studying this section you should be able to:

1. Provide an in-depth understanding of trace elements in soils, plants and animals.
2. Understand the mineral nutrient requirement and the potential disorders in grazing ruminants.

Section 5 has been developed mostly (with permission of the authors of the book) from Chapter 7 and Chapter 9, of 'The Sheep: Health, Disease and Production.' Second Edition, 2002. Authors: D.M.West, A.N. Bruere and A.L.Ridler.

Introduction

In any discussion of trace elements, the requirements of both sheep and cattle for these elements must be considered because of the close association of the animals in most New Zealand farms. Veterinarians are frequently faced with determining the trace element status of farm animals and this is of particular importance because of the common occurrence of deficiencies of selenium (Se), copper (Cu), cobalt (Co) and occasionally iodine (I). New Zealand farmers are conscious of the impact of trace element deficiencies on production. Bush sickness or Co deficiency once made the raising of sheep and cattle almost impossible in certain areas, particularly the pumice country in central North Island. White muscle disease and Se responsive ill thrift, in lambs, were widespread in Canterbury and other areas of New Zealand until, in the 1950s, Se supplementation was shown to prevent these conditions. Copper deficiency, especially in calves on peat land has been recognised for many years.

Despite its small size, New Zealand has an extremely varied geology because it is situated on the interface of two of the world's tectonic plates. When two plates meet the heavier plate is forced under the lighter plate with the lighter material being forced up into mountain chains such as the Southern Alps. The volcanic ash from which some soils are derived is geologically very recent and the mineral content of these soils is dependent on both the type of material that has erupted from the volcanoes in the area and the subsequent effects of weathering. Soils from older ash showers are usually higher in trace element content because they have had more time for weathering to release minerals containing high concentrations of trace elements.

It is essential that veterinarians have a sound understanding of the diagnosis of trace element sufficiency/deficiency and are able to prescribe the most effective supplementation. To a farmer confounded by poor thrift in young animals or a poor lambing performance from ewes, the "magic of mineral supplements" offers an easy but frequently a wasteful and ineffective answer to problems. The tendency, over the years to "shotgun" treat animals with mineral mixes has often been wasteful and in some instances quite ineffective or even unnecessary. It must be emphasised that excellent diagnostic tests for trace elements in animals are now available, so that there is no longer any need for the indiscriminate use of mineral supplements.

Another significant trend in the development of good animal health is the move towards the diagnosis of sufficiency and ensuring that animals have adequate supplies and reserves, rather than concentrating solely on the diagnosis of deficiency. Except perhaps for Se and I, most animals, if fed an adequate diet will absorb the required levels of essential minerals in their foraging. This fact is frequently overlooked and it is not always appreciated that most supplementation supplies only minor quantities of the required mineral, relative to the quantities available in the diet.

From the extensive research and mass of clinical data and experience that is available, the diagnosis of trace element sufficiency/deficiency is no longer difficult. Most tests can be conducted on animal tissue and fodder at low cost. The tests are accurate and offer a more professional approach to determining the mineral status of a property and its animals than has previously been possible. In the development of any animal health programme it is essential that the veterinarian soon presents the farmer with a clear picture of the farm's trace element levels and requirements. From this information a logical programme of mineral supplementation can be developed and applied on a regular basis.

If such a procedure is followed the occasional monitoring of mineral element levels in the tissues of target animals will allow adjustment to the supplementation to be made as required. In most cases the monitoring of the mineral element levels, once the programme is up and going, will give the farmer the assurance that animals are acquiring all the essential minerals needed to maintain full health and production.

It is with this approach in mind that the following section is presented - sufficiency for good health - rather than deficiency and supplementation when the health of the animals is seriously affected and their production reduced.

In this section the common mineral nutrient disorders affecting sheep and cattle grazing pastures in New Zealand are presented. The disorders are due to deficiency [Cu, Se, Co, I, manganese (Mn), magensium (Mg), and calcium (Ca)] or toxicity [fluorine (F)] of a mineral nutrient or one nutrient influencing the deficiency of another nutrient (e.g. High levels of molybdenum (Mo) and sulphur (S) in diet causing Cu deficiency, high levels of potassium (K) in diet causing Ca deficiency). The mineral nutrients are discussed here in terms of their requirements for the animals, the diagnosis and the factors causing the nutrient disorders, and

methods of prevention and treatment of the disorders. The study material in Section 5 is arranged in the order of elements, in 9 different modules:

5.1   Selenium (Se)

5.2   Cobalt (Co)

5.3   Copper (Cu)

5.4   Iodine (I)

5.5   Molybdenum (Mo)

5.6   Manganese (Mn)

5.7   Calcium (Ca)

5.8   Magnesium (Mg)

5.9   Fluorine (F)

5.1 Selenium (Se)

Introduction

Plants do not need Se, but it must be present in adequate amounts in forage since it is essential for animals. However, at very high concentrations in forage it can be toxic to animals. Unlike the situation with most other elements Se deficiency or toxicity can be indicated by soil or pasture Se concentrations.

Se in soils

Se concentration in New Zealand soils range from 0.03 to 2 mg Se/kg of soil. Selenium exists in soil in several chemical forms, which differ widely in their solubility and availability to plants. Table 5.1 shows the different forms of Se in soil.

Table 5.1   Forms, chemical symbols and plant availability of Se in soil.

Form	Chemical Symbol	Plant Availability	Remarks
Selenides	Se^{2-}	No	Insoluble
Elemental Se	Se^0	No	In neutral and basic soils Se^0 is oxidized to selenites and selenates by microorganisms.
Selenites	SeO_3^{2-}	Yes	A large fraction of Se in acidic soils may occur as stable complexes of selenites with hydrous iron and aluminium oxides. Dominant form in acid soils.
Selenates	SeO_4^{2-}	Yes	Dominant form in calcareous soils.
Organic Se	-	No	Soluble organic Se are liberated through the decay of soil organic matter.

Transformations between Se pools are slow. The chemical forms present in soil depend largely on soil pH and soil redox potential. In aerated soils, selenate and selenite forms mainly occur, whereas in acidic waterlogged soils the selenides and elemental Se forms dominate. Selenites are adsorbed rapidly and strongly by soil minerals and appreciable more so than selenates and are therefore less available to plants and less subject to leaching. Most of the Se ingested by livestock is excreted in faeces, largely as organic Se, elemental Se and selenides, which are not immediately available for plant growth. In a Se metabolism study conducted on ewes, Krishnamurti *et al.* (1997) showed that 82% of Se ingested by ewes was excreted by the animals when the animals were fed with hay having normal levels of Se, whereas the excretion was 54% for ewes fed with hay having inadequate levels of Se.

Limited quantities of selenate occur in acidic and neutral soils. Selenates are highly soluble and readily available to plants and are largely responsible for toxic accumulations in plants grown on higher-pH soils (pH >7.5).

There are between three to five million hectares of New Zealand soils that are considered to be Se deficient and these are shown in Figure 5.1. The soils most affected by Se deficiency in New Zealand are sandy soils, including sandy Pumice Soils, and the Semiarid Soils and Pallic Soils formed on loess in the South Island. However it is wise to be cautious over too rigorous extrapolation from Figure 5.1 to individual farms. In many instances quite noticeable Se deficiencies have been recorded in areas considered marginal or even to have normal soil levels of Se. This may indicate that there can be small regional differences or seasonal variations in the occurrence of Se deficiency.
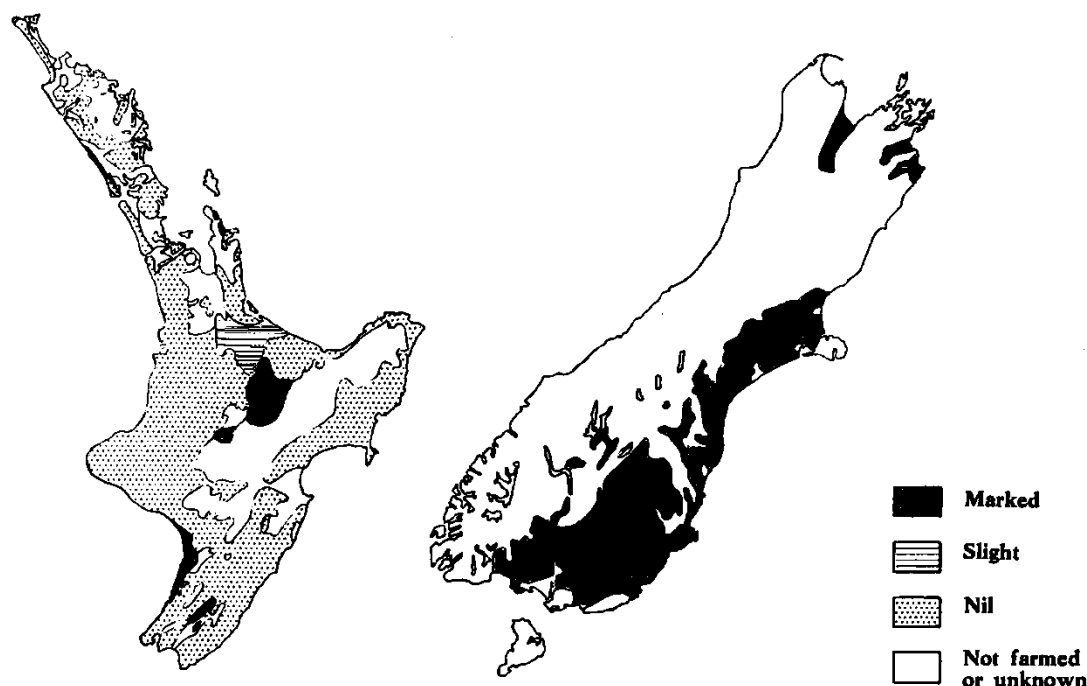

Figure 5.1. *Selenium responsive areas of New Zealand as determined by lamb growth trials (adapted from Andrews et al., 1968).*

Soil test for Se

Total Se concentrations in New Zealand surface soils have been rated by Wells (1967) as values below 0.5 mg/kg being low and likely to be associated with Se-responsive disorders in livestock, and values above 1.5 mg/kg being very high. However, the uptake of Se by plants should be determined by the soluble Se concentration and not by the total Se content in the soil.

Se in plants

Plants do not need Se, but it must be present in adequate amounts in forage since it is essential for animals. Se has no known role in the nutrition of plants, but high Se concentration in forage can be toxic to plants. Plants absorb Se mainly in the form of selenate (SeO_4^{2-}) and to a lesser extent in the form of selenite (SeO_3^{2-}).

Plant species differ in Se uptake. Certain species of Astragalus absorb many times more Se than other plants growing in the same soil, because they utilize Se in an amino acid peculiar to the species. Plants such as the cruciferous (e.g., cabbage, mustard) and onions, which require large amounts of S, absorb intermediate amounts of Se, while grasses and grain crops absorb low to moderate amounts. Among the pasture species, white clover contains less Se than ryegrass, which contains less than browntop. Thus improving a low-producing browntop pasture to a

high-producing rye grass-white clover pasture can lead to a decline in animal Se status and perhaps lowered animal production particularly in areas of marginal Se status.

Low total Se in the soil parent material or low availability of Se in acidic and poorly drained soils usually causes insufficient plant uptake of Se. Plant uptake of Se is generally greater in high-pH soil (pH >7.5) than in acidic soils. This is because high soil pH facilitates the oxidation of selenites to the more readily available selenates.

Climatic conditions can also influence the Se status of pastures, either by affecting the legume/grass balance or by affecting the Se status of soils or pastures. For example in Australia Se concentrations in pastures were negatively related to annual rainfall. In New Zealand recent volcanic activity was reported to have raised Se concentration in soils and pastures (Cronin *et al.*, 1997).

Variations in Se status of pastures can also be affected by applying fertilisers to correct other recognised nutrient deficiencies (Gardiner, 1969; Spencer, 1982). This has been attributed to stimulated growth decreasing Se concentrations in pasture plants.

Se in animals

Selenium was first identified as an element in 1817 and was named from the Greek word - selene, meaning the moon. Early interest in Se was on its toxic properties when in 1930 it was shown to be the agent that caused in "alkali disease" or in "blind staggers" in cattle and horses in North America. It was not until 1957 that Se was shown to be an essential trace element for animals when it was discovered that Se could prevent liver necrosis in rats. Soon after, work in United States of America and New Zealand showed that white muscle disease in sheep and cattle could be prevented by Se therapy. Growth rate responses in lambs to Se were identified throughout much of the eastern South Island and on the coarse pumice soils and some coastal sands of the North Island.

Curiously many of the diseases Se was effective in preventing could also be prevented by Vitamin E or slowed by S containing amino acids, but by the late 1960s the essential nature of Se even in the presence of Vitamin E had been firmly established. However the discovery of a biochemical role for Se and an explanation for its intriguing relationship with Vitamin E was not made until 1973. Until the discovery of the prophylactic effect of Se in the animal myopathies (white muscle disease), which became apparent in New Zealand during the 1950s, dosing lambs and calves with vitamin E was used to prevent such diseases developing.

It is now known that the most important function of both Se and vitamin E is the protection of biological membranes. Peroxides and oxygen radicals are serious cellular toxins, which can destroy connective tissue, damage biological membranes, oxidise sulphydryl groups, inactivate enzymes and cause peroxidative damage of nucleic acids. Lipid peroxides are produced particularly during the breakdown of polyunsaturated fatty acids. The functional combination of Se and vitamin E helps to prevent the damage. Vitamin E is localised in the cell membranes as a biological anti-oxidant and inhibits the formation of lipid peroxides. Selenium is an essential part of glutathione peroxidase (GSH-px) which catalyses the reduction of peroxides to less harmful hydroxyacids in the cytoplasm. If this protective function of Se and vitamin E fails, the increased quantities of lipid peroxides may trigger a chain reaction which causes further peroxides and free radicals to be formed, and may lead eventually to the damage of biological membranes and cell death.

Selenium and vitamin E are also important for the maintenance of resistance to infectious disease. Reactive oxygen metabolites produced by granulocytes and macrophages are eliminated by Se and vitamin E. Various functions of the immune system are inhibited by Se and vitamin E deficiency, including the migration of leucocytes and phagocytosis. This is particularly important in dairy cows when it has been found that mammary polymorphonuclear leucocytes from Se-deficient cows can destroy microorganisms less efficiently than those from Se-adequate cows.

While the most important function of Se is in the protection of biological membranes, a range of other functions have been suggested such as a role in the arachadonic cascade, production of prostaglandin $F_{2\alpha}$, cell-mediated and humoral immunity and conversion of thyroid hormone thyroxine (T_4) to the active tri-iodothyronine (T_3) form.

Deficiencies of Se

The Se concentration in blood is very responsive to and dependent on the level of Se in the diet. New Zealand has a low Se status as compared with other countries (Table 5.2). When visitors from UK or USA come to NZ their blood levels of Se fall to those of NZ's and vice versa. However, there has been a significant increase in the Se status of New Zealand humans since 1988 and it is not known whether this is due to increased supplementation of livestock or importation of food such as grain from overseas.

Table 5.2: **The Se concentration in whole blood of normal people in New Zealand and other countries (Robinson 1975).**

Country	No. Of Subjects	Mean Blood Se (ug/l)
New Zealand	170	68
New Zealand	24	69
UK	8	320
USA	280	210
Sweden	6	120
Guatemala	4	230

Earlier live weight gain trials conducted on lambs in New Zealand showed Se responsiveness to be area dependent, and the responsive areas corresponded closely to regions of low soil Se content.

A number of factors influence pasture and animal Se levels, but ultimately the Se level in plants is highly related to soil levels. Some plants are Se selectors and concentrate the element many times in their foliage. Such plants do not occur naturally in New Zealand but are present in parts of Queensland, Australia.

Other factors influencing Se uptake by stock are rainfall and pasture composition. In general legumes tend to be lower in Se compared to grasses, particularly native species such as browntop. The pH of the soil is also important in the availability of Se to plants. Alkalinity encourages the absorption of Se as seen in "Alkali disease" in horses. Fertiliser application, particularly the S in superphosphate may compete for absorption sites with Se in both plants and animals.

There is also seasonal variation in the Se content of pasture, the content being generally lowest in spring when rainfall is heaviest. Seasonal variations in cattle blood have been as much as from a low mean concentration of 10 μg/l Se to 20 μg/l Se, with similar variations recorded in sheep as well.

Other factors are associated with variations in the blood levels of Se. Marked differences are seen between animal species; whether these reflect an intrinsic difference or differences in diet is unclear (Table 5.3).

Table 5.3:   Blood Se levels between species (Fraser, 1980) in New Zealand.

Animal	Number Examined	Median (95% range) ug/l
Cattle	772	32 (6-110)
Sheep	168	58 (20-200)
Horses	135	84 (30-200)
Pigs	10	160 approx.
Dogs	171	208 (65-410)

There may be a relationship between low Cu levels in sheep and a Se deficient diet. The dosing of Se deficient sheep with Se not only raises the Se level of the animals but may also increase Cu storage and retention.

There does not appear to be an age influence on Se levels in blood of various species and any variations between sex and breeds are minor compared with the dominating influence of dietary Se levels.

A deficiency of Se can occur when the total Se level in soil is below 0.5 ppm, and unthriftiness is likely when Se levels in pasture are less than 0.02 ppm. It is not seen in pastures above 0.03 ppm of Se.

Clinical Signs

Unthriftiness in young animals

One of the most economically important effects of Se deficiency occurs in sheep and cattle up to approximately 15 months of age and is manifest as slow growth and poor production (e.g. wool). Calves may show faded coats with delayed shedding of the winter coat. These signs are non-specific and must be differentiated from under nutrition, parasitism and other deficiencies.

Weight responses of lambs to dosing with Se salts have occurred widely in New Zealand. In most instances these responses, some quite remarkable, have been related to areas that could be described as severely Se deficient as judged by soil analysis. Extensive trials conducted in 1974-1975 emphasised the importance of the marginally deficient situation. In some of these trials weight gain responses were achieved when the mean soil Se levels were 0.56 ppm (below 0.5 ppm is likely to produce unthriftiness).

The challenge facing the veterinarian is the diagnosis of marginal deficiencies where Se-responsive ill-thrift may go unrecognised by the farmer. There are many areas of New Zealand that come into this category. No accurate estimate of the economic losses that accrue from uncorrected Se-responsive unthriftiness can be made, but even on farms where expected weight responses may be small the low cost of supplementation would be more than offset by extra returns as a result of improved animal health.

Myopathy - white muscle disease

The first reports of white muscle disease in lambs in New Zealand were in 1953. The following year severe losses from the disease occurred in newborn lambs and lambs a few weeks of age, the main losses reported were from Canterbury and the pumice land of the North Island. Losses of 500/900 and 850/1650 lambs born were recorded on two properties in North Canterbury. These examples were not unique. Because of the differing age occurrence of the deficiency in lambs and calves the names congenital and delayed white muscle diseases were given.

(a)   Congenital white muscle disease

This has been reported in lambs, calves and kids. Lambs are often born dead or die suddenly soon after birth. In some cases lambs die of starvation as they are unable to suckle and lesions of the lingual muscles are not uncommon.

At necropsy affected lambs usually show distinct white necrotic lesions on the myocardium that may be streaked with calcified deposits. Animals that have survived for a few days may also have symmetrical myonecrosis especially of the hind limbs. The muscles taking on the appearance of "cooked chicken flesh".

(b)   Delayed white muscle disease

Delayed white muscle disease may occur in lambs from a few days to even several months but is usually seen at about 2-6 weeks of age. It may be precipitated by some procedure such as moving lambs and ewes or yarding for docking. Lambs may walk with a stiff gait and hunched back. They may be unable to suckle. Frequently they are unable to walk at all and will just become recumbent and rather unresponsive. In some cases they will be found dead having died from respiratory failure.

At necropsy the familiar lesions of myonecrosis may be seen in skeletal muscles. Frequently such lesions are glistening with streaks of calcified tissue.

White muscle disease is a cause of death in goat kids between birth and three months of age. Losses are usually below 10% but in some flocks over 50% of kids have died. Affected kids are often found dead but others show signs of dyspnoea, depression, stiffness and nervous signs for 1-2 days prior to death. It has also been reported in calves and deer fawns.

The features seen at necropsy are largely the result of right-sided heart failure. There is variable amount of clear fluid containing fibrin in the thoracic and peritoneal cavities, enlarged liver and typical lesions in the heart and possibly skeletal muscle.

Because severely Se deficient areas of New Zealand are now recognised and appropriate supplementation is undertaken, it is rare to see clinical white muscle disease. The subclinical effects of Se deficiency are more common.

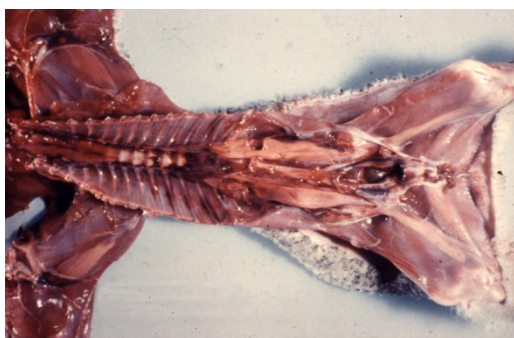*Figure 5.2.   Lambs suffering from delayed white muscle disease. North Canterbury, 1955.*

Figure 5.3.
Delayed white muscle disease. Note the pale colour of the skeletal muscles.
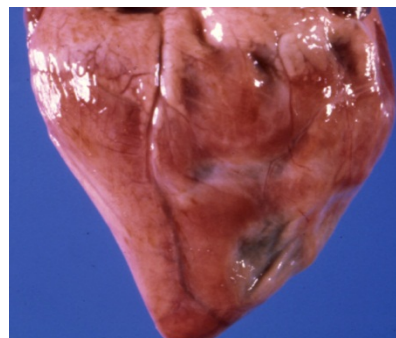

Figure 5.4.
Congenital white muscle disease causing scarring of the heart muscles.

Impaired reproduction

While an early study by Hartley indicated that infertile or dry/dry ewes were a consequence of Se deficiency it has not always been possible to reproduce this effect experimentally, nor have veterinarians found a consistent link between the two to anywhere near the extent of the earlier observations. The earlier work described infertility in ewes with lambing percentages as low as 25% because of embryonic death within 3-4 weeks of conception. Affected ewes may conceive a second time and appear as late lambers, but in most cases became dry/dry ewes for that season.

An experiment at Wallaceville Research Station in 1983 showed no appreciable difference in conception rate between a control group of ewes and a group dosed with Se before mating: All foetuses in both groups were alive up to 23 days after mating. However, in the Se deficient ewes, an increasing proportion of foetuses were dead after 23 days and by 30 days none were alive. This contrasted markedly with Se-dosed flockmates in which all foetuses remained alive over the same period. The trial suggested that the critical level for foetal survival was approximately 10 ug/ml of Se in the blood. This work supports Hartley's earlier observations.

Occasional reports have also associated low Se levels with reduced fertility in cows. In Taranaki, Tasker, *et al.* (1987) indicated reduced submission rates and conception rates in unsupplemented dairy cattle when mean glutathione peroxidase levels were below 1KIU/l.

Selenium deficiency has also been implicated in retained foetal membranes of dairy cows overseas but this condition has multiple causes.

Inflammation and immunity

Selenium deficiency has been shown to inhibit (a) resistance to microbial infections, (b) neutrophil functions, (c) antibody production, (d) proliferation of lymphocytes in response to mitogens, and (e) cyto-destruction by lymphocytes. There is evidence that the effects of Se on disease resistance may operate at levels higher than those found to be necessary to prevent traditional disease syndromes. Hence there is a need to be aware of these other roles for the element, and there is also a need for further studies to be conducted to investigate the role of Se in disease resistance under New Zealand conditions. The need for trials to be conducted under New Zealand conditions is paramount because Se plays a part in only one of a number of antioxidant systems that protect against various types and levels of oxidant challenge.

The anti-oxidant properties of the Se-containing enzymes glutathione peroxidase, and the closely related enzyme phospholipid glutathione peroxidase, explain the role of the element in many of the diseases of domestic livestock, where it acts along with membrane-bound vitamin E (α-tocopherol) and other anti-oxidants to protect cells from oxidant injury. At a cellular level, Se is involved in three major types of activity. The role of Se in inflammation and the immune response is thought to be related to the protection of sensitive cellular membranes and enzymes from oxidant attack and also to Se's involvement in eicosanoid metabolism. The latter has recently been implicated in transmembrane signalling in T lymphocytes.

There have been few field reports linking low Se to an increased susceptibility to disease. A small number of research trials assessing the relationship between Se status of dairy cows and somatic cell counts in milk have given mixed results.

Reduced milk production

A number of research trials have investigated milk volume production in response to Se supplementation. These indicate that economically significant milk production increases to Se supplementation are unlikely at mean blood Se levels greater than 250 nmol/l or GPx levels greater than 2 KIU/l. At Se levels below this, responses are variable but there is a tendency for the magnitude of the response to increase with decreasing blood levels (Ellison, 1992; Wichtel *et al.*, 1998).

Other species

Selenium deficiency and vitamin E deficiency have been associated with hepatosis diatetica and mulberry heart disease in pigs; myopathy and exudative diathesis in poultry; myopathy and steatitis in horses, cats, dogs and ferrets.

Diagnosis of Se Deficiency

A diagnosis of Se status can be made by one or a combination of three methods:

1.   On farm assessment - previous experience

The history of the farm including previous experience with clinical manifestations of Se deficiency, Se supplementation, knowledge of other farms in the same district and results of previous chemical analysis should be considered.

Other relevant factors would include the clinical signs, age, species, pasture species, stage of growth and management. The usefulness of this approach is limited by how much is known about the area and the vague nature of the clinical manifestations of marginal Se deficiency.

2.   Chemical analysis

As previously discussed, Se levels in soil, plants and animals in the same location show a very close relationship. Thus if the soil is known to be deficient in Se the likelihood of livestock problems occurring is very high. However, tissue samples from animals, often young animals, are preferred as they give the best indication of the Se absorbed. As there is usually little variation between animals in Se levels in samples taken from the same farm the number of samples required is small and usually 3-5 samples will suffice. Soil and plant levels may be useful in forecasting the possibility of future Se deficiency; for example on an unstocked farm or land being converted to livestock from cropping or horticulture.

Selenium is present in all tissues, but the liver and kidney normally have the highest Se concentrations. Liver is the organ normally used for Se assay and liver biopsies may be taken serially from an individual animal.

Selenium levels can be determined from either serum or whole blood samples. Alternatively blood glutathione peroxidase (GSH-px) levels may be used. If the latter are used it must be remembered that there is a delay in the response of erythrocyte GSH-px activity to changes in Se status. Erythrocyte GSH-px activity depends on the Se availability during erythropoiesis and as erythrocytes remains in circulation for some months, sudden changes in Se status will not immediately be reflected in erythrocyte GSH-px activity.

The type of sample selected will differ depending upon the objective of testing, as indicated by Table 5.4 (adapted from Clark and Ellison, 1993);

Table 5.4.   Sampling for Se depending upon the reason for sampling.

Reason for sampling	Time to sample	Species and age	Sample type	Sample #	Interpretation
Poor performance	At the time of the problem	Affected animals	EDTA blood	**3**	Means
Farm deficiencies	Seasonal variation minor. Could be any time, esp. late spring/early summer	Unsupplemented adults or young stock	EDTA blood	3	Means
Adequate reserves	Late winter/early spring	Unsupplemented adults or young stock If supplemented:	EDTA blood Serum/liver	**3** Pref. 10	Means Individual values
Supplementation	Half-way point between planned supplementations	Animals being supplemented	Serum or liver best	Pref. 10	Individual values

Reference ranges

Reference ranges may vary with different laboratories; the one to consult is that from the laboratory performing the analysis in question.

Table 5.5.   Reference ranges for Se in sheep and cattle.

	Low	Marginal	Adequate
Glutathione peroxidase - whole blood (KIU/L); Sheep	<1	1-3	>3
Glutathione peroxidase - whole blood (KIU/L); Cattle	<0.5	0.5 - 2	>2
Blood Se - nmol/l	<130	130 - 250	>250
Liver Se - nmol/kg (sheep)	<250	250 - 450	>450
Liver Se - nmol/kg (cattle)	<600	600-850	>850
Serum Se (cattle) - nmol/l	<85	85-140	>140

The interpretation of analytical results requires careful consideration and experience by the advising veterinarian. In some cases a decision may be made to supplement animals even though the likelihood of a production response is uncertain. Always remember that sufficiency throughout the year is required for good animal health and therefore for preventative animal health the focus should be on ensuring sufficiency rather than diagnosing deficiency.

Clear growth rate responses to Se supplementation have been demonstrated, as demonstrated by Figure 5.5. Each dot on the graph indicates a separate supplementation trial.
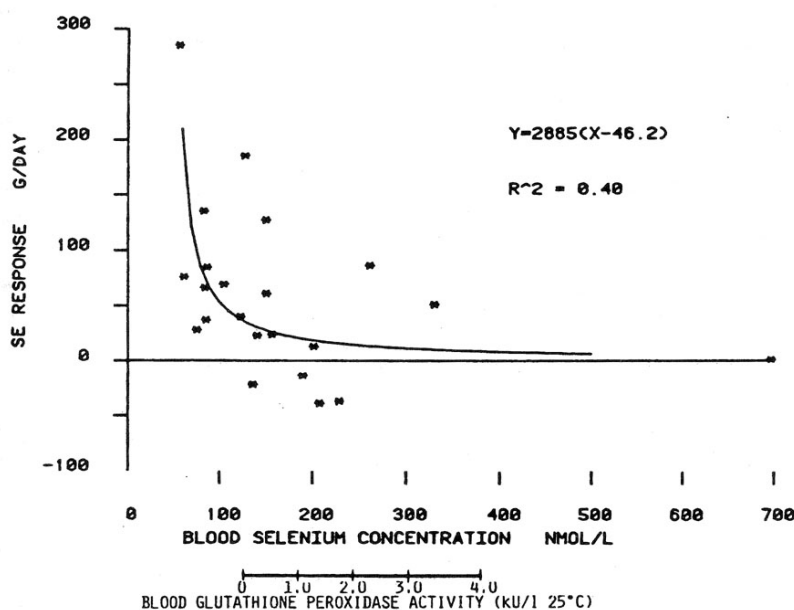

Figure 5.5. *Live weight responses to Se supplementation compared with blood Se and glutathione peroxidase levels (from Ellison, 1992).*

3. Controlled trials

Due to increased sophistication of analytical methods it is no longer necessary or desirable to conduct controlled treatment trials to diagnose Se deficiency. The clinical manifestations of Se deficiency are varied and marginal deficiencies are difficult to confirm using controlled trials because of seasonal variations in Se uptake, the vague nature of some of the signs of deficiency, farmer reluctance to leave animals unsupplemented and the cost and time involved in conducting trials. However where the reference ranges are uncertain there is a need for carefully conducted response trials.

Prevention of Se Deficiency

Following a decision to supplement, advice is given on which animals should be supplemented, by what method, how much and how often. These decisions are made on the basis of the clinical findings, past local experience, appropriate research findings and economics. It is often wise to monitor the effect of a programme and modify it accordingly, because the degree of deficiency varies markedly from farm to farm and supplementation programmes vary also.

Young stocks in particular are usually supplemented and in addition ewes before tupping and lambing and cows before calving may be treated. In many instances all animals may require treatment. The lactating dairy cow is a special case, not considered in this text, but such animals may be at high risk of Se deficiency in many parts of New Zealand.

It should be noted that with the increased awareness of New Zealand's marginal Se status and the availability of a range of supplements, animals are in danger of being supplemented unnecessarily without tissue Se analysis to justify the decision. Selenium is extremely toxic in

overdose and supplements should be used with caution. For more information on Se toxicosis, refer to Parton *et al.* (2001).

Methods of supplementation

Drench

As a cheap and safe method of supplying Se to sheep, drenching is effective provided it is done at the appropriate times of the year and periodic monitoring of animals is undertaken. A single dose of Se usually provides adequate supplementation for 1-3 months. Frequently Se as sodium selenate or sodium selenite is mixed with anthelmintic drenches. Lambs and hoggets, in most cases, receive quite adequate Se supplementation by this method because of routine monthly drenching programmes to control internal parasites. In the case of older sheep, if Se supplementation is required, both ewes and rams should be dosed at least 1-2 months before joining and the pregnant ewes may need to be dosed once or twice during pregnancy. Further dosing may be necessary after lambing depending on the degree of deficiency.

Because of better bioavailability of Se, sodium selenate is the usual oral form of Se used and is available in various concentrations (25 mg/ml, and 5 mg/ml) for dilution with either anthelmintic drench or water before dosing. Great care should always be taken when using Se concentrates as many instances of Se poisoning in sheep, particularly young lambs, have been reported in New Zealand.

The oral dose of Se as sodium selenate is:

Ewes	5 mg
Lambs birth to 1 month	1-2 mg
Lambs 1 month and over	2-3 mg

Subcutaneous injection

Sodium selenate is available for animal use in injectable form and in the case of sheep they are often given in association with clostridial vaccines. However the "safety factor" with injectable forms of Se is considerably less than Se given orally so that extreme caution should be applied in their use. There are numerous reports of Se toxicity following the use of injectable forms, particularly when selenised clostridial vaccines are given to lambs at docking.

A long-acting product containing barium selenate (Deposel, Novartis New Zealand Ltd.) is also available. Because this product is slowly released the risk of toxicity is reduced. This gives a supply of Se for 6-18 months depending on the dose rate and class of livestock in which it is used. Tissue reactions may occur so that care in choosing the site of injection is essential. Trials have shown that lambs treated with a single dose of Deposel maintained a rising Se status after 5 months, whereas lambs treated with potassium selenate (single injection) had returned to control levels after 2-3 months.

Intra-ruminal pellets (Permasel, Schering-Plough Animal Health Ltd.)

Intra-ruminal pellets containing 5% W/V of elemental Se are available for long-term release of Se (in the vicinity of 9-12 months). They may have a use for high country sheep when mustering is infrequent and dosing is difficult. Animals must be yarded 3-4 hours prior to dosing to ensure pellet retention and kept yarded after dosing for a further 2 hours to watch for regurgitation. Their main disadvantage, like other bullets that release in the reticulo-rumen, is that they may become coated with insoluble calcium phosphate.

A number of anthelmintic capsules also contain trace element additives that include Se.

Se pour-on

Se can be absorbed dermally and a pour-on formulation is available for use in cattle and deer (Selpor, Ancare New Zealand Ltd).

Topdressing with Se prills

In New Zealand, prior to 1980 pastures were topdressed with sodium selenites to prevent Se deficiency in grazing stock. From early 1980s sodium selanate at rates up to 10g/ha of Se has been recommended instead of sodium selenite because only one-fifth the rate of sodium selenite is required to raise pasture to the same Se concentration. Use of sodium selanate is safer since foliar contamination effects are about 5 times less than sodium selenite and is 4 to 20 times less toxic to animals and less of an irritant (Watkinson, 1983).

In New Zealand there are two types of Se fertilisers, namely Agsel prills (1% Se) and Selcote Ultra prills (1% Se), currently been used to topdress pastures (Morton and Roberts, 1999). Selenium in Agsel is present in the form of sodium selenate and that in Selcote Ultra is present as 50% sodium selenate and 50% barium selenate. Selenium in Agsel is 100% water-soluble whereas Se is Selcote Ultra is 50% water-soluble. Whelan and Barrow (1994) in Australia showed that a single application of sodium selenate fertiliser at 10 g/ha of Se maintained an adequate Se status of sheep for about 15 months, whereas a single application of barium selenate at the same rate was effective for 4 years. The difference between the two fertilisers was attributed to the rate of release of Se. The sodium selenate was a quick release of Se form while the barium selenate was a slow release of Se form. Recent glasshouse (Loganathan and Hedley, 2005) and field (McLaren, 2005) studies in New Zealand also showed that Agsel prills containing Se only in the form of sodium selenate was effective in supplying Se to pasture only for 5 to 7 months, whereas Selcote Ultra which contain both sodium selenate and barium selenate was effective for at least 12 months.

The shorter duration of effectiveness of sodium selenate was also shown in experiments carried out by Watkinson (1983). In one of Watkinson's (1983) experiments, pasture Se increased by about 2 μg/g of Se within a week after sodium selenate top-dressing and then decreased exponentially with the relatively short half-life of 3 to 4 weeks. The initial levels were virtually reached after 6 to 9 months (Fig. 5.6). However the body store of Se was sufficient to maintain adequacy for at least the full 12 months after top-dressing. The Se content in blood of animals grazing pasture increased slowly to a maximum after about 3 months and decreased slowly to almost the initial level 9 months later. Figure 5.6 shows the response curves where lambs contained less, but nevertheless adequate Se, than ewes just before the next topdressing is applied.
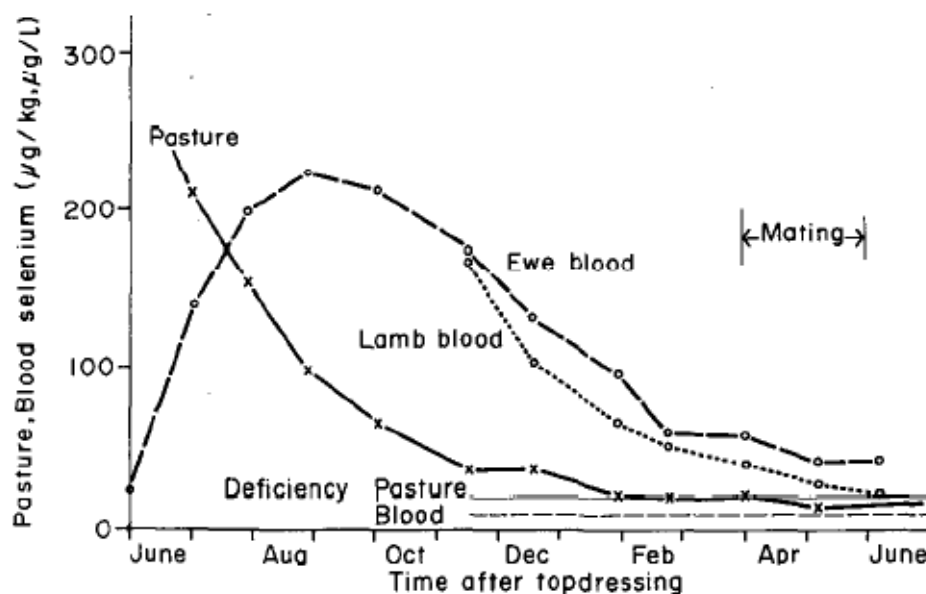
Figure 5.6. Se in pasture, and blood of ewes and lambs after May selenate topdressing at 8.5 g/ha Se (From Watkinson, 1983)

Selenium topdressing, even when used in strips, rather than as a blanket treatment will raise blood levels of Se in sheep and cattle several times. Further, these levels are maintained for several months and in most cases remain elevated for up to a year. Following topdressing, blood Se raises rapidly to a maximum after 2 to 3 months (as measured by GSH-px in blood) and then decreases at a slightly slower rate usually reaching pre-topdressing levels in 12 months.

When sodium selenate is used as topdressing 15% of Se is absorbed into foliage within 24 hours to increase the concentration to perhaps 100 times. Thereafter it decreases rapidly and exponentially with a half-life of only 3-4 weeks, so that after 2-3 months the pasture concentration is only 10% of the peak. The soil adsorbs some Se very rapidly and most of the remainder is reduced to selenite within a week. Slow release Se prills incorporate barium selenate and provide a longer period of supplementation.

Even under conditions of very high rainfall, such as encountered in South Westland, the use of Se topdressing to supplement animal's Se has proven highly successful. An annual rainfall in that area of 4,000-5,000 mm per annum is regularly recorded and sheep grazing topdressed areas maintained more than adequate Se levels for twelve months.

The time of application for Se topdressing is of vital importance when making fertiliser recommendations. Topdressing in autumn provides less Se than in spring to sheep at the critical periods for ewe fertility and lamb growth (Watkinson, 1983).

Liver Biopsy

Trace element monitoring, particularly of Cu and to a lesser extent vitamin B_{12} levels, is best done by analysis of liver. Liver can either be collected from animals at slaughter or can be obtained from the live animal by liver biopsy. Liver biopsy is a relatively straightforward technique, which, if done correctly, results in very few if any complications. The general procedure can be used for sheep, cattle and deer.

Preparation

Animals should be biopsied straight off pasture, as the full rumen keeps the liver in a consistent position. Adequate restraint is essential. For cattle, restraint is best achieved in a cattle crush and head bail that allows access to the right hand side of the animal. Sheep should be held in left lateral recumbency on a table or similar. Deer should be sedated and restrained in left lateral recumbency.

Procedure

On the right hand side of the animal an area should be clipped and surgically prepared, centred over the eleventh intercostals space (second to last intercostals space) at approximately one-quarter of the distance between the thoracic vertebrae and the sternum. Inject local anaesthetic (2% lignocaine) into the biopsy site, both subcutaneous and into the muscle. Once the local anaesthetic has become effective, make a small stab incision of approximately 1cm in length into the biopsy site using a scalpel blade.

A 4mm x 230mm stainless steel trochar and cannula is used to make the liver biopsy. This should be introduced into the stab incision perpendicular to the body wall. It must be advanced through the body wall, pleural cavity and diaphragm before entering the liver. Therefore there should be two distinct feelings of 'popping' through tissue – once through the body wall and once through the diaphragm. Once through the diaphragm, the trochar should be removed and the cannula twisted as it is advanced through the liver. Advancing the cannula through the liver gives a gritty or grating feel (similar to coring an apple) which is particularly apparent with cattle and less so for sheep and deer. If necessary the biopsy cannula may be withdrawn slightly and redirected one or two more times to ensure adequate sampling. The cannula is advanced through the liver for a distance of 2-3 cm and then a 5 ml syringe is attached to the end of it and the plunger withdrawn 2-3 ml to provide sufficient negative pressure to retain a core of liver in the cannula. Maintaining this negative pressure, the cannula should be quickly withdrawn.

A core of liver of approximately 1 cm in length is required for each analyte. This should be put onto blotting paper to remove excess blood and then transferred to a plain vacutainer tube or clean pottle for transport to the laboratory. The skin incision at the biopsy site may be left unsutured, sutured, or closed with a Michel clip depending upon personal preference and the size of the incision. The trochar and cannula should be immersed in aqueous hibitane solution before use on the next animal. Parenteral antibiotics may be used if it is thought necessary, but are not used routinely.

Using this technique, with increasing experience 6-12 animals per hour can be sampled with a success rate of greater than 90% even in fractious animals. If no liver sample is obtained on the first attempt the animal should be passed. Complications following liver biopsy are rare.

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5.2   Cobalt (Co)

Introduction

The importance of Co in New Zealand is due to its requirement by the grazing animal. Cobalt is not generally considered to be an essential nutrient for plants, although recent studies suggest that it might be required by some plant species. Cobalt is however, essential for the fixation of nitrogen by rhizobium bacteria. The amounts of Co required are so minute that deficiency severe enough to affect nitrogen fixation is extremely unlikely.

Cobalt deficiency in ruminants is primarily a wasting disease characterised by anorexia, illthrift, cachexia, and anaemia and in some cases death of the affected animals. It is important to recognise that there is an "order" of sensitivity to the disease among grazing animals. The most sensitive are young sheep, then in order, mature sheep, calves in the 6-18 month age group, and least sensitive of all, adult cattle. Horses are not affected.

Co in soils

Cobalt is adsorbed on the soil exchange complex, mainly on the clay-OM complexes. Total Co content range in soils is 0.3 to 200 ppm but Co concentration in the soil solution is very low, often <0.5 ppm.

Soil Co availability to plants is sensitive to both pH and soil moisture status. Plants growing in waterlogged or very acid soils often have much higher Co levels than plants growing on freely drained soils or soils with high pH values. Liming can substantially reduce Co uptake by plants.

Other factors that influence Co availability is the presence of Fe/Al/Mn oxides. These minerals have a high adsorption capacity for Co and are capable of fixation of Co applied in fertiliser to soils. Cobalt appears to replace Mn in the surface layers of these minerals. Mclaren and Metherell (2004) showed that soil Mn plays a crucial role for soil Co availability. Soils with high Mn contents have a high probability of strong fixation of soil Co and showed negligible responses to Co fertiliser. In these soils, soil Co extractants are poor predictors of Co availability to plants. It was suggested that Co in the organic-bound fraction is the most important source supplying Co in soils for plant uptake. Nickel has also been reported to be a competitive element. It is closely related to Co and plants appear to take it up in preference to Co.

Cobalt deficient soils occur in several regions of New Zealand (Figure 5.8). Soils from acid igneous rock, such as granite, generally lack Co while those of basaltic origin have adequate Co. Soils in which Co deficiency can occur are: (1) acidic, highly leached, sandy soils with low total Co; (2) some highly calcareous soils; (3) some peaty soils. Specifically, the main deficient areas are the soils formed on rhyolitic volcanic ash in the central North Island (Pumice Soils), soils formed on granite parent materials in northwest Nelson, and some leached Brown Soils in Southland.

Soil test for Co

Soil Co is measured as either total soil Co or extractable soil Co. The latter method is more likely to measure the Co to be available to the plant. In general, where Co deficiencies in animals are present total Co concentrations are usually less than 2 mg/kg, or extractable soil Co is below 0.25 mg/kg, although soil samples from Morton Mains in Southland have relatively high total Co levels of 3.3-4.8 mg/kg and deficient in Co.

Co in plants

Cobalt is needed for the bacteria in the nodules of legumes and N_2-fixing algae. Co forms a complex with N that is important for the synthesis of vitamin B_{12} coenzyme. Cobalt is also important in the synthesis of vitamin B_{12} in ruminant animals; thus, soil is an important source of plant Co for animals. Plant species differ in their ability to take up Co with legumes tending to have greater uptake than grasses (Table 5.6). However, when soil Co is very low, differences in Co concentrations between grasses and clovers become negligible.

Table 5.6. **Cobalt contents of different pasture plants grown under the same conditions (Andrews, 1971)**

Plant	Cobalt (mg/kg DM)
Timothy	0.09
Cocksfoot	0.11
Meadow Fescue	0.12
Short-Rotation Ryegrass	0.13
Perennial Ryegrass	0.16
Red Clover	0.23
White Clover	0.24

Seasonal variations occur in pasture Co concentrations. The pasture levels are generally lower in the spring and summer and higher in the autumn and winter (Andrews, 1971 and Andrews, 1965). Deficiencies of Co in grazing animals are likely to occur if pasture levels are less than 0.08 mg Co/kg DM.

Historical

"Bush sickness" is a term seldom heard nowadays, yet up until the early 1930s it signified a mysterious deficiency which had taken out of production vast areas of land on which stock sickened and died. In the first report of the New Zealand Department of Agriculture, dated 1893, E Clifton, Stock Inspector for the Auckland district, reported that excessively high losses of sheep caused by a condition known locally as "Tauranga disease", had stopped all progress and settlement in that district. In other areas the disease acquired further regional names such as "Morton Mains Disease" in Southland and "Mairoa dopiness" in the Mairoa region of the King Country.

Until the early 1930s New Zealand investigations were based on the theory that bush sickness was due to a deficiency of iron. In 1933 Australian workers established that it was not the iron

in ilmenite ($Fe_2O_3 \cdot H_2O$) which cured "coast disease" and "wasting disease" but that Co, a trace constituent of the crude material, was the key dietary element. The connection between "bush sickness" and Co deficiency was soon made in New Zealand also.

The fact that Co deficiency was in reality vitamin B_{12} deficiency was not recognised until after the isolation of vitamin B_{12} by American and British workers in 1948. Research workers in New Zealand, of whom Andrews was the most prominent, developed an intense interest in Co deficiency, its diagnosis via tissue vitamin B_{12} levels, and its control. By 1940, areas of deficiency had been mapped in broad outline, diagnostic criteria had been established and methods of controlling the disease, based largely on the use of cobaltised superphosphate, had been established.

In New Zealand the regular use of Co on farms on severely deficient soils has resulted in the virtual disappearance of Co deficiency as a major disease in cattle and to a large extent in mature sheep. However, in young sheep Co deficiency remains a problem and the clinical syndrome is often far from clear cut, a point to be emphasised. The main losses in production from Co deficiency are now in those areas where deficiency is marginal, and a low grade or "sub-clinical" disease is produced where poor performance of stock may be unrecognised or accepted as normal. A further contributing factor to the re-emergence of Co deficiency has been a fall in the use of Co in fertiliser on deficient or marginally deficient areas.

In addition to the classical form of Co deficiency, animal B_{12} status has been linked to a variety of conditions such as polioencephalomalacia in sheep, white liver disease of lambs, infertility and metabolic disease of cattle, and depressed milk production in cows. Also suggested are a variety of lesions of the brain, spinal cord and peripheral nerves, myocardium, and skeletal muscle. Cobalt deficiency has also been linked with Phalaris staggers, and ragwort toxicity.

Function and Metabolism of Co

Cobalt is the essential constituent of true vitamin B_{12}. The term vitamin B_{12} is loosely applied to a group of four metabolically active cobalamines which may be present in animal tissue. The term vitamin B_{12} should correctly be given only to one of these, cyanocobalamin, which is in fact about the rarest of the tissue cobalamines. Microbial production of cobalamin and cobalamin analogues in the rumen is dependent on Co and the organic substrates that the organisms have.

Absorption of true vitamin B_{12} is considered to occur mainly from the small intestine and appears to be enhanced by slower rates of passage of ingesta through the intestine. The intestinal absorption of vitamin B_{12} may vary from about 3% up to 33%. The principal storage depot for vitamin B_{12} is the liver although true storage for a water-soluble vitamin has been debated. Serum vitamin B_{12} levels tend to reflect dietary Co and to a lesser extent liver vitamin B_{12} status.

The primary metabolic defect in vitamin B_{12} deficiency is a block in the utilisation of propionic acid, one of the essential volatile fatty acids produced in the rumen and a main source of blood glucose.
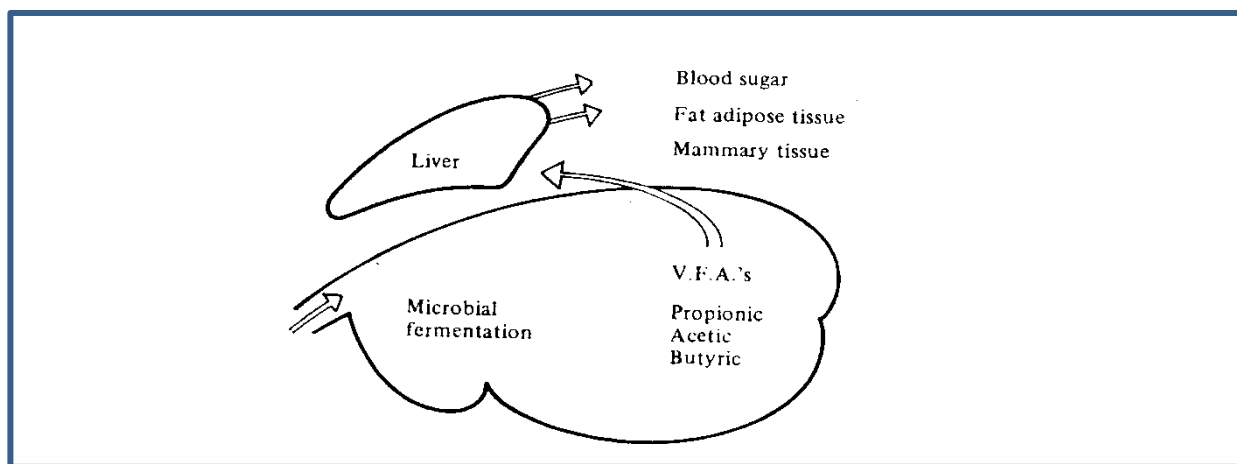
Figure 5.7.   Ruminant carbohydrate metabolism.

Propionate is produced as a result of the fermentation of soluble carbohydrate either directly or through succinate (Figure 5.8).

> *Source*   - Dietary sugars and starches (i.e. soluble carbohydrates)
>
> *Synthesis* - Rumen - microbial fermentation
>
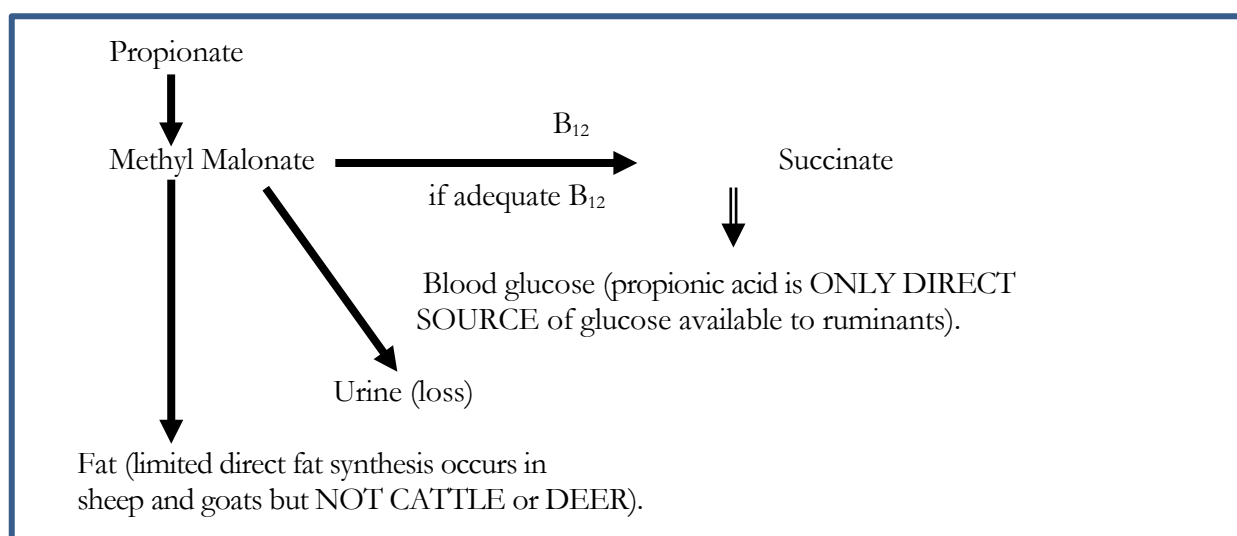> *Transport* - via blood to liver where:-

Figure 5.8.   Ruminant metabolism of propionic acid.

From Figure 5.8 it can be seen that the main pathway of propionate metabolism is via methyl malonate to succinate, in which form it enters the Krebs cycle and can function as a source of glucose. The transformation of malonate to succinate is dependent on the methyl malonyl Coenzyme A isomerase enzyme system, which is dependent on vitamin B_{12}. Hence the failure of this system is the basis of the starvation aspect of Co deficiency. In Co deficient sheep there is a marked increase in methyl malonic acid and propionic acid in the blood with a concurrent increase in methyl malonic acid in the urine also.

Deficiencies of vitamin B_{12} in sheep also interfere with the metabolism of folic acid, because vitamin B_{12} is required for the metabolism of methionine which facilitates the transport of folic acid into liver cells. The fatty infiltration of the liver is considered to be secondary to a deficiency of methionine. Methionine is essential also for optimum wool growth.

Co Requirements

It is believed that pastures that contain 0.11 mg Co/kg DM or greater are adequate to meet the Co requirements of sheep and cattle. Weaned lambs have a vitamin B_{12} requirement of 11 µg/day. Vitamin B_{12} is present in milk at 10.3 µg/l in Co supplemented sheep and may fall to 2.5 µg/l in ewes grazing Co deficient pasture.

In some soils the Co level *per se* is adequate for animal requirements but is not fully available in their diet. In New Zealand one area of classical Co deficiency is the Morton Mains region of Southland that has relatively high total soil Co levels, 3.3-4.8 mg/kg. The development of deficiency depends largely on the extractable Co available. Where animal deficiencies occur, Co concentrations are usually less than 2 mg/kg and the extractable Co is below 0.25 mg/kg.

Acid soils such as granite generally lack Co while the basaltic soils are usually adequate. Sedimentary and volcanic soils often reflect the Co content of the parent rock. For example, the central plateau of the North Island has ash soils from volcanic eruption and is recognised as a Co deficient area.

Weathering, leaching and repetitive cropping can decrease the Co content in soil. Plants growing in waterlogged soils appear to have Co levels many times higher than the same soil types with good drainage. Other minerals such as Mn, Fe and Ni can reduce Co uptake. Liming, by reducing soil acidity, also reduces Co uptake by plants. The legumes tend to have a greater uptake of Co than grasses (Table 5.6), but the differences between plants become negligible when the soil Co is very low.

There are also seasonal variations in pasture Co concentrations in deficient and marginally deficient regions. Pasture levels are generally lower in spring and summer and higher in autumn and winter. In New Zealand Co deficiency appears to be associated with lush spring growth, although the severe clinical signs may not become evident until summer.

The ingestion of Co from soil is important in deficient areas so that in marginally deficient pasture a low grazing intensity is more likely to induce deficiency than heavy grazing. Because liver reserves of vitamin B_{12} can be adequate for 3-4 months the signs of deficiency do not necessarily coincide with the time of lowest intake.

Clinical Features of Co Deficiency

Young growing sheep, particularly weaned lambs, are the most sensitive of all animals to Co deficiency and it is this class of stock that veterinarians are most likely to use to assess the adequacy of Co status. The next group that may be affected are adult sheep, then calves 6-18 months of age and mature cattle in that order. If the weaned lambs are healthy, Co deficiency is unlikely in other classes of animals. The sensitivity of deer to Co deficiency is unknown. No production responses to vitamin B_{12} supplementation have been reported in this species.

Cobalt deficiency is characterised by loss of appetite producing poor growth and hence is essentially a simple starvation, although animals are usually grazing on adequate feed. In sheep, a watery discharge from the eyes may be present and the wool is white or washy and has a reduced growth rate. Normocytic normochromic anaemia may develop later in the disease. A fatty liver may be present on necropsy.

These classical signs of Co deficiency may still be seen, but more commonly one would expect to be confronted mainly with a problem of impaired weight gain in lambs after weaning. Because inappetance is also an important clinical sign of nematode parasitism, assessing the Co status is one aspect of a comprehensive approach to investigating hogget and lamb illthrift.

A number of diseases have also been linked with low vitamin B_{12} status; in sheep polioencephalomalacia, ovine white liver disease and phalaris staggers; in cattle infertility and metabolic disease and depressed milk production. Experimental work in sheep in Scotland has linked Co deficiency in ewes with fewer lambs, more stillbirths and neonatal mortalities (Fisher and MacPherson, 1990).

Diagnosis of Co Status

Of all the trace element deficiencies, the diagnosis of Co deficiency may be the most protracted and requires the use of a full range of diagnostic criteria.

1.   Geographical position of the farm

It is important to consider the location of the property in relation to soil maps of New Zealand (Figure 5.9). Most Co deficient and marginally deficient areas have been well defined. In addition it is important to consider the history of the property with regard to topdressing, pasture species, season, and any previous investigations into illthrift in young sheep. In some cases the results of previous analyses of animal tissue may be available for consideration.
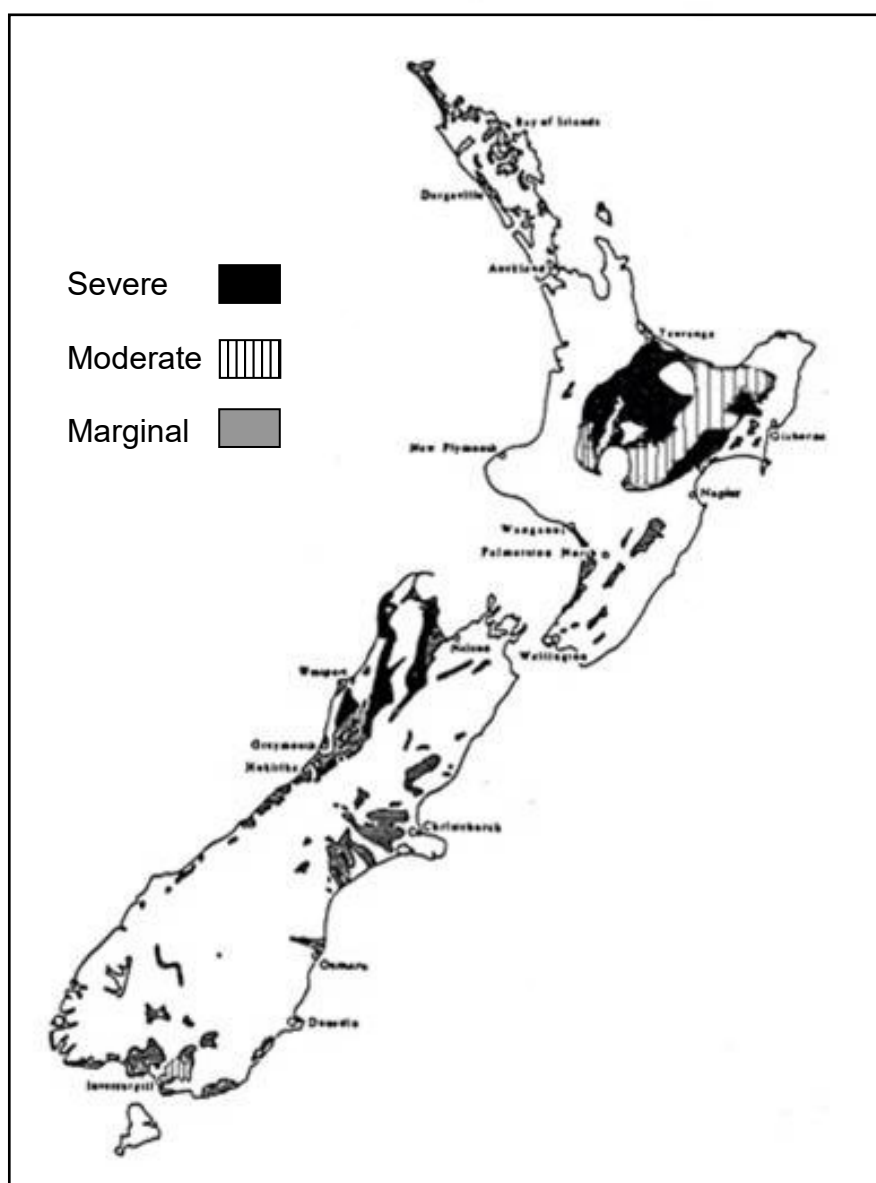
Figure 5.9   Areas of Co deficiency.

2.   Clinical signs

Clinical signs themselves are obviously not diagnostic of Co deficiency but the weighing of a representative sample of hoggets or lambs and comparing the data with a standard scale may convince the farmer just how depressed the animals have become. Observant farmers will usually notice flock anorexia which is always an important feature of Co deficiency.

Figure 5.10.   *A Co deficient hogget (right) compared with a normal hogget of the same age and breed.*

Figure 5.11.   *A group of ewe hoggets with Co deficiency. In May, the average weight of these hoggets was 22 kg.*

3.   Chemical analysis of tissues and blood

Liver and serum vitamin B$_{12}$

The liver appears to be the essential site where vitamin B$_{12}$ is utilised. Thus, liver vitamin B$_{12}$ levels measure reserves and responsiveness and is considered one of the more accurate ways of assessing Co status. While the reference range for sheep has been derived from a number of response trials, there is relatively little reliable data for cattle and levels must be interpreted with caution. At least three liver samples and preferably more are required.

Serum vitamin B$_{12}$ levels reflect Co intake but when liver reserves are low and sheep are grazing pasture of low Co content, then the serum vitamin B$_{12}$ also indicates responsiveness.

Approximately 10 serum samples are required and at this time the reference range only applies to sheep. Serum vitamin B$_{12}$ levels must be interpreted with caution as levels may be elevated if liver damage is present, if sheep have been yarded for even short periods or if sheep have grazed Co sufficient pasture for a few days before they were sampled. The time of sampling and the type of samples collected is likely to vary depending upon the objective of testing, as demonstrated by Table 5.7.

Table 5.7.   Sampling for Co depending on the reason for sampling (adapted from Clark and Ellison, 1993).

Reason	Time to sample	Species and age	Sample type	Sample number	Interpretation
Poor performance	Time of problem	Affected animals	Serum or liver	10 serum, 3 liver	Means only
Farm deficiencies	NI: Nov-Feb SI: Feb-Mar	Weaned lambs	Liver	3	Means only
Adequate reserves	Late spring/ early summer	Weaned lambs	Liver	3	Means only
Supplementation	Halfway through expected period of insufficiency	Animals being supplemented	Liver best or serum	At least 3 liver or 10 serum	Means only

NI: North Island
SI: South Island

In areas classified as marginally Co deficient, year to year variations are likely to occur in the incidence of deficiency in lambs. In these areas it is advisable to sample unsupplemented lambs at weaning, especially in seasons with lush spring growth.

Interpretation of laboratory results

Table 5.8.   Diagnostic criteria for Co deficiency.

	Sheep Vitamin B_{12} Serum pmol/l	Sheep Vitamin B_{12} Liver nmol/kg	Cattle Vitamin B_{12} Liver nmol/kg
Responsive	<336	<280	<75
Marginal	336-500	280-375	75-220
Adequate	>500	>375	>220

It is important to recognise that the probability of achieving a growth rate response to supplementation decreases as the serum or liver vitamin B_{12} levels increase. Thus even lambs with levels in the "responsive" range may not show a significant growth response to supplementation in every case. The following graphs, Figures 5.12 and 5.13 best illustrate this.

The interpretation of these graphs is as follows:

a)   the individual open circles and crosses (o, x) relate to individual experiments investigating Co deficiency.

b)   the dotted line (- - -) shows the linear regression best fit curve of the liveweight response (g/day) for these experiments.

c)   the solid line (——) represents the probability of obtaining a weight gain response of >10 g/day at various serum or liver vitamin B_{12} levels.
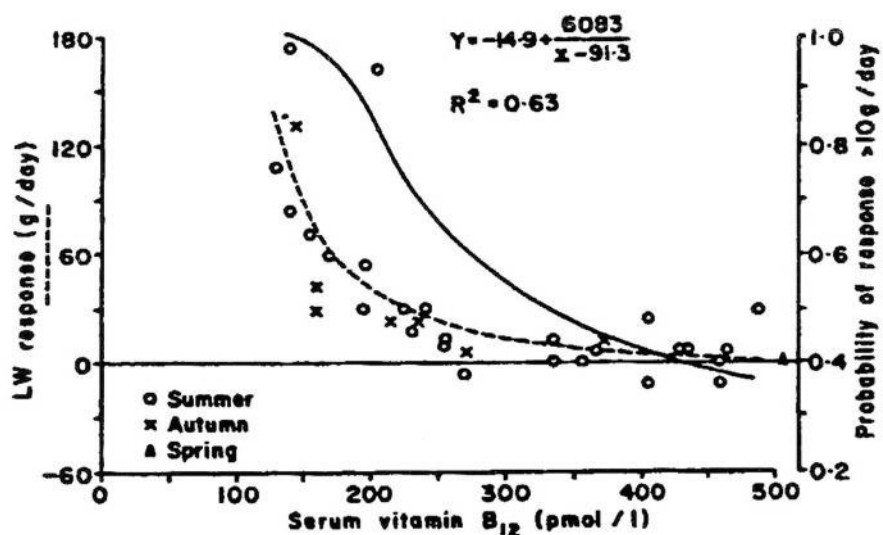

Figure 5.12.   *Serum vitamin B_{12} levels related to weight gain response and probability of a weight gain response of <10 g/day in response to vitamin B_{12} treatment for data closest to the middle of January (from Clark, 1989).*

$$Y = -24.3 + \frac{8240}{x - 42.0}$$

$$R^2 = 0.84$$

Figure 5.13. *Liver vitamin B_{12} levels related to weight gain response and probability of a weight gain response of <10 g/day in response to vitamin B_{12} treatment for data closest to the middle of January (from Clark, 1989).*
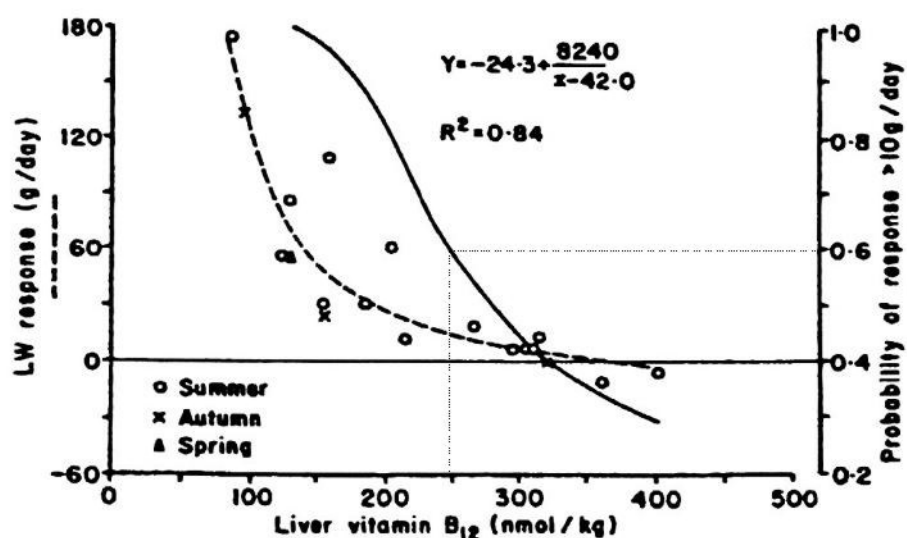
It can be seen from these graphs that, for example, lambs in January with mean liver vitamin B_{12} levels of 250 nmol/kg (in the "responsive" range) have an approximately 60% probability of achieving an economically significant growth rate response to supplementation. It would be prudent to supplement these lambs but a response is not guaranteed.

It has been suggested that the reference range for vitamin B_{12} in sheep should vary for different age groups. Thus the cut-off values for ewes should be lower than that for hoggets, which should be lower than that for lambs (Clark, 1998).

Other diagnostic tests

Urinary methylmalonic acid and/or formiminoglutamic acid are possible diagnostic tests as both these metabolites are elevated in Co deficiency. They are not currently offered by the diagnostic laboratories as they are more costly than liver and serum vitamin B_{12} tests and while they confirm the presence of deficiency they are not as useful in predicting the likelihood of a response.

Pasture analysis for Co levels are of limited value in assessing the Co status of animals unless they are performed on a regular basis (probably monthly). This is because animals can survive for considerable periods on low pasture Co due to a liver storage of vitamin B_{12}. Soil analysis may give an indication of deficiency but many factors influence the level of Co in the soil that is available to the animal.

4. Controlled response trials

Controlled response trials have been used extensively in the past to diagnose Co deficiency and while they are a definitive test they may fail to diagnose marginal deficiencies in certain years or instances where the trial is not conducted at the correct time. Chemical analysis should always be conducted in conjunction with these trials.

The most commonly used trial is a weight gain response in weaned lambs using approximately 50 control lambs and 50 lambs treated with an injection of vitamin B_{12}. Regular weighing is required and the lambs must be individually identified with an ear tag.

Treatment and Prevention of Co Deficiency

Topdressing

Following the discovery of Co deficiency as the cause of "bush sickness", topdressing pastures with cobaltised superphosphate at a rate of 350 g Co sulphate/hectare/year became a standard practice in deficient areas of New Zealand. Although only between 1% and 4% of Co is taken up by plants and utilised by animals it proved to be effective, simple and economical. On pumice soils where Co has been applied regularly for 10 years or more, Co levels have remained satisfactory for three or four year following cessation of topdressing and as a result it was recommended that the rate of application on these farms be reduced to 175 g/ha of Co sulphate, every 3 years. In some instances, even lower rates have been applied and Co deficiency has occurred as a result. In Co deficient areas Co topdressing gives good protection of young lambs for up to 7 months that in most cases should take them through the danger period. It should be applied before lambs are weaned, because of the short-term nature of Co sulphate in raising the Co levels. On soils with a high Mn content, Co topdressing may be less effective. Although a valuable and effective way of supplementing Co to animals, the relatively high cost of Co has diminished the attractiveness of topdressing as the sole means of supplementation. In areas where deficiency is infrequent, direct supplementation of lambs with Vitamin B_{12} is likely to be a more-cost effective method of supplementation.

The use of slow release Co fertilisers, have been suggested to avoid the pattern of immediate elevation of herbage Co concentration to high levels followed by a rapid decline to deficient levels. This is specially the case with soluble Co fertilisers such as $CoSO_4$. Sherrell (1990) proposed the use of a slow release source of Co. However, initial field evaluation of slow release Co fertiliser prills based on Co hydroxide ($Co(OH)_2$) showed that they were ineffective. The release rate was too slow and the fertiliser was not able to elevate the herbage Co concentration to the require level. Recently, Perrott *et al.* (2004) reported the results from a field experiment using Co-containing phosphate glasses with a range of Co-release rates. The fastest release was found to elevate herbage Co concentration above control values for 2 to 3 months longer than $CoSO_4$. They concluded that the length of periods of elevated Co concentration in herbage could be increased by using material with slightly slower release rate.

Oral dosing

Because animals require a regular dietary intake of Co, drenching is not a practical form of treatment. The recommended oral dose for lambs is 7 mg Co (35 mg $CoSO_4.7H_2O$) per week or 35 mg Co (175 mg $CoSO_4.7H_2O$) for cattle per week. Monthly dosing with 300 mg Co has been used to reduce death rates in severely deficient country but does not prevent sub-optimal growth. Mineralised anthelmintics contain about 2-3 mg of Co/10 ml and are not a sufficient form of supplementation.

Vitamin B$_{12}$ injections

In New Zealand two types of vitamin B$_{12}$ injections are available. Water-soluble formulations will boost levels of the vitamin for a period of about a month. The period of protection given by vitamin B$_{12}$ injections depends on the liver reserves of the animal and the level of Co deficiency in the pasture. A long-acting product incorporating vitamin B$_{12}$ in a lactide/glycolide polymer is also available. This product has been reported to raise liver and serum vitamin B$_{12}$ levels of lambs for at least 210 days and of calves for at least 110 days and may be given to lambs in Co deficient areas at docking time. Vitamin B$_{12}$ injections are widely used in many areas of New Zealand to protect lambs against Co deficiency and may be combined with injectable anthelmintics or vaccines.

Co bullets

Cobalt bullets are available in New Zealand and give a period of protection for at least a year. Care must be taken in dosing to avoid damage to the larynx or pharynx. Anthelmintic capsules frequently include mineral additives including Co.

Pasture foliar spraying

Boom spraying of pasture with 0.14 kg CoSO$_4$ per/ha is a supplementation method that may be utilised on flat or rolling country. The mixture should be sprayed onto actively growing pasture 2-3 weeks pre-grazing to allow suitable uptake by the grass before it is grazed.

Other methods of supplying Co include the use of CoSO$_4$ in drinking water and Co in stock licks. Neither of these methods is particularly satisfactory in ensuring that animals ingest adequate levels of Co.

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5.3   Copper (Cu)

Introduction

Copper is an essential element required by a number of enzymes involved in specific oxidase-type reactions in the animal's body. Copper is derived by grazing animals from plants, which in turn have drawn their Cu from the soil. Under many grazing situations the Cu in soil and plants is not easily available for absorption by the animal because of interference by other elements. Large areas of New Zealand have soils, which are either low in available Cu, or have elements that interfere with Cu uptake so that the Cu requirements of many animals, particularly cattle, are inadequate. Copper deficiency, both clinical and sub-clinical, is common and always represents a challenge to the veterinarians in diagnosis and effective therapy. If this is not conducted carefully, ineffective therapy may result. In some cases Cu poisoning may occur, particularly in sheep.

Cu in Soil

New Zealand soils commonly contain about 17 ppm of Cu. Some New Zealand soils have levels as low as 2 ppm, but most rarely exceed 25 ppm of Cu. The availability of Cu in soils to plants and animals is extremely complex. Copper may be bound in the soil, in water-soluble form, as exchangeable ions, as organic complexes, in association with Fe, Al, Mn and other mineral complexes.

The cycling of Cu in soil is similar to that of Fe and Zn. Primary and secondary minerals dissolve to initially provide Cu to the soil solution, which is then adsorbed onto the soil exchange complex and incorporated into the microbial biomass and complexed by organic compounds in the soil solution. Up to 99% of Cu in soil may be complexed with organic matter. The inorganically and organically complexed solid phase Cu is in equilibrium with soil solution Cu of which generally less than 5% is present as the free Cu^{2+} ion, the remainder being bound to water-soluble organic matter complexes (Loganathan *et al.*, 1998).

Soil texture, drainage, redox potential, pH and organic matter govern soil solution Cu concentration and its effects on Cu uptake by plants.

Increasing soil pH with lime application generally decreases the plant availability and the mobility of Cu in soil by increasing the adsorption of Cu^{++} ions onto iron and Mn oxides, clay minerals and organic matter. Over-liming can render Cu largely unavailable from soil because Cu precipitates as $Cu(OH)_2$ at pHs above 6.5. Lime topdressing raises the soil pH and increases the availability of Mo. This has been associated with Cu deficiency in New Zealand and is also an important cause of Cu deficiency in Scottish hill country.

There are numerous interactions involving Cu and other nutrients. Increasing the N supply to crops can reduce mobility of Cu in plants, since large amounts of N in plants impede translocation of Cu from roots to shoots and from older leaves to new growth. Also, high concentrations of Zn, Fe, and P in soil solution can depress Cu absorption by plant roots and may intensify Cu deficiency.

Because of these complicating factors the diagnosis of Cu deficiency in animals by the measurement of Cu in soil over which they graze is of little value.

Soil test for Cu

Soil test such as 0.02M EDTA, 0.005M DTPA and 0.1M HCl extractions of soil Cu have been used to determine plant-availability of soil Cu. In a glasshouse study on New Zealand pasture soils (Loganathan *et al.*, 1998), it was found that the Cu concentration in clover, grass shoots and old grass roots had no relationship with soil properties, soil Cu extracted by the above soil tests and soil solution Cu^{2+} concentration. Based on later studies it was hypothesised that the rate of Cu uptake by plants depends both on the concentration of organically complexed Cu in the soil solution and the stability of this complex to pH change (Liao *et al.*, 2000). As the pH decreases the proportion of dissolved Cu in free Cu^{2+} form will increase thus, increasing the plant-availability of Cu. The extent of the increase in free Cu^{2+} concentration per unit decrease in pH is dependent on soil type. The rhisosphere soil pH of plants in most situations is lower than that in the bulk soil. Therefore, as the organically bound Cu in soil solution moves from bulk soil towards the plant roots, it dissociates to release Cu^{2+} for uptake by plant roots.

Cu in Plants

Plants absorb Cu as the cupric ion, Cu^{2+}. Copper is involved in many important plant processes. It is present in several oxidase enzymes, including cytochrome oxidase and ascorbic acid oxidase, and is essential for photosynthesis and protein and carbohydrate metabolism. Copper also appears to be a requirement for nitrogen fixation by rhizobium bacteria. Plant tissues normally contain 5-20 ppm dry matter of Cu. Copper deficiency usually occurs when the concentration falls below 4 ppm while toxicity occurs when levels rise above 20 ppm. Critical Cu concentration in mixed pasture herbage for adequate Cu nutrition of young sheep has been reported to be 10 ppm (Morton and Roberts, 1999). In New Zealand very few areas grow pastures that contain more than 13 ppm of Cu. The young shoots of plants contain the highest amounts of Cu, and the levels decrease as the plant matures.

Legumes have a higher affinity for Cu than do grasses, especially when they are growing in soils of high Cu content. Within the legume species, lucerne appears to be more sensitive to Cu deficiency than clover (Table 5.9). Seasonal variation in pasture Cu concentration is small. The application of Cu fertilisers to Cu deficient soils will increase the Cu content of plants growing in that soil, but nitrogenous fertilisers, that promote plant growth, tend to reduce the pasture Cu content. The Cu content of plants along with Mo, S and Fe levels are frequently measured during the investigation of the Cu levels in animals as they are important factors in deciding the most appropriate form of therapy. See later sections for more information on Mo and S influencing Cu absorption by animals and its dependence on Mo and S levels in pasture.

Table 5.9.   Sensitivity of crops to low levels of soil available Cu

High sensitivity	Mild sensitivity	Low sensitivity
Wheat	Oats	Canola
Lucerne	Corn	Soybeans
Sudangrass	Timothy	Forage grasses
Carrots	Clover	Potatoes

Cu in Animals

Animals obtain Cu mainly from plants but also from ingested soil. Copper is absorbed from the intestinal tract, transported in the blood loosely bound to albumin and most is stored in the liver which may contain between 40-70% of the total body Cu.

By the twelfth week of gestation the lamb foetus is beginning to accumulate Cu in its liver. This coincides with a concurrent decrease in the ewe's liver stores. The newborn lamb absorbs Cu very efficiently. Immediately after birth almost 100% of the lamb's Cu intake is absorbed, but this decreases to 10% by weaning.

Adult sheep absorb less than 10% of dietary Cu and only about 5% of this intake is liver stored. The absorption of Cu is partially controlled by the sheep as during lactation there is a temporary increase in the efficiency of absorption. A similar situation is also found in sheep with hypocupraemia. The efficiency with which Cu is assimilated from the diet depends on the solubility of the Cu, which in turn is dependent upon pH. In the rumen, most of the Cu is present in an insoluble form, while abomasal absorption is minimal, but with the rising pH of the ingesta distally, Cu is freed and absorbed in the ileum and large intestine.

The differences in the absorption rate and in the excretion rate of Cu between species may affect the Cu requirements of the animal. Sheep are more susceptible than other domestic ruminants to Cu accumulation and toxicity. The reason for this is that they have less control over intestinal homeostasis, a limited capacity to excrete Cu from the liver and their lysosomes are unable to sequester large amounts of Cu.

Factors influencing Cu absorption and metabolism

(i)   Age

Newborn lambs have a higher total Cu concentration than their dams; up to 50% of their Cu being present in the liver. However their plasma Cu levels are lower than those of their dam as the foetus is unable to synthesise caeruloplasmin. The latter rises rapidly to adult levels seven days after birth. Young lambs are able to absorb Cu very efficiently while they are on a liquid diet. This is due to the high absorption capacity of the large intestine, and the lack of interference by sulphide in the rumen. The efficiency of absorption falls with age as the rumen becomes functional and only 2-10% of ingested Cu is then absorbed.

(ii)   Pasture composition

The amount of Cu absorbed from pasture varies considerably, depending on the species of plant and the season. The Cu contained in stored fodder such as hay or silage may be absorbed at a higher rate than that of pasture. However silage can have very low available Cu which may be related to the amount of soil (and thus iron and Mo) that contaminates the silage when it is being made. The Cu absorption from various feeds is shown in Table 5.10.

Table 5.10.   Copper absorption from various feeds (Suttle, 1981).

Feed	% Absorption
Summer pasture	2.3
Autumn pasture	1.2
Hay	7.2
Silage	4.9
Leafy brassicas	12.8
Root brassicas	6.7

(iii)   Genetic influence

There is considerable variation between breeds and strains of sheep in their ability to absorb Cu.

(a)   Sheep on the island of Orkney would normally eat a considerable amount of seaweed which interferes with Cu availability. By a process of natural selection they have become very efficient at absorbing Cu from the diet and when shifted to Britain, sheep died of Cu poisoning once seaweed was absent in their diet.

(b)   Wiener in Scotland has demonstrated the genetic influence affecting the occurrence of swayback in lambs. Not only was the incidence of swayback variable between breeds, he was able to select sheep for high and low Cu absorption rates based on serum Cu levels from within a population.

Occurrence of swayback in different sheep breeds run on the same pasture:

Scottish Blackface	40%
Welsh mountain	0%
Cheviot	11%
S. Blackface x Cheviot	20%
S. Blackface x Welsh Mountain	15%
Cheviot x Welsh Mountain Weiner (1966)	5%

Thus Orkney, Texel and Welsh Mountain sheep are more efficient at absorbing Cu and are less likely to show signs of Cu deficiency. They are however, more susceptible to Cu poisoning. In New Zealand sheep breeds, the Texel is efficient at absorbing Cu and is more susceptible to Cu toxicity than other breeds. In contrast the Finnish landrace is more susceptible to Cu deficiency and have similar Cu requirements to cattle.

The absorption differences for Cu shown between breeds of sheep, diminishes as the Mo content of the diet is increased. It is also believed that the genetic variation is due to differences in absorption of Cu rather than in its utilisation and excretion.

(iv)   Gastrointestinal parasitism

It has been suggested that gastrointestinal parasitism may reduce Cu absorption. This is probably similar to the parasitic effects shown in the absorption of macro-elements such as Ca and phosphorus.

(V)   Molybdenum and S

Molybdenum is present naturally in some soils and in others it has been used as a fertiliser to

stimulate legume growth. Sulphur is applied in fertiliser as in superphosphate, gypsum or elemental sulphur and may reach quite high levels, particularly on dairy farm pastures (sheep farms 0.1-0.25%, dairy farms 0.3-0.4%). Pasture Mo levels increase during spring and along with sulphur form insoluble thiomolybdates in the rumen which then combine with Cu, forming insoluble Cu thiomolybdates. In addition to the interference of Cu absorption in the gut tetrathiomolybdate has a post-absorptive effect on systemic Cu metabolism by increasing Cu excretion in the bile and thus reducing liver Cu reserves.

Depending on the species, cattle or sheep, and the relative levels of Mo and sulphur present, it is possible to predict, at least roughly, whether a given diet is likely to be Cu deficient.

It must be emphasised that plant analysis should not be used alone to establish the Cu status of a farm and its animals. It must be remembered:

- That animals can store (particularly sheep) considerable reserves of Cu which can be mobilised during periods when dietary supply is inadequate.
- Copper, Mo and sulphur concentrations in herbage vary throughout the year.
- The concentrations of the three elements vary between plant species and considerable variation is possible within a single paddock. As a result, care must be taken when collecting pasture samples to ensure that they are representative of what the animals are eating.

Naturally high soil Mo occurs in few places in the world, e.g. the "Teart" areas of Somerset, England. In New Zealand a few Northland soils give pastures with >3 ppm Mo. Some of the Kaipara site soils and some peats may also approach this level also the Balmoral soils in a small area of North Canterbury.

The heavy use of molybdic fertilisers and lime altered the Cu:Mo ratio in herbage on many farms in the 1960s and 1970s, causing a spring rise in Mo to >5 ppm. The excessive use of these fertilisers was believed to have been responsible for the first Cu deficiency diseases in Northland in the same period (post parturient haemoglobinuria). Since then the disorders have been largely controlled because of more informed fertiliser usage and Cu supplementation.

As a rule of thumb Cu:Mo ratios of 2:1 in the feed of housed sheep lead to Cu deficiency but in pasture fed animals because of high levels of sulphur, higher ratios of Cu:Mo may be dangerous and Mo levels as low as 2 ppm in pasture may induce Cu deficiency in cattle.

(vi)   Iron

Iron is a major component of soil and high levels of soil ingestion are common in New Zealand stock. In some circumstances up to 30% of total dry matter ingested may be soil.

Dietary Fe intake can have a marked inhibitory effect on the utilisation of dietary Cu. Levels as low as 250 mg Fe/kg diet are sufficient to reduce hepatic Cu reserves of calves. Concentrations of Fe in soils are frequently approximately 20,000 mg/kg. If soil ingestion accounts for 10% of an animal's dry matter intake and if only 25% of Fe were released, Fe would be equivalent to 500 mg/kg which would significantly reduce the utilisation of dietary Cu.

Workers at the Rowett Institute in Aberdeen found that 800 mg Fe/kg diet had the same effect on Cu status, as did 5 mg Mo/kg diet. Unfortunately, little is known of the mechanisms whereby Fe disturbs Cu metabolism.

In New Zealand there are several reports where Fe appears to have been the main element associated with severe Cu deficiency syndromes. In the Waipipi ironsand area of South Taranaki, classical cases of Cu deficiency were reported in cattle in 1980-1982. In the animals examined, the liver reserves of Cu were very low, but the pasture levels of Cu, Mo and S were such that a severe Cu deficiency should not have existed (Table 5.11).

Table 5.11   **Animal tissue and pasture analysis from a Taranaki farm with severe iron induced hypocuprosis in cattle (Bruère, 1982).**

	Liver Cu mg/kg	Required by cattle mg/kg	Pasture Analysis			
			Cu mg/kg	Mo mg/kg	S%	Fe mg/kg
1	24.0	10.0	12.0	.20	.24	10,740
2	6.3	11.0	13.0	.10	.30	3,260
3	16.0	10.0	9.0	.20	.23	2,060
4	6.0	11.5	12.0	.20	.31	4,280
5	4.2	10.0	18.0	.09	.24	29,200

Mean pasture levels   Fe 9908
Range   2,000-29,200 mg/kg
Normal   legumes   200-300 mg/kg
         lucerne   700-800 mg/kg
         grass   100-200 mg/kg

The Fe levels on the other hand were extraordinarily high (range 2,000-29,299 mg/kg/DM). Such high levels of Fe were due to soil contamination of the fodder which it is believed led to the severely depressed Cu status of the grazing cattle.

Many New Zealand soils have a naturally high Fe content and other factors may have to contribute before a serious effect on Cu uptake occurs, as cited above. High levels of Fe are found in some ground waters and particularly bore water may be seriously contaminated. An aspect that warrants further investigation in New Zealand is drinking water as a source of Fe, particularly for dairy cows. Many dairy sheds have water supplies heavily contaminated with Fe. Similarly water logged soils tend to support plants with high available Fe and a positive relationship has been established between waterlogging and hypocuprosis.

A more general association of hypocuprosis with waterlogging has been observed at times on the Hauraki plains and is combined with a low pasture Cu. The high ingestion rates of soil by some stock, particularly over the winter period, in this area is also associated with qualitative differences between soil types in the extractability of their mineral elements in rumen liquor. Iron in the Hauraki Plains clay loam appears to be more soluble in rumen liquor than in other soils. This solubility is also retained in duodenal liquor which is a major site of Fe absorption.

(vii)   Other metals

Other metals which may affect the uptake and/or utilisation of Cu by grazing ruminants include zinc, cadmium, silver and mercury. Zinc (Zn) is regularly used in facial eczema prevention in New Zealand and could be a contributing factor to hypocuprosis, particularly in dairy cattle, which may be dosed with Zn drenches each day for several weeks.

Concern has also been expressed about the levels of cadmium in some lines of superphosphate, which could also contribute to inducing Cu deficiency.

The role of Cu and its metabolism

After intestinal absorption much of the Cu is rapidly deposited in the liver hepatocytes: the distribution being 20% in the nuclear fraction, 10% in microsomes and 20% in the large granules of mitochondria and lysosomes. The remainder is stored in the cytosol as either copper-dependent enzymes or metallothionein. As the liver becomes saturated with Cu the kidneys become the secondary site of Cu deposition.

The main transport of Cu is by the globulin caeruloplasmin. Caeruloplasmin is synthesised in the liver and its rate of formation depends on the hepatic Cu concentration. Blood actually contains Cu in five separate fractions: in erythrocytic superoxide dismutase (60% of erythrocyte Cu) in an erythrocyte Cu complex, in plasma caeruloplasmin (60-95% of plasma Cu) albumin Cu and bound to plasma amino acids. The total Cu content of the erythrocyte does not fluctuate in spite of variations in the Cu status of the animal and erythrocytes are not involved in Cu transport.

Copper is incorporated into the molecular structure of five major enzymes and several less important ones, as well as being part of several proteins and amino acids. The main Cu dependent enzymes are listed in Table 5.12.

Table 5.12.   Essential Cu containing enzymes and their functions.

Enzyme	Activity	Source	Function
Caeruloplasmin	Ferroxidase	Plasma	Iron transport
Cytochrome oxidase	Terminal oxidase	Mitochondria	Energy metabolism and phosphorylation
Lysyl oxidase (amine oxidase)	Peptide cross-linkage	Aorta and cartilage	Collagen and elastin formation
Tyrosinase (polyphenol oxidase)	Oxidase	Melanocytes	Tyrosine to melanin
Superoxide dismutase	Dismutase	All aerobic cells	$O_2 + O_2 + H_2O_6 H_2O_2 + O_2$ (antioxidant)
Dopamine-B-hydroxylase	Oxygenase	Adrenal gland	Dopamine to norepinephrine

Cytochrome oxidase is the terminal enzyme in the oxidative phosphorylation process. Young animals appear to be more severely affected by deficiency than older stock, probably because faulty oxidation interferes with the development of growing tissues. Loss of cytochrome oxidase activity is associated with a reduction in the formation of myelin lipids, as seen in the central nervous system of swayback affected lambs.

Caeruloplasmin (ferroxidase I) is an oxidative enzyme involved in releasing Fe into plasma from stores during erythropoiesis. Thus Cu is essential for the normal functioning of Fe in the living organism. Some Cu is also essential in erythrocytes where it is found in the same concentration as plasma, and when deficient, the life span of the red cell is reduced. A deficiency of Cu can thus lead to anaemia resulting from shortened red cell life span, and from limited capacity of the bone marrow to produce red cells.

The enzyme lysyl oxidase (amine oxidase) is involved in elastin and collagen synthesis. These give strength and elasticity to connective tissue and cartilage, thus a deficiency may lead to

skeletal defects, abnormal gait and fragility of blood vessels (aorta). In sheep and cattle grazing Cu deficient pastures spontaneous fractures have often been observed.

Tyrosinase, an oxidase important in the keratinisation of fibres, is also required for the conversion of tyrosine to melanin, needed for pigmentation. In this respect the wool of black sheep has been found to contain twice as much Cu as that of white sheep.

Cu Requirements of Sheep and Cattle

If the Cu associated with the liver is excluded, a fully fleeced sheep contains about 60 mg of Cu; each kilogram of bodyweight containing 0.8 mg of Cu and each kilogram of wool 6-8 mg of Cu. A newborn lamb contains a total of 10 mg of Cu. The net requirements for maintenance rarely exceed 4 μg of Cu per kilogram of bodyweight per day and show no relationship to metabolic rate. Growth requires approximately 1.1 mg of Cu per kilogram of bodyweight increase and the lactating ewe requires an extra 0.3 mg of Cu for each litre of milk produced.

The amounts of Cu associated with the endogenous loss, growth, pregnancy and lactation in sheep and cattle are shown in Tables 5.13, 5.14 and 5.15 (Grace, 1983).

Table 5.13. **The amounts of Cu associated with the endogenous loss, growth, pregnancy and lactation in sheep and cattle.**

	Sheep	Cattle
Endogenous loss (inevitable loss)	4 μg Cu/kg liveweight	7.1 μg Cu/kg liveweight
Growth	1.1 mg Cu/kg gain	1.0 mg Cu/kg gain
Lactation	0.32 mg Cu/kg milk (early) 0.22 mg Cu/kg milk (late)	0.1 mg Cu/kg milk
Pregnancy (daily increment in conceptus)		
- early	0.015 mg Cu/day	0.60 mg Cu/day
- mid	0.085 mg Cu/day	1.63 mg Cu/day
- late	0.186 mg Cu/day	2.07 mg Cu/day
Wool	7mg Cu/kg	

Table 5.14. Estimated dietary Cu requirements of sheep.

	Liveweight (kg)	Weight gain (g/day), stage of pregnancy or milk yield	Dietary req. mg Cu/day	mg/kg DM
Growing lamb	5	150	0.21	1.0
	10	150	0.40	1.0
	20	150	1.2	1.8
	30	150	3.7	2.9
	40	75	2.7	2.1
		150	3.7	2.2
		300	5.6	2.7
Adult ewe				
- maintenance	55		4.8	5.0
- late pregnancy	55	single	6.6	5.0
		twins	10.0	5.9
- lactation	55	1kg milk/day	10.0	4.4
		2kg milk/day	15.5	5.2

Table 5.15. Estimated dietary requirements of cattle.

	Liveweight (kg)	Weight gain (g/day), stage of pregnancy or milk yield	Dietary req. mg Cu/day	mg/kg DM
Pre-ruminant calf	40	0.5	1.2	-
Growing calf	100	0.5	25	10
		1.0	35	10
	200	0.5	39	9
		1.0	48	8
	300	0.5	54	9
		1.0	65	8
Dairy cow				
- maintenance	380		54	10
- late pregnancy	380	9 months	95	10
- lactation	380	10 kg milk/day	74	8
		20 kg milk/day	94	7
		30 kg milk/day	114	7
Beef cow				
- lactation	450	10 kg milk/day	84	10

Clinical Features

In general, cattle and deer are more susceptible to Cu deficiency than sheep so in a mixed-farming enterprise clinical signs of deficiency usually develop in these species first.

Cattle

One of the earliest signs to develop in cattle is loss of coat colour, followed in order by diarrhoea and unthriftiness, skeletal defects and anaemia. It is important to recognise that these are relatively non-specific signs and similar signs would be seen with other causes of ill-thrift such as internal parasitism.

(i)   Growth rate

The effect of Cu most frequently responsible for economic loss is the decline in growth rate occurring in calves and yearlings when deficiency becomes established. The reason why this effect arises and why it is often such a variable characteristic between individual animals exposed to the same dietary circumstances is not known. Research has shown that the small intestine of animals undergoing Cu depletion suffers marked cellular damage that is probably of sufficient severity to affect absorptive function, and this may underlie the marked decline in efficiency of food utilisation that has been shown to accompany the early development of Cu deficiency induced experimentally.

(ii)   Diarrhoea

Marked persistent diarrhoea occurs in many cases of secondary Cu deficiency induced by hypermolybdenosis (peat scours and teart) providing further evidence of a key role for Cu in the maintenance of normal function of the gastro-intestinal tract.

(iii)   Skeletal defects

These are relatively uncommon in cattle in New Zealand but when they do occur it is mainly in young rapidly growing animals. Affected animals commonly have a stilted gait, show ataxia during movement, but they may recover temporarily after rest. Boney changes include marked reduction in the thickness of the shaft wall, thickened epiphyses at the fetlock of calves causing them to become stiff and swollen. Spontaneous fractures of ribs and limbs may also be seen when hypocuprosis is severe.

(iv)   Coat colour changes

Copper is involved in the production of hair pigment so that in a deficiency there is a lightening of the coat colour. Black areas have a brown-grey tint (Angus cattle) and red areas become yellow-sandy (Hereford cattle). In black cattle "spectacles", patches of grey hair, often develop around the eyes.

(v)   Poor reproductive performance

The direct effects of Cu deficiency on reproduction are still debated. Reproductive performance may suffer when animals are exposed to pastures of high Mo concentration because Mo inhibits the activity of reproductive hormones and responses to Cu may arise secondarily because it acts as an antidote to Mo.

(vi) Depressed milk yield

It is thought that Cu deficiency can have an adverse effect on milk yield but there have been few published experiments detailing the milk yield response of dairy cows to Cu supplementation. In these experiments the response to supplementation was variable.

(vii) Anaemia and post parturient haemoglobinuria

In Northland, New Zealand, a relationship was found to exist in cattle between low Cu status and the incidence of post parturient haemoglobinuria. In addition the occurrence of post parturient haemoglobinuria in a herd can also indicate the presence of an anaemic state which usually affects the majority of the herd. The Cu deficiency is usually associated with the excessive application of Mo fertiliser and lime to pastures. However other factors such as the excessive excretion of phosphorus play an important role in the cause of this disease.

(viii) Cardiovascular disorders

These have been reported in Western Australia and involve slow degeneration of the myocardium with replacement fibrosis. Occasionally cattle die of acute heart failure or "falling disease".

Figure 5.14.   *Severely Cu deficient cow and her calf.*

Figure 5.15.
Bilateral spontaneous fractures of the humerii of a hogget due to Cu deficiency. This problem was induced following topdressing with Mo.
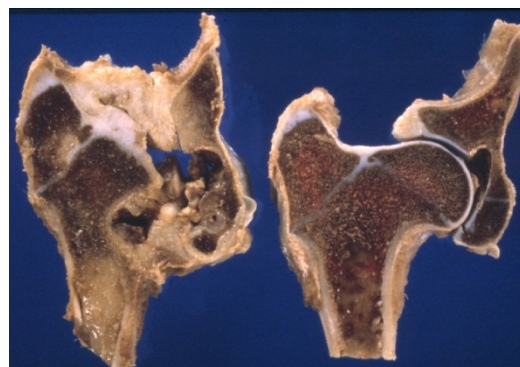

Figure 5.16.
Osteochondrosis of the femoral head of a Cu deficient deer calf (left) compared with a femur from a normal deer calf.

Deer

Enzootic ataxia

The most common manifestation of Cu deficiency in deer has been enzootic ataxia which occurs mainly in adult deer. There is the gradual onset of incoordination especially of the hind limbs and on necropsy, demyelination of the spinal cord. The reason for the age difference between deer and sheep in the onset of enzootic ataxia has not been explained. Bone disorders such as osteochondrosis have been reported. Reduced growth rates may also occur in deer with Cu deficiency.

Sheep

In New Zealand visiblesigns of Cu deficiency are seldom seen in adult crossbred sheep even when there is a marked depletion of liver Cu. However a wide variety of clinical abnormalities of grazing sheep have been attributed to a dietary deficiency of Cu. These include loss of wool crimp, loss of pigment, anaemia, loss of condition, bone disorders, impaired reproductive performance and neonatal ataxia (swayback).

In Britain, the commonest manifestation is "swayback" in lambs while in Australia wool abnormalities and anaemia are seen. In New Zealand probably bone fragility of lambs and hoggets and occasional cases of swayback are most commonly reported.

(i)   Swayback - enzootic ataxia

This condition is characterised by hypomyelinogenesis of the central nervous system so the lambs are unable to stand or have incoordination of the hind limbs. It may be congenital which involves hypomyelinogenesis largely of the cerebrum, or delayed in which the lambs are apparently normal at birth but become affected in the first few weeks of life. This is due mainly to hypomyelinogenesis of the spinal cord.

It is thought that there are two peak periods during which myelination of nervous tissue takes place in the sheep's development: one period around twenty days prior to birth, the other ten to twenty days after birth.

(ii)   Bone fragility, osteoporosis

Bone fragility, particularly of long bones (humerus and femur) is recorded in New Zealand as a result of Cu deficiency. This may be seen at docking time as an increased occurrence of fractured bones when lambs are released from the docking cradle, or it may occur in hoggets during autumn and winter and is usually associated with yarding or stock movements (Figures 5.14 and 5.15).

(iii)   Wool abnormalities

In black sheep in New Zealand Cu deficiency has been shown to be associated with loss of pigmentation, the wool becoming a gingery-brown colour. However, as black sheep age their fleece often becomes a gingery-brown to grey colour so that differentiation of the normal from the deficient is difficult.

Loss of crimp has been reported mainly in Merino sheep in Australia, but rarely in New Zealand. Colloquially it was called steely wool because of its reduced lustre.

(iv)   Fertility (ewes)

Copper deficiency has been associated with infertility in ewes, but mainly on an experimental basis. The feeding of high levels of Mo and sulphur were thought to have increased foetal loss and a decrease in the number of lambs born. However ewes must be exceedingly Cu deficient before fertility suffers.

(v)   Fertility (rams)

After a year living on an experimentally induced Cu deficiency South African Mutton Merino rams produced semen ejaculates of low volume, low sperm concentration, poor sperm motility and morphology. Histologically the testes of such rams showed poor development of the seminiferous tubules and sertoli cells which were also inactive. Such changes were reversible when the Cu deficiency was corrected.

Diagnosis of Cu Status

When the clinical signs of hypocuprosis appear, Cu reserves in the animal are very severely depleted and it is likely also that other members of the herd or flock are in a precarious health position. Therefore veterinarians should be concerned with adequacy rather than deficiency. Further there is much clinical data which indicates clearly that animals which have low but adequate levels of blood Cu may within a very short time fall into the danger area of hypocuprosis. In other words levels of adequacy must not be taken too rigidly when interpreting diagnostic material, but must be used with full consideration of the health status of the flock, time of year and feed supplies available.

The schematic illustration of Suttle (1976) demonstrates the relationship between clinical signs and Cu depletion. It is the objective of good animal health advice to ensure that a herd or flock of valuable animals does not deplete the so called non-essential Cu pool putting animals at risk of clinical hypocuprosis.
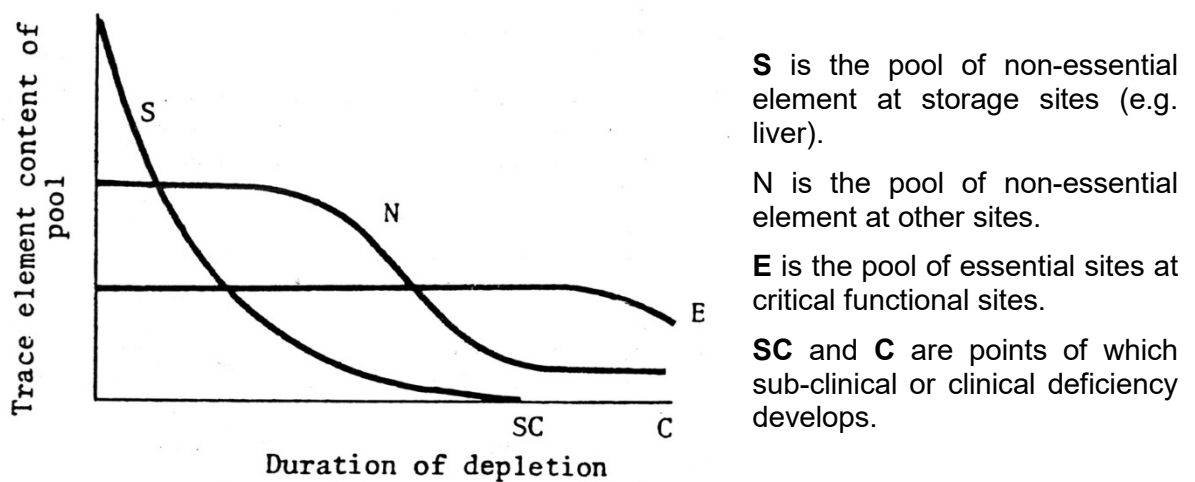
S is the pool of non-essential element at storage sites (e.g. liver).

N is the pool of non-essential element at other sites.

E is the pool of essential sites at critical functional sites.

SC and **C** are points of which sub-clinical or clinical deficiency develops.

Figure 5.17. Schematic illustration of relationships in the development of Cu deficiency (From Suttle, 1976).

It can be seen that the storage sites (S) are depleted first, followed by other non-essential sites (N). Only when these two pools are depleted do the amounts in essential sites decline and deficiency signs develop. Thus, examining either or both non-essential sites (blood or liver) does not estimate the essential sites, and hence the degree of deficiency.

It is therefore essential to follow the routine clinical procedure when attempting to determine Cu status of a farm and its animals and to consider all the factors involved, such as the following:

- age, species and breed of animals,
- feed type,
- concentrations of Cu, Mo, S, Fe in feed,
- the length of time animals have been on that feed,
- clinical features,
- previous Cu supplementation,
- tissue levels.

Liver and blood Cu measurement

When the supply of Cu from the diet declines, Cu stored in the liver is mobilised for use, and maintains blood levels. When liver levels become depleted blood levels also fall and clinical signs appear. Thus the measurement of liver Cu gives a direct and reasonably accurate means of diagnosing Cu status. It can also be used to predict the likelihood of supplementation being required in the next few months, given a knowledge of likely Cu nutrition. There can be considerable variation in the liver Cu levels between individuals within a herd and laboratories recommend that up to 16 liver samples are collected for analysis. However, cost becomes an issue if this number of samples is analysed so more commonly four or five samples are used to give an indication of whether levels are deficient, marginal or adequate. For more precise estimates, a larger number of samples are needed and more frequent sampling intervals may be required. As a generalisation, the within-herd variation is most marked when the mean level is in the mid-range, and reduces at low and high ranges.

While giving a useful measure of Cu reserves, liver analysis will not indicate whether the reserves are increasing, static or declining. Furthermore, liver reserves may fall from normal values to very low levels before plasma Cu declines. Also a severe Cu deficiency induced by excess Mo in the diet could cause signs such as diarrhoea before liver reserves are diminished if liver Cu cannot be mobilised fast enough.

Blood levels are more easily collected and are usually low at the time of deficiency. Either serum Cu or serum ferroxidase can be measured and these tests give similar information. In cattle and probably deer, measuring serum Cu from approximately 7-10 animals can confirm a diagnosis of Cu deficiency but is of little value in predicting the need for supplementation in the future if levels are normal. In sheep, blood Cu levels fall late in the development of a deficiency and while they are of value in controlled experiments, are considered too insensitive an indicator for field use, so that in sheep, liver Cu levels are the more appropriate indicator of Cu status.

As with other trace elements, the samples collected depend on the objective of testing, as illustrated by Table 5.16.

Table 5.16.   Sampling for Cu depending upon the reason for sampling (Clark and Ellison, 1993).

Reason	Time to sample	Species and age	Sample type	Sample number	Interpretation
Poor performance	Time of problem	Affected animals	Serum (or liver)	7-10 serum	Means only
Farm deficiencies	Late winter/spring	Cattle – late pregnancy/early lactation or fast growing cattle >6 months	Liver	At least 4, preferably more	Mainly means, take notice of individuals
Adequate reserves	Early winter	Cattle	Liver	At least 4, preferably more	Mainly means, take notice of individuals
Supplementation	Halfway through expected period of insufficiency	Animals being supplemented	Liver	At least 4, preferably more	Compare individual animals with previous samples

It must be remembered that Cu deficiency is a dynamic condition within a flock or herd and at the time of sampling the status of individuals within the group may be improving or declining depending on recent diet. Therefore in checking with reference ranges (Table 5.17) the clinician should be mindful of the appended note that these levels refer to deficiency risk and not to a projected adequate level for the maintenance of good health. This latter is a very important point and cannot be overemphasised.

Table 5.17.   Reference range for Cu levels in sheep and cattle.

		Low	Marginal	Adequate
Cattle	Liver μmol/kg*	<45	45-95	>95
	Serum μmol/l*	<4.5	4.5-8.0	>8.0
	Ferroxidase (U/l)	<7	7-14	>14
Sheep	Liver μmol/kg	<65	65-300	>300

* Cu levels have been measured in New Zealand diagnostic laboratories in standard units since 1982. Unfortunately the majority of international literature still records levels as weight, e.g. mg/kg DM or ppm (parts per million). To convert weight units mg/kg to molecular units x by 15.7. Conversely ÷ by 15.7.

Note: These levels are guidelines for the diagnosis of deficiency and not for the need for supplementation. For instance, it is recommended that to ensure adequate Cu status, breeding cows should have liver Cu levels over 300 μmol/kg at the onset of winter.

There is limited information on which to develop reference values for deer although it has been suggested by Wilson and Grace (2001) that for this species liver levels <60μmol/kg or serum levels <5 μmol/l are considered "deficient" and liver levels of 60-100μmol/kg are considered "marginal".

Feed Analysis

The concentration of Cu in animal feeds is only of limited value in assessing the necessity of Cu supplementation of the animal. This is because most Cu deficiencies are caused by the interaction of other elements such as Mo, Fe or S and are not related to the actual levels of Cu in the plant or soil. However, if Cu deficiency occurs in stock, feed analysis may help in determining why the deficiency has occurred.

Treatment and Prevention of Cu Deficiency

Clearly, the number of complicating factors affecting Cu uptake and usage by animals demands on a wide knowledge of the Cu status not only of the flock or herd, but also the farm and its pasture before Cu therapy is instituted. Unfortunately, in many cases the approach to Cu therapy on many New Zealand properties can at best be described as empirical and has not been based on sound analytical facts which are, in most cases, easy to obtain.

Oral administration of Cu

(i)   Drenching

Where antagonistic factors are low, oral drenching with Cu sulphate or Cu oxide may supply adequate Cu levels for some weeks but the traditional recommendation has been the weekly administration of Cu. Drenching at this frequency is impractical for sheep and may be practical for dairy cattle where the daily Cu requirements may not be easily met from feed or other means of supplementation. Anthelmintics with mineral additives do not contain sufficient Cu for effective supplementation.

(ii)   Cuised salt licks and Cu added to drinking water

Cuised salt licks under nearly all circumstances should be discouraged as a source of Cu for grazing animals. Some people have prepared their own salt licks with dangerously high levels of Cu salts (5%). Further the grazier has no control of the dose of Cu ingested by individual animals and some sheep have an affinity for licks and ingest a toxic dose of Cu quite readily. Similar comments may be made about Cu in drinking water unless it is controlled by a dispensing device which delivers an accurate amount of Cu to a given volume of drinking water. The latter are used successfully in some areas of New Zealand, particularly for dairy cattle.

(iii)   Cu oxide capsules (needles)

Copper oxide capsules are small particles of Cu oxide wire that are designed to lodge in the abomasal region and provide a source of Cu for months and possibly up to a year. Data available for both cattle and deer support that this form of therapy may effectively raise Cu storage levels in animals for 5-12 months depending upon the species and the circumstances in which they are used.

(iv)   Slow release rumen bullets

Soluble glass bullets have been used successfully experimentally but are not commercially available in New Zealand. A balanced trace element sustained release bolus (All-trace; Elanco Animal Health) is available which provides eight months supply of Cu, Co, Se, Mn, Zn, I, S and vitamins A, D, E.

(v)   Topdressing

Topdressing of pastures with 5-10 kg of $CuSO_4.5H_2O$/ha (1.1 to 2.2 kg Cu/ha) has been a traditional method of preventing Cu deficiency. This rate applied at three to four year's interval will ensure an adequate Cu intake for the grazing animal. However, cost may be a limiting factor on more extensive areas. The Cu is usually applied in conjunction with superphosphate and should not be grazed for at least three weeks or until heavy rain has washed all traces of fertiliser away in case Cu poisoning occurs. For animal supplementation purposes, Cu is normally applied annually.

The length of time that Cu is available following topdressing is variable. It is difficult to increase the Cu content of herbage since most plants have mechanisms to prevent the uptake of heavy metals. Suttle (1981) claimed that Cu topdressing was generally disappointing and the effect on pasture is largely gone in six months. Experiments have been undertaken grazing bulls (West et al, 1997) and deer (Grace et al, 2001) on Cu topdressed pasture. For the first 100-200 days the liver Cu levels of animals grazed on topdressed pasture declined in a similar way to those of control animals, but levels then slowly increased and were significantly different from controls to day 300-350. The reason for this apparent delay between topdressing and increasing liver Cu levels is unknown. More research is required to evaluate the use of Cu topdressing as an animal supplement for Cu deficiency.

Where low Cu levels occur in animals, caused by induced Cu deficiency (e.g. high Mo and/or S) then directly supplementing the animal may be more effective than Cu topdressing. Plant analysis for Cu, together with other interfering elements, will be necessary in deciding the benefits of Cu topdressing in such situations. The possibility of topdressing hay paddocks, particularly lucerne, from which hay can be fed out during the next winter has not been explored but may be a useful recommendation.

Parenteral administration

There are a variety of products that have been developed for the parenteral treatment of Cu deficiency. Apart from economic factors the parenteral administration of Cu is therapeutically sound since it bypasses the inhibition of Cu absorption in the gut. Its main disadvantages are that at a given injection only a limited amount of Cu can be given, because of toxic implications which have been recorded regularly in New Zealand. Injection site lesions may occur.

a)   Copper calcium edetate

This is an injectable form of Cu based on ethylene'diamine tetra-acetic acid (EDTA). This is a chelating agent which enables it to form a very stable ring structure with metallic ions. In association with Cu it is readily absorbed from the injection site thus minimising the risk of local reactions. It also allows non-ionised Cu to circulate which under normal circumstances makes it non-toxic. There is usually a small local reaction at the site of injection which quickly subsides.

b)   Cu glycinate

This is usually a depot formulation which ensures slow release of Cu into the circulation and uptake by the liver. Products contain 60 mg elemental Cu per ml. These products should only be given subcutaneously and care must be taken to ensure intramuscular injection does not occur.

Precautions With Cu Therapy

Death through overdosing with Cu is distinct from chronic Cu poisoning and does not usually involve a haemolytic crisis. The lesions of acute liver damage are characteristic with speckled dark areas in a usually pale liver. Subendocardial and epicardial haemorrhages and accumulation of fluid in serous cavities are also seen. Liver Cu levels are not markedly elevated.

In general, sheep are more susceptible to Cu toxicity than cattle, and there is often little difference between normal blood levels of Cu and those associated with toxicity. The situation of parenteral administration of Cu to sheep is also exacerbated by the fact that the transplacental transfer of Cu to the foetus by the ewe is poor thus, in order to give sufficient Cu to the foetus, the dose given to the ewe has a small therapeutic index. It must also be borne in mind that there is generally a great deal of variation in bodyweight within a flock of ewes, all being given a standard dose of Cu. Superimposed upon this are the genetic differences in sheep and their ability to metabolise Cu.

The warnings associated with injectable Cu use should be clearly stated to the user and include:

- Confirmation of a deficient Cu status before administration.
- Take care when administering Cu to young and debilitated animals, particularly where liver damage is suspected.
- The simultaneous administration of other compounds is contraindicated.
- Injections must be subcutaneous.
- Avoid prolonged yarding periods and other stress.

Frequency of Cu Therapy

The frequency of Cu administration cannot be accurately predicted for each situation. As we have seen, many factors affect the ultimate absorption and retention of Cu by individual animals and the degree of deficiency varies markedly from farm to farm and at different times of the year. The general approach is therefore somewhat empirical but nevertheless should be based on regular monitoring to assess the adequacy of therapy on a particular farm.

The following is given as a guideline and should be used with caution.

For sheep, one annual dose of Cu may be adequate to control Cu deficiency. Injecting prior to mating should be sufficient to reduce the incidence of swayback and prevent spontaneous bone fracture.

In beef cattle it is generally suggested that three doses of Cu are needed in young animals and thereafter twice yearly dosing. Frequency of dosing may have to be increased in adults if they show signs of Cu deficiency or if liver biopsy samples reveal very low levels. In dairy cattle, Cu requirements are usually higher and more regular supplementation may be needed.

Molybdenum levels in pasture are highest and Cu levels lowest during late winter/early spring. Therefore monitoring Cu reserves before winter is useful in predicting if supplementation is required.

Mo and Cu Deficiency

In many parts of New Zealand the excessive use of Mo on pastures has precipitated a Cu deficiency problem in animals grazing these pastures. On some farms in the past molybdenised superphosphate was used as frequently as once a year and at very high levels. Trials conducted at Wallaceville research station showed that the regular application of molybdate each year to light stoney soils raised pasture concentrations of Mo to dangerous levels, which rose even higher during spring. These coincided with clinical signs of lameness and loss of mobility in young stock grazing the pastures. It was concluded that there are few soils in New Zealand that would require amounts of molybdate in excess of 200 g/ha every 4 to 5 years.

Cu Deficiency in Cattle and not Sheep on the Same Property

In Table 5.18 a number of properties are shown where Cu deficiency has been clinically diagnosed and confirmed in cattle but not in sheep. Analysis of liver material from six of these properties demonstrated that a serious deficiency of Cu may have existed in some cattle while sheep on the same property had adequate Cu stores which were unlikely to be depleted. These observations should not necessarily be taken to infer that the clinical signs of Cu deficiency would always appear in cattle before sheep. On some properties cattle have apparently thrived, while young sheep showed swayback and osteoporosis.

Table 5.18. Liver Cu levels (μmol/kg) in cattle and sheep on the same property.

Property	No.	Mean	Range
A	10 cattle 10 sheep	91.8 476.0	38.0 - 282.6 94.2 - 1884.0
B	5 cattle 3 sheep	56.5 1051.9	18.8 - 152.0 736.0 - 1381.6
C	5 cattle 3 sheep	251.2 1476.0	12.5 - 540.0 1256.0 - 1876.0
D	7 cattle 6 sheep	252.0 845.0	31.4 - 262.0 628.0 - 1099.0
E	3 cattle 6 sheep	105.0 1868.0	26.7 - 177.0 785.0 - 3768.0
F	6 cattle 3 sheep	75.36 1582.6	28.2 - 208.8 850.9 - 2088.0
Total	Cattle Sheep	114.6 1213.6	7.8 - 540.0 204.0 - 3768.0

Conclusion

In any animal health investigation or programme the monitoring of the Cu status of a property should be a routine procedure. The application of history clinical signs and the chemical analysis of animal tissues and blood are essential. Pasture samples from several areas of the property should also be monitored for Cu, Mo, S and Fe and sometimes levels of other elements such as Zn and Mn. This full diagnostic routine is required before sound professional advice can be given on the use of any of the several forms of Cu therapy available.

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"	(1978): 4: 22	Paresis in deer.
"	(1978): 5: 19	Copper deficiency and ovine enzootic ataxia.
"	(1978): 5: 22	Enzootic ataxia in deer.
"	(1980): 5: 10-11	Hypocupraemia: what does it mean.
"	(1981): 1: 11	Parasitically induced copper deficiency in cattle.
"	(1975): 4: 19	Low copper and sudden death.
"	(1975): 3: 9	Low copper status of Northland cattle.
"	(1977): 1: 14	Copper deficiency.
"	(1977): 4: 22	Falling disease.
"	(1977): 5: 24	Follow up on suspect falling disease.
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5.4 Iodine (I)

Introduction

The occurrence of goitre in lambs and calves in New Zealand was first noted by Gilruth in 1901. However it was not until 1925 that I deficiency was documented as the cause of goitre in human and animal populations in this country. The latter discovery led to the widespread use of dietary iodised salt in the 1930s and a consequent decline in the prevalence of goitre in humans.

Although a dietary deficiency of I has commonly been regarded as a cause of goitre, goitrogens that occur in many animal fodders (particularly brassica crops) are also an important cause of the disease. In sheep, congenital goitre occurs when the I concentration of herbage falls into the range 0.09 - 0.18 mg/kg DM. Goats appear most susceptible to I deficiency, followed by sheep. Occasional cases of goitre have been reported in calves.

Metabolism and Function of I

In adult sheep I is effectively trapped in the thyroid gland, which contains approximately 80% of the body's I. These reserves may give adult sheep sufficient I for adequate thyroid hormone secretion for many months after I intake decreases. This situation is different in the developing foetal lamb. The lamb's thyroid gland develops independently from that of the ewe and production of adequate levels of thyroid hormone for foetal development and maturation depends on sufficient amounts of I, derived from its mother's daily intake, being transported across the placenta. Therefore the lamb cannot use its mother's reserves and for this reason subclinical I deficiency usually affects lamb health and production.

The only known role of I in animals is in the synthesis of the thyroid hormones, thyroxine (T4) and tri-iodothyronine (T3) in the thyroid gland. Thyroid hormones are important for energy metabolism and protein synthesis of cells. Tri-iodothyronine stimulates the production of enzymes involved in energy metabolism and is thus closely related to heat production and oxygen usage. Thyroid hormones are essential for the development of foetal brain, lungs, heart, and wool follicles. Along with cortisol the thyroid hormones are important in stimulating the maturation of the cells lining the lung alveoli and can stimulate the production of surfactant, which is important in lung maturation and necessary for the survival of the newborn lamb.

In ruminants, I is mainly absorbed in the rumen. Some endogenous I is secreted into the abomasum but is reabsorbed in the small intestine. The ingestion of I is not wasteful, as with many other elements.

Dietary I, as well as I produced by degradation of thyroid hormones, is trapped in the thyroid gland where it combines with tyrosine to form a range of iodinated amino acids. The main one is thyroxine which is stored, in combination with protein, as thyroglobulin. Iodine is released from the thyroglobulin in the thyroid gland as thyroxine which is bound to globulin, prealbumin and albumin and as such is transported in the blood. Typical plasma concentrations of thyroxine in adult and foetal sheep are 40 to 70 and 100 to 120 mmol/l respectively.

Thyroxine is reduced to tri-iodothyronine which is the active form of the hormone chiefly in the thyroid and pituitary glands, liver and kidneys. Tri-iodothyronine is almost non-detectable

in foetal blood while in normal healthy sheep plasma tri-iodothyronine levels are 1.8 to 2.2 mmol/l. During tissue metabolism tri-iodothyronine is degraded to inorganic I most of which is again trapped by the thyroid gland. A small amount is excreted in the urine.

The Requirements for I

The presence of goitrogens in feed is a major factor affecting I requirements. Only one of the two types of goitrogens (thiouracil-type and thyiocyanate-type) has been detected in vegetation in New Zealand. The thiocyanate-type goitrogens found in New Zealand vegetation blocks the uptake of inorganic iodide by the thyroid gland and hence reduced thyroxine synthesis. The thiouracil-type goitrogens found overseas in tropical legumes block the conversion of thyroxine to tri-iodothyronine. Plants of the *Brassica* spp which includes the forage kales, cabbages, brussel sprouts and broccoli contain glucosinolates which are broken down during cudding to form inorganic thiocyanate ions. Swedes and turnips do not produce thiocyanate to any significant extent, but the forage sorghums yield hydrocyanic acid on chewing which combines with sulphur in the rumen to produce thiocyanate. New Zealand cultivars of clover also contain high levels of thiocyanate, as this confers resistance to slug predation,

Fortunately the goitrogenic properties of inorganic thiocyanate can readily be prevented by I supplementation. The thiocyanate ion can also readily cross the placenta as can inorganic iodide, hence I supplementation of pregnant ewes consuming a goitrogenic diet is especially important to ensure that the developing foetus, which is particularly vulnerable to I deficiency, obtains adequate levels of I.

The ingestion of soil caused by high stocking rates is said to reduce the incidence of goitre. However as a practical point, the effects of overstocking can be deleterious in other ways so that this does not really offer a realistic alternative to the prevention of goitre. Similarly, anecdotal information suggests that I deficiency is more common during dry seasons and this may be related to lower levels of soil intake compared with wet seasons.

Iodine requirements are increased in high producing animals where energy intake, heat production and protein synthesis are all high. This includes pregnant ewes, lactating ewes and cows and rapidly growing animals.

Occurrence of I Deficiency

Iodine deficiency may occur in high rainfall areas such as in Westland or inland areas such as Otago, Canterbury and Manawatu. In New Zealand most occurrences of goitre seem to have been associated with goitrogens in feed.

While the goitre producing areas of New Zealand have been identified for sheep in the past, the occurrence of goitre in goat kids on farms where sheep have been run without ever having exhibited signs of goitre suggests that goats may be more susceptible to I deficiency. The main goitre producing areas in New Zealand are shown in Figure 5.18. However the possibility of subclinical I deficiency occurring outside of these areas cannot be discounted.

Figure 5.18.   Goitre producing areas in New Zealand.

The I concentration has been found to be much higher in perennial ryegrass and white clover than in browntop and the *Poa* spp.   Seasonal trends are also likely, with the highest concentrations occurring in the improved species during winter, corresponding to their lowest rates of DM production.   In contrast, highest concentrations in the unimproved species are seen during spring and summer.   Table 5.19.

The content of I in forage kale is consistently lower than that of ryegrass/clover pasture sampled over autumn and winter.   Hence kale is not only goitrogenic but can be I deficient as well.   Within each type of pasture, considerable variation occurs from year to year which is reflected in the severity and incidence of neonatal goitre of lambs born of ewes grazing such pasture.   Plant concentrations of I are affected by the locality in which they are grown.   For example in one series of data the I concentration in ryegrass/clover grown on Manawatu hillcountry was 5-7 times higher than equivalent pasture grown at Invermay.

Table 5.19: Mean I concentrations (mg/kg DM) in pasture species sampled at three monthly intervals (Manawatu hillcountry). (Johnson and Butler, 1957).

Pasture Species	Season				Mean Annual Concentration
	Autumn	Winter	Spring	Summer	
Perennial ryegrass	1.35	1.86	1.30	1.30	1.45
White clover	0.77	1.23	0.80	0.83	0.91
Browntop	0.19	0.18	0.22	0.30	0.22
Poa annula	0.08	0.09	0.08	0.19	0.11
Poa trivialis	0.09	0.08	0.28	0.19	0.16

Clinical Findings

Enlargement of the thyroid glands of lambs (goitre) is the most obvious sign of I deficiency and is seen more commonly when the ewe has eaten either I deficient or goitrogenic fodder during the last half of pregnancy. In addition, neonatal mortality may be severely increased and has been reported as high as 60% where ewes have been grazed on kale. Gestation lengths may be slightly reduced (3-7 days) and in severe cases lambs are born with little wool and pink bare skin. Lack of I may also impair brain development and reduce lung development and maturation. These factors contribute to making the weak newborn lamb highly susceptible to cold stress.

Subclinical I deficiency is associated with an increased perinatal lamb mortality rate (see also Chapter 5). In some experiments I supplementation resulted in an increase in mean litter sizes in treated compared with untreated ewes. It has been suggested that small depressions in wool production and milk production may also occur. There is one report of a weight gain response in hoggets on improved native tussock in the South Island.

Figure 5.19.   Goitre in lambs. Note the swollen thyroid glands of the neck.

Figure 5.20.   A weak lamb with goitre and unlikely to survive.

Diagnosis

In comparison with the other trace elements, the I status of ruminants is perhaps the most difficult to define for the practising veterinarian.

Clinical goitre may be detected by palpation of the thyroid gland, by determining the thyroid to bodyweight ratio, and by histopathology. Goitrous lambs usually have a thyroid to bodyweight ratio greater than 0.7 g/kg or a total weight of more than 2 g for both thyroid glands. In many cases the goitre is obvious, but mild individual cases may be harder to detect in the live lamb. A mean thyroid to bodyweight ratio of greater than 0.4 g/kg from necropsy examination of 10-20 newborn lambs, in conjunction with a high perinatal mortality rate, is considered highly suggestive of subclinical I deficiency. Both thyroid glands should be carefully dissected and weighed accurately and the weight in grams compared with the weight of the lamb in kilograms. Histologically, thyroids from goitrous animals have follicles depleted of colloid and lined with columnar epithelium.

Determination of serum or plasma thyroxine and tri-iodothyronine concentrations can be undertaken but there are no reference ranges for these values and their use is limited. It has been suggested that a better assessment of I status is obtained from measuring milk I concentration. Milk I concentration less than 80 µg/l indicate inadequate I intake by the ewe. Milk I concentrations are sensitive to daily changes in dietary intake, and this may limit the usefulness of single milk samples to assess I intake. Another limitation is due to the critical period being before the lamb is born and the ewe is lactating. Serum I concentrations have been used to assess the effects of I supplementation of ewes. Goitre did not occur in lambs when the mean I concentration of the ewes exceeded 43 µg/l but did occur when the concentrations fell below 30 µg/l.

Herbage I levels are of limited use. However Morton and Roberts (1999) suggest that mixed pasture herbage I concentration of at least 0.25 ppm is required to provide adequate I for young sheep and this concentration is raised to 2 ppm if feed contains goitrogens (e.g. Forage kales, other brassicas).

Treatment

Some affected lambs, kids and calves with moderate to severe goitre will survive if treated with I, either as a drench of potassium iodide (20 mg per lamb) or injected with iodised oil preparation (Flexidine; Bomac Laboratories Ltd).

Prevention

Iodine deficiencies are overcome by direct treatment of the livestock rather than by applying iodine-containing fertilisers to the soil. The routine procedure to prevent I deficiency in ewes grazing goitrogenic or I deficient pasture is either:

(i) inject ewes with iodised oil (Flexidine) given by intramuscular injection. Doses of 1.5 ml are required for ewes and does. This should be given one month before mating or not less than two months before the start of lambing. It is recommended to treat stock at least two months prior to the feeding of goitrogenic crops. This product provides a long-term depot of I in the muscle which is slowly released. Annual re-treatment is recommended.

(ii) to prevent goitre in lambs, drench ewes with 280 mg potassium iodide eight and four weeks prior to parturition. Given that some experiments have found that treated ewes have an increased mean litter size, it may also be prudent to dose ewes prior to mating.

Although I supplements have been incorporated in the past into licks, intake is variable and there is extensive loss of I by oxidation, volatilisation and leaching.

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5.5 Molybdenum (Mo)

Introduction

The importance of Mo is due to it being an essential element for plant growth, especially clover and lucerne because of its role in nitrogen fixation. In New Zealand, Mo deficiency is not a problem in sheep and cattle but excessive Mo intakes can be. High Mo intakes by grazing livestock in the presence of adequate levels of S markedly reduce the absorption of Cu and thereby can induce Cu deficiency. Molybdenosis is the disease in cattle caused by an imbalance of Mo and Cu in the diet and when the Mo content of the forage is greater than 5 ppm. Molybdenum toxicity causes stunted growth and bone deformation in the animal and can be corrected by oral feeding of Cu. Other practices used to decrease Mo toxicity are application of S or Mn, and improvement of soil drainage.

Mo in Soil

The total Mo content of soils is usually in the range of 0.07 to 5 ppm. The main forms of Mo in soil include: non-exchangeable Mo in primary and secondary minerals, exchangeable Mo held by Fe/Al oxides, Mo in the soil solution and organically bound Mo. In soil solutions above pH 4.3 the predominant form of Mo occurs as MoO_4^{2-}.

Factors affecting Mo availability

Soil pH: concentration of MoO_4^{2-} increases dramatically with increasing soil pH. Soil Mo availability to plants increases with soil pH, and deficiencies are most common in acid soils, specially in soils with pH values less than 5.5. Liming can sometimes overcome Mo deficiency, but some soils have such low values of Mo that Mo fertiliser must be applied.

Adsorption to Fe/Al oxides: Mo is strongly absorbed by Fe/Al oxides, a portion of which becomes unavailable to the plant. Soils that are high in amorphous Fe/Al oxides tend to be low in plant-available Mo.

Interactions with other nutrients: P and Mg enhance Mo absorption by plants. In contrast high levels of SO_4^{2-}, Cu, and Mn in solution depress Mo uptake by plants. Nitrate N encourages Mo uptake, while NH_4^+ sources reduce Mo uptake.

Climatic effects: Mo deficiency is more severe under dry soil conditions, probably due to reduced mass flow or diffusion under low soil moisture content.

Mo in Plants

Plants absorb Mo as molybdate (MoO_4^{2-}). The importance of Mo to plants is due mainly to its presence in two enzymes, nitrogenase and nitrate reductase. Nitrogenase is involved in the basic mechanism for atmospheric nitrogen fixation by rhizobium, and nitrate reductase controls the first stage in the conversion of nitrate-N to amino acid and protein-N.

The Mo content of pasture can vary from about 0.2 to over 10 mg Mo/kg DM depending on factors such as soil pH, season, pasture species and amount of Mo applied as fertiliser.

In New Zealand, Mo deficiency occurs in both clovers and lucerne. Other legumes, peas and beans, are also susceptible to Mo deficiency, as are some of the brassicas, especially cauliflower and broccoli.

Table 5.20.   Sensitivity of crops to low levels of soil available Mo

High sensitivity	Mild sensitivity	Low sensitivity
Clover	Lettuce	Ryegrass
Lucerne	Potatoes	Corn
Rapeseed	Soybeans	Wheat
Peas		Oats

Mo in Animals

Requirements for Mo

The Mo requirements of sheep and cattle are not clearly defined. Signs of Mo deficiency such as reduced feed intake, poorer weight gains, a higher mortality at birth and a shorter life expectancy were observed when goats were fed a diet containing 0.06 mg Mo/kg DM (Ankle *et al.*, 1977). However, in another study where an experimental diet was fed a significant growth response and an improved cellulose digestibility was reported in sheep when their basal diet containing 0.36 mg Mo/kg DM was supplemented with 2 mg Mo/kg DM (Ellis *et al.*, 1958). It appears therefore that the Mo requirements for ruminants are very low. Under New Zealand conditions a Mo deficiency is most unlikely. The problem is usually an excess of Mo that increases the Cu requirements of sheep and cattle (Suttle and McLauchlan, 1976).

Effects of excess Mo intakes

An increase in the Mo content of the pasture can cause stock health problems. The Mo content of the pasture can increase several folds during late winter and early spring, particularly on peat soils. The high Mo intake in the presence of adequate levels of S markedly reduces the absorption of Cu and thereby can induce Cu deficiency in grazing livestock. Mo alone has little influence.

The sign of an excess of Mo or molybdenosis really reflect an induced Cu deficiency. Molybdenosis is characterised by a decrease in liver Cu levels and in many cases decreased blood Cu levels in sheep and cattle. More recently in dairy cows a condition termed post parturient haemoglobinuria has been found to be associated with excessive intakes of Mo.

Mo Fertilisation

On some soil types applying Mo at the rate of 50 to 75 g/ha (as sodium molybdate, 40% Mo) is needed to stimulate the growth of the clovers and thereby increase the DM production of the pasture. This rate should be applied every 4-5 years when both clover Mo and N are deficient (Mo <0.1 ppm, N <4.5%). Also, when applying molybdenised fertilisers the Mo and Cu levels of the topdressed pasture should be checked to ensure that the Mo levels are not becoming too high in relation to the pasture Cu levels.

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5.6 Manganese (Mn)

Introduction

Manganese is essential for many important plant functions, including photosynthesis, nitrogen metabolism and nitrogen assimilation. Also, Mn is an important co-factor for many enzymes with a wide range of activities in animals. Manganese deficiencies in livestock are virtually unknown either in New Zealand or overseas. In fact and excess of Mn could be a problem in some areas. Growth rates of young lambs have been observed to be depressed by excessive Mn intakes equivalent to grazing pastures containing over 400 mg Mn/kg DM.

Mn in Soil

Mn exists as solution Mn^{2+}, exchangeable Mn^{2+}, organically bound Mn, and various Mn minerals. Examples of Mn minerals are pyrolusite (MnO_2); manganite ($MnOOH$); and hausmannite (Mn_3O_4).

Factors influencing the solubility of soil Mn

Soil pH: liming very acidic soils decreases solution and exchangeable Mn^{2+}, due to Mn^{2+} precipitation as MnO_2. High pH also favours the formation of less available organic complexes of Mn.

Excessive water and poor aeration: soil water logging will reduce O_2 and lower redox potential, which increase soluble Mn^{2+}, specifically in acidic soils.

Organic matter: the low availability of Mn in high-OM soils is attributed to the formation of unavailable chelated Mn compounds.

Interactions with other nutrients: high levels of Cu, Fe, or Zn can reduce Mn uptake by plants. Addition of NH_4^+ to soil will enhance Mn uptake.

Climatic effects: wet weather favours the presence of Mn^{2+}, whereas warm, dry conditions encourage the formation of less available oxidized forms of Mn.

Most New Zealand soils contain substantial amounts of Mn, and deficiencies in both agricultural and horticultural crops are due to low Mn availability in soils with high pH values (6.5 and above). In acid soils, high availability of soil Mn can give rise to Mn toxicity in plants. However, because of its mobility, Mn^{2+} can leach from soils, particularly from acidic podzols.

Mn in Plants

Plants absorb Mn as Mn^{2+} as well as in molecular combinations with certain natural and synthetic complexing agents which moves to the root surface by diffusion of principally chelated Mn. Manganese is essential for many important plant functions, including photosynthesis, nitrogen metabolism and nitrogen assimilation. It is also involved in oxidation-reduction processes and is required for the activation of several enzymes, including decarboxylase and dehydrogenase. Manganese can substitute for Mg in many of the phosphorylating and group-transfer reactions. Manganese is a relatively immobile element, and deficiency symptoms usually show up first in the younger leaves.

Deficiencies can affect wheat, barley, beans and potatoes, but in New Zealand deficiencies in pasture are unknown. The Mn content of pasture can vary from about 50 to over 500 mg Mn/kg DM with grasses usually having lower levels than clover. The soil pH strongly influences the Mn levels in plants with the Mn uptake being greatest in acid soils. Also, under conditions of poor drainage the reducing conditions in the soil favour the formation of more mobile forms of Mn and cause a greater increase in the accumulation of Mn in plants.

Table 5.21. Sensitivity of crops to low levels of soil available Mn

High sensitivity	Mild sensitivity	Low sensitivity
Lucerne	Barley	Cotton
Wheat	Corn	Fruit trees
Oats	Rice	Vegetables
Potatoes	Soybeans	

Mn in Animals

Functions of Mn

Manganese is an important co-factor for many enzymes with a wide range of activities. Mn containing enzymes are important for:

(a) the synthesis of mucopolysaccharides which make up the organic matrix in bone and teeth (Leach *et al.*, 1969).

(b) The synthesis of cholesterol that is the precursor of the steroid hormones.

(c) Gluconeogenesis, that is the synthesis of glucose from precursors such as amino acids (Mildvan *et al.*, 1966)

(d) Glucose utilisation.

Requirements for Mn

The Mn requirements for growth should be met by a diet containing 10 mg Mn/kg DM, but for optimum skeletal development and to prevent reproductive problems the dietary levels should be increased to 20 or 25 mg Mn/kg DM (Agricultural Research Council, 1980).

Signs of Mn Deficiency

Lambs and calves fed Mn deficient diets for 7 and 16 weeks respectively developed stiff gaits attributed to joint abnormalities and bone deformities. Low dietary Mn levels (7 to 17 mg Mn/kg DM) also cause reproductive problems such as delayed or irregular oestrus and poor conception rates in cattle. Normal reproductive function was observed on diets containing 21 to 25 mg Mn/DM kg (Rojas *et al.,* 1965).

Effect of Excess Mn Intakes

The significance of excessive Mn intakes on animal performance has not yet been fully documented. However, no ill effects were noted in weaned calves fed diets containing 820 mg Mn/kg DM, although diets containing 2600 mg Mn/kg DM significantly reduced their feed intakes and growth rate.

Mn Fertilisation

Manganese sulphate ($MnSO_4$ with 26-28% Mn) is widely used for correction of Mn deficiency and may be soil or foliar applied.

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5.7 Calcium (Ca)

Introduction

Calcium is a macronutrient and is essential for both plant and animal growth. Calcium deficiency is rarely seen in pasture or field crops but is occasionally observed in fruit crops and livestock. Calcium deficiency in livestock causes a disorder called hypocalcaemia or milk fever.

Soil Ca

Calcium is the fifth most abundant element in the earth crust (approximately 3.64%) and its concentration in most acidic soils ranges from 0.1 to 5%. Some calcareous soils contain more than 20% Ca. Calcium is present in three major forms in soils: in solution, on exchangeable sites, and in minerals. Plants take up Ca from the solution and exchangeable forms in soils. Plant-availability of Ca depends primarily on the exchangeable Ca concentration and on the percent saturation of the exchange sites by Ca. Calcium is commonly added to soil as an accessory element in many fertilisers and liming materials and therefore Ca deficiency in soils is rare. In a review of Ca requirements of pastures in New Zealand, Edmeades and Perrot (2004) reported that the current Ca concentration in New Zealand topsoils are more than adequate for optimal pasture and animal production, and that this situation is sustainable based on Ca budgets given a continuance of the traditional fertiliser practices of super-phosphate and liming. However, increasing use of Ca-free ammonium phosphate fertilisers and reduced use of liming materials may result in increasing incidences of Ca deficiency in soils in the future.

Ca in Animals

Hypocalcaemia, milk fever

Clinical hypocalcaemia (milk fever) in pregnant ewes occurs when there is insufficient intake and absorption of Ca, and insufficient resorption from skeletal reserves to meet foetal demands. Up to 2.8g of Ca per day is required to meet foetal needs and hypocalcaemia is usually associated with plasma Ca levels below 1.7 mmol/litre. It is often complicated by low blood Mg and low serum phosphorus levels. It occurs mainly in mature ewes during late pregnancy but occasionally occurs after lambing and has been reported in dry sheep as well.

As in pregnancy toxaemia the losses due to death from hypocalcaemia and overt milk fever are important, but even more important are the production losses which may arise from ewes which have recovered from hypocalcaemia or those which have had subclinical hypocalcaemia. The lambs born from such ewes have a poorer chance of survival as was shown in one study where 22% of lambs died which were born from ewes which were hypocalcaemic during late pregnancy. In the normocalcaemic ewes of the same trial only 3% of lambs died.

Ca Requirements of the Pregnant Ewe

Two important predisposing factors are necessary before hypocalcaemia occurs. These are the high requirement for Ca by the pregnant ewe and a low dietary intake over a period of weeks or months. Normal circulating serum Ca levels are reported to be 2.0-3.0 mmol/litre and in cases of hypocalcaemia, levels as low as 0.80 mmol/1 may occur. Dietary Ca levels should be 1.5-2.6g Ca/kg dry matter.

Irrespective of Ca intake, ewes do not absorb sufficient dietary Ca to meet their requirements during late pregnancy, and for milk secretion in early lactation. Ewes may mobilise up to 20% of their skeletal Ca reserves during this time, with the proportion mobilised depending on the pre-existing degree of mineralisation, and on the Ca content of the diet. Replacement of lost skeletal reserves is usually complete by 130 days after parturition on diets providing plentiful Ca (250 mg Ca/kg liveweight/day) and phosphorus (160 mg P/kg liveweight/day). Because the rate of accretion of Ca on to bone decreases with age, the replenishment of the partly depleted skeleton will take longer in older ewes. If skeletal reserves are not replaced adequately, ewes may become susceptible to hypocalcaemia during subsequent pregnancies.

Calcium deficiency can occur in sheep on a variety of diets. Under drought conditions, sheep fed wheat or concentrate diets (<1.0g Ca/kg DM) containing low Ca and high phosphorus may become Ca deficient. Further, low phosphorus intake associated with mature or dry summer grass may also limit repletion of skeletal reserves. Under wet conditions and when pasture is growing rapidly, the Ca level may be at its lowest. Hypocalcaemia frequently occurs in sheep grazing lush pasture and this has been seen in late winter in sheep fed on oat crops which have a very low Ca content.

Predisposing Factors

In many cases the factors which precipitate hypocalcaemia are similar to those associated with pregnancy toxaemia. Usually sudden changes in feed, either in feed type or the grazing regime, will cause short-term starvation and lead to clinical hypocalcaemia. Sudden increases in green feed or management procedures such as holding for crutching or shearing are all contributing factors. Extensive droving of sheep and access to sorrel (or other oxalate containing plants) have also been associated with hypocalcaemia.

Clinical Signs and Diagnosis

The diagnosis of hypocalcaemia is usually based on a history of a sudden feed check or change, the characteristic clinical signs and a rapid response to treatment. Serum Ca levels may also be assessed to confirm a diagnosis but this is seldom necessary. Blood samples should be collected from at least five different ewes.

Affected ewes are initially ataxic and hyperactive but rapidly become recumbent and comatose. There may be no corneal reflex. They are generally found in sternal recumbency with their head turned into the flank, but seldom go into lateral recumbency like cattle. Tympany usually occurs and regurgitation of rumen contents is often seen. In some cases prolapse of the vagina may also occur. Untreated animals usually go into a deep coma after 24 hours and die.

It should be noted that similar clinical features to hypocalcaemia are seen in ewes suffering from phosphatic fertiliser toxicity which is also more common in pregnant ewes. This is discussed in further detail later in this chapter.

Treatment

Many sheep with hypocalcaemia also have low blood Mg and low glucose levels so that treatment with Ca borogluconate, Mg sulphate and glucose solutions is desirable, although in most cases Ca borogluconate is used on its own.

Ready-made solutions of Ca borogluconate are available at concentrations of 25%, 37.5% and 50% w/v and depending on the concentration 30-100 ml should be given. The higher concentration solutions should be injected subcutaneously because if given intravenously they may cause cardiac arrest. The response of the ewe following treatment is usually quick and dramatic. They will usually get up within 15 to 30 minutes, urinate, show muscle tremor and walk away and feed. Sheep that do not respond quickly should be checked for pregnancy toxaemia as this may be a sequel to hypocalcaemia in ewes.

If a number of ewes are treated it is wise to go around those that have not risen of their own accord and try to gently prod them into movement.

Prevention

The prevention of hypocalcaemia largely depends on avoiding stressful conditions for ewes in late pregnancy or early lactation. Avoid unnecessary mustering and holding for long periods without feed. Do not initiate sudden changes in feed and introduce sheep to crops gradually over several days. Avoid droving for long periods and do not transport sheep which are heavily pregnant.

Under drought conditions, as seen in parts of Australia limestone as a Ca supplement should be given to sheep which are grain fed. Usually 1.5 per cent limestone is recommended (1.5 kg to 100 kg grain).

It is generally considered that, given the dry matter intakes are adequate, the Ca requirements for sheep and cattle are met by pastures containing 0.30 to 0.50% Ca in dry matter (concentrations considered optimum for pasture growth as well). The continual applications of superphosphate and lime should maintain these Ca levels in pasture in many farms.

Recently it has been shown that the different basic cations in pasture herbage influence the animal's relative absorption of Ca. For example, increasing intake of potassium has been identified as the major cause of the decrease in the absorption of Ca in grazing animals that results in milk fever. It has been shown that diets with high dietary cation-anion difference (DCAD = $Na^+ + K^+ - Cl^- - SO_4^{2-}$) values tend to increase the incidence of milk fever, and the supplementation of precalving rations with anionic salts (low DCAD values) reduces the incidence of milk fewer.

The manipulation of the cows' diet in terms of Ca and phosphorus intakes has been suggested as a way to lower the incidence of milk fever. The idea is that low intakes of Ca and phosphorus during the later part of pregnancy should stimulate the mobilisation of Ca and this together with the increase in Ca intake at calving should provide adequate amounts of Ca to meet the lactation demands the animals for Ca. This approach has limited application in the grazing situation but if supplementary feeding is being done then the manipulation of Ca and phosphorus intakes could be considered. For example, Lucerne and Lucerne products are high in Ca and should not be given 4 to 6 weeks before calving but maize silage, which is low in these elements, would be satisfactory as a supplement.

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5.8 Magnesium (Mg)

Introduction

Magnesium is a macronutrient and is essential for both plant and animals growth. Magnesium deficiency in livestock causes a disorder called hypomagnesaemia or grass tetany.

Soil Mg

Magnesium is the eighth most abundant element in the earth crust (approximately 2%). Soils generally contain between 0.1 and 1% Mg but not all of this is available to plants. Magnesium is found in three forms in soil: mineral Mg (very slowly available), exchangeable Mg (Mg^{2+}, readily available) and soil solution Mg (mostly Mg^{2+} readily available). Exchangeable Mg represents about 5% of the total soil Mg and usually occupies between 4 and 17% of the soil exchange capacity. Plant-availability of soil Mg depends primarily on the exchangeable Mg and K concentrations. High concentrations of exchangeable K reduces Mg uptake by plants.

Mg in Animals

Hypomagnesaemia, grass tetany

Hypomagnesaemia (grass tetany) is a disorder usually occurs in recently calved dairy and beef cows and less frequently in ewes pre- and post-lambing. The disorder is associated with a decrease in plasma Mg levels and is characterised by tetany and convulsions. Death occurs if the animals are not treated with Mg sulphate solutions. Sub-clinical hypomagnesaemia results in a lowering of milk yields. Hypomagnesaemia has been reported to be affecting approximately 30-50% of the dairy heards in the major dairying areas of New Zealand.

Mg Metabolism in the Ewe and Predisposing Factors

The absorption of Mg from the rumen is dependent on its concentration in the rumen fluid and is affected by other factors such as potassium and ammonia concentrations. If potassium concentrations in herbage increase (from 10 to 40 g/kg DM), and/or salivary secretions decrease, there is an increase in rumen potassium (from 5 to 40 nmol/l) and a decrease in rumen sodium (from 135 to 80 nmol/l). As a result active Mg absorption is decreased by the direct effects of high potassium on the Mg pump. High ammonium ion concentrations (30 nmol/l) also decrease Mg absorption and this effect is additive and independent of the effects of potassium. Simply changing the diet, from hay to lush pasture, which may not directly affect mineral uptake, can lead to similar effects because reduced mastication and salivary secretion increases the rumen K^+ to Na^+ ratio.

In pasture and fodder crops, a grass staggers index (GSI) ($K^+/(Ca^{2+} + Mg^{2+})$ where the ion concentrations are in milliequivalents per kg dry matter) is used to predict the chances for the occurrence of Mg deficiency. A GSI value of greater than 2.2 has been suggested to enhance the risk of grass staggers, which is linked to animal serum Mg levels less than 1.0-1.5 mg per

100 ml, compared with normal levels of 1.7-3.0 mg per 100 ml. GSI values increase with increasing levels of potassium addition. Application of Mg fertilisers is expected to decrease the GSI values. Irrigation of pasture with dairy shed effluents rich in potassium has been shown to increase the incidence of Mg deficiency.

There are virtually no available body reserves of Mg, so that animals are dependant on a continued dietary intake to meet their requirements. This intake is affected by a number of factors. There is good evidence to suggest that, prior to clinical hypomagnesaemia occurring, the ewe may already have a low blood Mg level. The situation is exacerbated, as the ewe is unable to quickly mobilise Mg reserves within her body. Seventy five percent of Mg reserves are in the skeleton and the other 25% are in muscle and other soft tissues. The demand of the lactating ewe for Mg is very high. It is twice that of a lactating dairy cow and is at its peak about two weeks into lactation. The loss of Mg in milk probably predisposes ewes to hypomagnesaemia and some workers have suggested that hypomagnesaemic tetany does not occur unless there is a concomitant hypocalcaemia. This has not been the finding in reported cases in New Zealand.

Finally, it is the failure of the Mg^{2+} ion to reach the central nervous system which produces the severe convulsions associated with the disease. The precipitating factor for clinical hypomagnesaemia is energy balance. This may be brought about by either inadequate feed over a period of time or the sudden stress of inclement weather. It may also be induced as a result of other diseases such as parasitism, pink eye or foot lameness (foot abscess and footrot).

Clinical Signs

Ewes with hypomagnesaemia are usually found dead in the paddock. Initially affected sheep may become dull and stop eating. Usually, such ewes if disturbed will develop muscle tremors and severe nervous excitement. They will collapse on one side with their head thrown back and throw convulsions with severe limb paddling (Fig. 5.21). Frothing at the mouth is a common feature as are nystagmus, rapid heartbeat and rapid respiration. Death usually occurs in 4-6 hours.

Figure 5.21. A seizuring ewe with hypomagnesaemia ("grass staggers").

Diagnosis

Special mention needs to be made of the diagnosis of hypomagnesaemia, as samples from overt cases for clinical pathology are not easy to obtain. Obtaining a good history of the feeding and management of the flock prior to the suspected occurrence of the disease is very important. Along with this must be a careful assessment of the condition of the ewes and the stage of lactation at which deaths have occurred. Knowledge of previous applications of fertiliser to the farm is also helpful as the outbreak may be associated with the previous heavy use of potassic topdressing. Typically a number of lactating ewes are found dead.

Post-mortem examination of dead ewes is seldom helpful but exclusion of clostridial diseases is important. Gas filled intestines and some pericardial fluid are seen at autopsy in some cases, these are also seen in deaths from clostridial diseases. A history of flock vaccination will also help to eliminate these diseases as a cause of death. If ewes are found within 12 hours death the Mg levels in CSF or vitreous humour can be analysed.

If clinical cases are seen, blood samples collected from them for serum Mg and serum Ca estimations may be helpful. Also the response to treatment of such cases may aid your diagnosis (Table 5.22).

Table 5.22. Serum Mg and Ca levels of a ewe showing signs of tetany.

Time of Sampling	Mg mmols/litre	Ca mmols/litre
When first showing clinical signs	0.43	2.41
18 hours later, during tetany	0.24	2.19
24 hours after treatment	0.97	2.20
Normal	0.7-1.2	2.0-3.0

In general, it is necessary to obtain blood samples from at least 10 other ewes from within the flock. Although these may not be showing overt signs of hypomagnesaemia, some will have very low serum Mg and the mean of the sample will be lower than normal (see Table 5.23).

Table 5.23. Serum Mg and Ca levels from a ewe flock in which clinical cases of hypomagnesaemia had occurred (West and Bruère, 1981).

Mg mmols/litre	Ca mmols/litre
0.16	2.53
0.26	2.28
0.27	2.44
0.34	2.66
0.41	2.58
0.44	2.79
0.55	2.61
0.56	2.87
0.58	2.21
0.66	2.46
0.71	2.35
0.72	2.37
0.72	2.33
0.74	2.53
0.78	2.38
0.80	2.18
0.80	2.05
0.82	2.68
0.94	2.79
0.99	2.58
mean 0.61	mean 2.47
'normal values' 0.7-1.2	'normal values' 2.0-3.0

Treatment

If clinical cases are treated quickly, there is a good chance of recovery. The subcutaneous injection of 50-70 ml Mg sulphate (20%) and Ca borogluconate 25% is recommended. These can usually be purchased as one solution primarily for use in cattle. In some instances intravenous administration is used in addition to subcutaneous injection. Ewes, which respond to treatment, may also be identified and drenched with Mg oxide to boost their Mg intake.

Magnesium oxide at a rate of 10 gram per sheep per day may be administered to the rest of the flock if this is practical. Simply increasing the hay ration may be useful to avert further cases in the remainder of the flock by encouraging salivation and thus increasing the rumen Na^+/K^+ ratio. If it is considered that access to lush greenfeed was the cause of the hypomagnesaemia, the flock should be gradually reintroduced to such feed. Again the use of good quality hay supplement to see ewes through the danger period may be helpful.

Prevention

Recommendations for preventing further cases of hypomagnesaemic tetany should aim at increasing the daily Mg intake, particularly that of older ewes which might be rearing twins. Magnesium in the form of calcined magnesite or Causmag (magnesium oxide), sprayed on to hay, is an effective preventative measure, but unless sheep are pre-conditioned to eating hay, it may not be eaten. In addition all procedures such as yarding, trucking and droving should be kept to a minimum to reduce stress and lessen the chances of precipitating an outbreak of the disease.

When introducing ewes to lush feed, this should be done gradually and for a few hours initially. Shelter should be provided during cold, windy weather either by grazing on undulating country or utilizing sheltered paddocks. Attention should also be given to dealing with any concurrent disease. Parasite levels should be monitored and the ewes treated if necessary.

In some instances it may be necessary to seek advice on fertiliser use. Magnesium fertilisers may need to be considered and the use of potassium fertiliser should cease unless shown to be necessary. Assuming an adequate feed intake, animal Mg requirements during early lactation can only be met if the pasture Mg concentration is greater than 0.20% (Edmeades, 2004). To achieve such concentration in spring requires that the soil quick-test Mg is > 25-30 (a soil Mg test related to soil exchangeable Mg concentration). On those soils in New Zealand with low Mg status such levels can only be achieved with large capital inputs of fertiliser Mg (>100 kg Mg/ha). This is in contrast to application of 25 kg Mg/ha to maintain soil quick-test value of 8-10 (herbage Mg concentration 0.10%) to achieve maximum pasture production.

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5.9 Fluorine (F)

For details on soil F, please refer to section 6.1 (Issues with Contaminants in Fertilisers and By-Products) of the Intermediate Sustainable Nutrient Management in New Zealand Agriculture study Guide.

Aetiology and Pathogenesis

Phosphates fertiliser toxicity is most commonly seen in heavily pregnant or lactating ewes and therefore it is commonly confused with the metabolic diseases. Sheep grazing pasture that has recently been topdressed with phosphate-based fertilisers such as superphosphate or diammonium phosphate (DAP) are at risk. It has also been reported in ewes that have access to fertiliser bins or sheds. In ewes, the major risk period is during late pregnancy or lactation (late winter and early spring), although there is the potential for toxicity to occur at other times. Toxicity has also been reported in lactating cows, goats close to kidding, and deer.

The major toxic principle is believed to be fluorine, which is present at concentrations of 1-3% as a contaminant of the phosphate rock used in fertilisers. The fluoride causes toxic nephrosis of the kidney followed by severe uraemia and death. The role, if any, of phosphorus in the pathogenesis of the disease has not been identified. It has been estimated that 200 to 300g of superphosphate would be sufficient to kill most sheep although this will vary depending on the fluoride concentration of the superphosphate. It is not uncommon for secondary hypocalcaemia or Salmonellosis to also be present.

Despite large quantities of phosphatic fertiliser being applied each year in New Zealand, fertiliser toxicity is relatively uncommon. Several predisposing factors are important in the occurrence:

- application of fertiliser in spring with little or no rain falling between application and grazing (it is suggested that at least 25mm of rain needs to fall before pasture can be considered "safe")

- grazing pregnant or lactating ewes, which have a high feed demand, on topdressed pasture

- high stocking rates and low pasture covers

- dew at the time of topdressing may allow fertiliser to adhere to the blades of grass, increasing the likelihood of toxicity

Other fertilisers have also been associated with toxicity. Basic slag, a by product of steel-making that is used to raise the pH of acid soils, has caused deaths predominantly in dairy cattle but also in sheep. It has been suggested that Vanadium toxicity may have been involved.

Clinical Signs

Signs usually appear three to five days after exposure and include ataxia, muscle tremors, depression, thirst, diarrhoea and death. These signs are similar to those of hypocalcaemia, and because affected ewes may show a temporary improvement following treatment with Ca borogluconate it is important to distinguish between the two conditions. Often ewes are simply found dead, which again may be mistaken for deaths due to other metabolic diseases. The

presence of watery diarrhoea may be mistaken for enteric salmonellois. Mortality rates are typically in the order of 1-5% although on occasion can be as high as 10%.

Pathology and Diagnosis

Necropsy findings vary although a common finding is pale, swollen kidneys (Fig. 5.22). Ewes may have reddened and ulcerated abomasal, and sometimes intestinal, mucosa and watery yellow-brown diarrhoea. In some ewes the liver has been slightly enlarged and pale with an accentuated lobular pattern although this is not a characteristic finding.

The characteristic histological finding is toxic nephrosis of the kidney. Serum biochemistry of affected ewes almost always shows marked haemoconcentration due to dehydration, and uraemia. In some cases, hypocalcaemia and hyperphosphataemia are present. Diagnosis is based on the history, necropsy findings, histopathology of the kidney, and analysis of rumen or serum fluoride levels. Rumen fluoride concentrations of greater than 200 mg/kg are consistent with fluoride toxicity. Granules of fertiliser may be visible at the base of the pasture on which the ewes have been grazing.
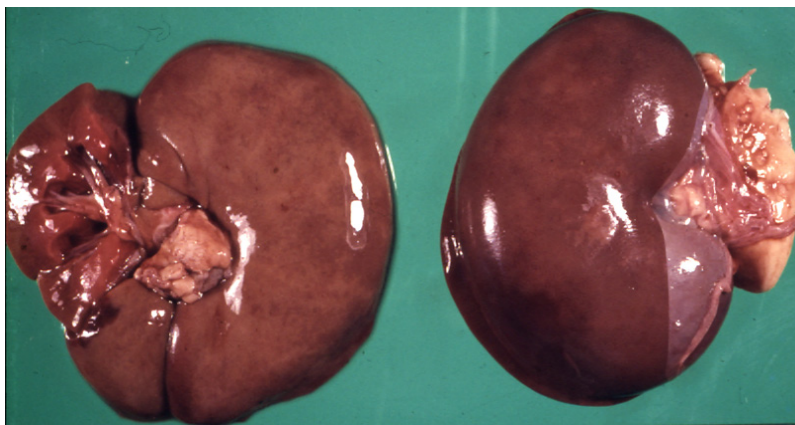

Figure 5.22.   Swollen kidneys from a ewe that died of phosphatic fertiliser toxicity.

Case example

The following outlines a case of fertiliser toxicity. Note that the mortality rate of 12% is higher than what would usually be expected:

On this property, DAP fertiliser was applied at 125 kg/hectare by aerial topdressing in late August onto paddocks where 770 mixed-age ewes were grazing. No rain fell for 5 days following the application. The ewes had been set-stocked about 2 weeks beforehand onto very short pasture and were due to start lambing at the time the fertiliser was applied. When the ewes were checked three days after fertiliser application about 30 were found dead and over the following week a further 56 ewes died, bringing the total number of mortalities to 86.

Serum collected from three affected ewes showed increased blood urea nitrogen (BUN) levels and hypocalcaemia. One ewe had hyperphosphataemia. The consistent post-mortem feature from six ewes examined was pale, swollen kidneys. In some of the ewes the liver was slightly

enlarged with rounded margins. In one ewe there was severe reddening of the abomasal mucosa and in another there were numerous erosions on the margins of the fundic mucosa. Histologically, the kidneys of all the ewes had a severe toxic nephrosis. In the livers there was varying degrees of cytoplasmic swelling of hepatocytes.

Treatment

Treatment of F poisoning of dairy cattle requires an immediate removal of the herd from the recently fertilised pasture. If this is not possible, offer the cattle with plenty of supplement so that the amount of fertiliser they are ingesting is diluted. Drenching the affected animal with calcium chloride is recommended in severe cases. Proper veterinary advice is required for the rate and frequency of drenching.

Prevention

Fertiliser toxicity of grazing livestock is uncommon compared with the amount of fertiliser that is applied annually in New Zealand. Pasture is more likely to be "safe" when 25 mm of rain has fallen or at least until a week after application. The most effective preventative strategy would be to avoid topdressing in spring, or to only topdress part of the farm and graze stock on the other part until the pasture is safe. However, for many farmers this may not be practical. Pregnant or lactating ewes are at greater risk so keeping them on pasture that has not been topdressed until the rest of the farm is "safe", or keeping them lightly stocked on long pasture may reduce the risk.

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Surveillance (2001): 28 (2) 20: Fluoride toxicity in sheep.

Reading

Refer to selected readings from the text Mineral Nutrition of grazing ruminants by N.D. Grace.

Activity

Make brief notes on the grazing animal's requirements for:

a. Copper

b. Cobalt

c. Selenium

d. Iodine

Bring notes to the Contact Course you are attending at Massey University.