Micronutrients and Animal Nutrition and the Link between the Application of Micronutrients to Crops and Animal Health

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Abstract: Micronutrients (or 'trace elements') are required in animal diets for health and welfare, and therefore they are essential for the agricultural production of milk, meat, fibre, and eggs. It is clear from the literature that deficiencies of micronutrients, particularly in their sub-clinical form where they are not visually apparent, can result in major reductions in productivity. Micronutrients are used mostly as the central elements of enzymes and co-enzymes in the biochemistry of ruminants and monogastrics. Thus, their deficiency often leads to sub-optimal growth and fertility. Within the farming system, the aim should be to only use supplementation with micronutrients where it is necessary; that is, where an actual or likely deficiency has been diagnosed. Further, the supplement used should be cost effective and appropriate to the farming system in question. Gaps in knowledge still remain, the most obvious being the use and levels of micronutrients that are typical in manures and the use of these in animal systems.

Key Words: Micronutrients, animal health, animal nutrition

Introduction

There is often some confusion about what is a 'micronutrient' in terms of soil/plant/animal interfaces. The first thing to note is that to be important an element must have nutritional relevance for livestock; this usually means that it is possible for an animal to become 'deficient' in that element and show physical signs of that deficiency. These signs may be 'clinical' or 'sub-clinical'. Clinical signs are those that are obvious to see, such as browning of the hair in the case of copper deficiency; in these instances diagnosis is relatively simple.

More commonly (and more problematically) animals can be sub-clinically deficient, where outward signs are not obviously seen, but where production is compromised. Examples here are loss of fertility through selenium deficiency and loss of immunity to infection through cobalt deficiency. The problem here is that the deficiencies can cause more harm because they are not clearly seen and so they can be more widespread, cause more loss of production, and be more difficult to diagnose than clinical deficiencies. Indeed, if animal health and welfare are obviously compromised by a clear clinical deficiency, then it is probably the case that loss of production has been occurring for a longer time though a sub-clinical deficiency in the animal population.

For the purposes of this paper, only those elements that are relevant to ruminant livestock will be considered in detail. These are:

Iron (Fe)

Copper (Cu)

Cobalt (Co)

Selenium (Se)

Iodine (I)

Manganese (Mn)

Zinc (Zn)

Boron (B)

Micronutrients are also essential for monogastrics such as pigs and poultry and a separate section will deal with these species. It should be noted that most deficiencies occur in free ranging ruminants where control of dietary intake is limited. For many

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monogastrics, micronutrient supply can be more closely directed through supplementary feeding.

The need for micronutrients

Each of the elements has at least one major role in the physiological functioning of the animal. This is usually also the primary cause for the clinical deficiency symptoms that may be apparent. It is worth noting that many of the functions that are dependant on micronutrients are delivered biochemically through the actions of enzymes and co-enzymes. Enzymes that are associated with micronutrients and dependant on them are often termed the 'metallo-enzymes' (McDonald et al., 1981). These are critical in all areas of physiology and assist mainly in the chemical transformations that enable biochemical reactions to occur, and, therefore, for the animal to gain energy, grow, and reproduce. Identifying which enzyme system the element is involved with usually leads to the discovery of why it is important and why the clinical and sub-clinical deficiency symptoms transpire.

In addition, the roles are often complex and different elements may interact with each other. Also, some elements may be toxic if supplied in greater quantities than the animal requires. However, the main roles, deficiency symptoms, and toxicities are given in Table 1.

The importance of micronutrients

A case study with cobalt

Probably the first useful description of Co deficiency was reported by the 'Ettrick Shepherd' in southern Scotland (Hogg, 1831). He noted a common wasting disease in sheep and, although he could not identify a causative agent, he did state that the problem was not contagious and was related to diet. In addition, he reported that the severity of this 'pining' varied from a marked wasting disease to a mild ill-thrift and that the best curative method was the periodic shifting of sheep to different pastures.

The biological form of Co in animal tissues was not apparent until Smith (1948a) isolated an anti-pernicious anaemia factor from liver. Four tons of material yielded 1 g of a red substance containing 2 pigments. The compound was named vitamin B12 and the presence of Co in its structure was quickly recognised (Smith, 1948b).

Cobalt has no physiological role in higher mammals except through vitamin B12 and different forms of this vitamin, the 'cobalamins', exist (Dryden et al., 1962). Only 2 of these, adenosylcobalamin and methylcobalamin are physiologically active. Adenosylcobalamin functions as a co-factor in the reversible conversion of methylmalonyl CoA to succinyl CoA.

This biochemistry is vitally important to the ruminant. The volatile fatty acid propionate is produced by microbes in the rumen and is used as a major energy source that feeds the Kreb's tricarboxylic acid cycle; a process that enables all animals to break down and store energy. The impairment of propionate catabolism is the primary metabolic defect supervening Co/vitamin B12 deficiency in ruminants and was first described by Marston et al. (1961). Basically, lack of Co leads to a lack of vitamin B12, which impairs the conversion of food into energy; hence animals suffering from deficiency go off their diets, are lethargic, and begin to waste away or 'pine'.

The effects of both clinical and sub-clinical Co deficiency on animal performance are based on a dearth of energy, and they are loss of appetite and weight loss in the clinical form and reduced feed intake and sub-optimal growth and yield in the sub-clinical form. However, the sub-clinical effects are more complex and damaging to the livestock farmer. That these include effects on immune function and fertility was shown by Fisher and MacPherson (1986). In a controlled experiment based on the feeding of a Co-deficient diet from before tupping, these workers kept 1 group of hill sheep (Scottish Blackface cross Swaledale) sufficient in Co ('OK') by weekly dosing with cobalt sulphate ($CoSO_4$) solution, whilst the cobalt status of 2 other groups was allowed to decline. In these latter animals, the disease in one group was allowed to descend to its clinical form ('clinical') and the others were supplemented with $CoSO_4$ from mid-pregnancy onward, holding the disease in its sub-clinical form ('sub-clinical').

The ability of isolated white blood cells to phagocytose (engulf) and kill yeast cells in culture was quickly depressed in the clinical and sub-clinical ewes (Figure). Thus, the ability of the sheep to withstand infectious disease was compromised.

The time taken for the lambs from clinically and sub-clinically Co-deficient ewes to stand and suckle was also much longer in comparison to those from mothers that

Table 1. The main roles, deficiency symptoms, and toxicities for micronutrients in ruminant livestock.

Element	Role	Deficiency symptoms	Toxic?	Notes
Fe	Protein and enzyme function. Blood haemoglobin.	Anaemia	No	
Cu	Haemoglobin formation, enzyme function, and pigments.	Anaemia, poor growth, bone disorders, digestive upsets, infertility, brain and spinal cord lesions. Decolouration of hair.	Yes	Deficiency commonly termed 'swayback' in sheep. Cu poisoning is a cumulative effect from high Cu intakes and tolerance varies considerably between species and breeds.
Co	Vitamin B_{12} function and energy assimilation.	Poor growth, anaemia, loss of coat, exudate from eyes, low immunity to disease, infertility.	No	The clinical deficiency is often termed 'pine' or 'pining'.
Se	Vitamin E function	Poor growth, white muscle disease, infertility.	Yes	Se is highly toxic at levels above 5 mg kg^{-1} DM in the diet and can cause death through respiratory failure in acute toxicity.
I	Thyroid gland function	Goitre and reproductive failure.	Yes	Very small amounts of I are needed. Toxicity symptoms are rapid loss of feed intake and weight.
Mn	Enzyme activation	Retarded growth, skeletal abnormalities, ataxia in newborns and reproductive failure.	Yes	Ruminant animals need Very little Mn and deficiency is rare. Toxicity requires high levels of Mn intake and is therefore also rare.
Zn	Enzyme function	Stiff and swollen joints	Yes	Deficiency and toxicity are rare. Response to Zn supplementation is rapid. Excess Zn intake can lead to Cu deficiency
B	Enzyme function	Weak bones, low conception rates, poor immune function	?	The importance of B is a relatively recent discovery and detailed information is scant.

were in the OK group (Table 2). It is safe to presume that in an outdoor lambing situation (as exists on many hill farms) the chances of hypothermia affecting the survival of lambs from Co-deficient ewes that took a long time (over 1 h) to start suckling would be considerably increased.

In addition, the levels of immunoglobulins derived from colostrum that were measured in the blood of lambs from Co-deficient ewes were only approximately two-

Table 2. Indicators of immediate post-lambing vigour in lambs from clinically and sub-clinically Co-deficient ewes and Co-sufficient counterparts.

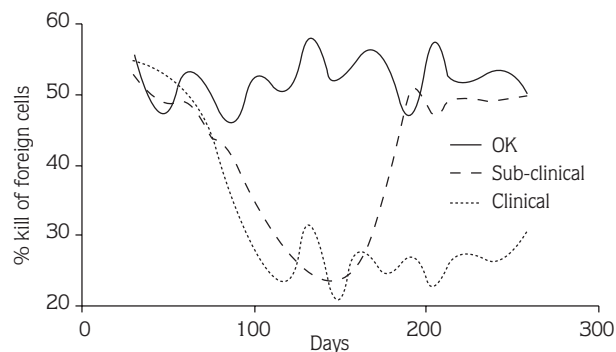| Time from birth to (average in minutes):	Clinical	Sub-clinical	OK
Standing	22	29	15
Finding udder	41	44	24
Suckling	76	61	31

Figure. The effects of clinical and sub-clinical Co deficiency on the ability of white blood cells from pregnant ewes to engulf and kill yeast cells in culture. Tupping took place from day 85 to 115 and lambing from day 225 to 255. The sub-clinical group were supplemented from day 170.

thirds of those found in lambs from sheep in the OK treatment (Table 3). This again suggests, as was found by Fisher and MacPherson (1986) in the recorded levels of neonatal disease and mortality in lambs, that progeny from Co-deficient dams will be more susceptible to life-threatening diseases such as clostridiosis.

Table 3. Levels of immunoglobulins derived from colostrums in the blood of lambs from clinically and sub-clinically Co-deficient ewes and Co-sufficient counterparts.

Lamb blood immunoglobulins (as % of OK)	Clinical	Sub-clinical	OK
2 weeks after lambing	69	61	100
4 weeks after lambing	62	52	100

These results demonstrate that sub-clinical deficiency in the micro-nutrients can be just as damaging to the physiological integrity of animals as can clinical deficiency. The most concerning aspect here is that sub-clinical deficiency can go undetected, often for generations on certain farms, simply because there are no outward signs or symptoms. However, dose/response trials have demonstrated that ruminants of marginal Co/B_{12} status show sub-optimal production. This was recognised by Dunlop (1946), who reported that non-supplemented sheep in '…sub-minimal Co-deficient areas' had depressed fertility and reduced lamb live weight gain, compared to Co supplemented controls. Working on marginally Co-deficient pastures in Wallaceville, Wellington, New Zealand, Andrews (1965) noted similar effects and also demonstrated the fluctuating state of

deficient animals. In some years, no response from growing lambs to Co supplementation could be reported, but in other seasons on the same pasture, Co supplemented animals showed lower mortality rates from parasitic infection and greater live weight gains compared to undosed controls.

Cobalt deficiency and fertility

Many of the micronutrient deficiencies that exist have a negative impact on the fertility of livestock. Cobalt is no exception and can be used to illustrate the point.

Dunlop (1946) was the first to recognise this link for Co deficiency. Working throughout the west of Scotland, he reported, as an example, that on one hill farm the administration of 100 mg Co per os on 3 occasions (before tupping, before lambing, and at the summer dipping) resulted in a reduction in the number of barren ewes and an increase in lambing percentage (Table 4).

Table 4. The effect of dosing hill sheep with Co on barren ewe and lambing rates (after Dunlop, 1946).

Treatment	% barren ewes (± SE)	% ewes with lambs at 8 weeks (±SE)
-Co	7.1 ± 1.5	76.3 ± 2.6
+Co	4.3 ± 0.5	89.4 ± 2.4

There is still a lack of evidence in the literature that would fully explain Dunlop's very practical observations and this is the case for most micronutrient effects, but there are some clues. Mgongo et al. (1985) found that goats with Co deficiency showed irregular oestrus, with lower cyclic progesterone and luetinising hormone concentrations in blood. In a later experiment (Mgongo et al., 1986), these workers also reported a greater number of anovulatory cycles ('false heats') in Co deficient animals and concluded that the site of action for the effects was the hypothalamus-pituitary axis. The inference here is clear; sub-clinical deficiencies of micronutrients can be very damaging to animal performance and profitability, simply by leading to small reductions in fertility. Every infertile cycle or missed fertile cycle of livestock means more time spent barren, higher culling rates, extended calving/lambing indices, and therefore higher costs and lower financial returns.

The occurrence and diagnosis of cobalt deficiency

It is also important to note that the occurrence and level of deficiency can vary within and between seasons

and farms. Thus, the effects may not be seen where soil, management, and sward factors combine to provide adequate micronutrient input in a particular season, but these circumstances may change given another set of interacting factors. The occurrence of deficiency diseases is also dependent on the level of animal performance targeted. Thus, high yielding dairy cows, fast growing beef animals, and sheep expected to show a high lambing rate are more susceptible to deficiency than those expected to attain lower levels of output.

For these reasons, it is difficult to predict exactly where and when a deficiency will be manifest. The impact of these complexities on diagnosis and treatment is discussed in later sections, but it is worthy of note here that the use of vitamin B12 in blood as a diagnostic technique only provides a historical look at Co intake. Indeed, a reduction in blood vitamin B12 probably lags some 4 to 6 weeks behind an actual reduction in intake (due to the 'buffering' capacity of the liver in its 'store' of vitamins) and certainly cannot be used in prognosis to predict a deficiency occurrence. For example, Paterson et al. (1991) found that Co concentrations in both soil and herbage failed to diagnose the Co status of grazing ewes and lambs in response to Co applications to pasture.

Selenium

The nutritional importance of Se became evident in the 1950s when it was shown that most myopathies in cattle and sheep could be prevented by supplementing the diet with the element or vitamin E. The role of Se containing vitamin E as a component of the enzyme glutathione peroxidase (see below) was demonstrated in 1973 (McDonald et al., 1981). This enzyme is involved in the removal of hydrogen peroxide from REDOX type reactions in cells and in modern parlance is thus termed an 'anti-oxidant'.

A substantial part of northern Europe produces herbage with levels of Se below the minimum range of concentrations, 30-100 µg kg^{-1} DM, required for satisfactory health and performance in grazing ruminants (Gissel-Nielsen et al., 1984). Deficiency is of far greater concern than toxicity in seleniferous soils. Low concentrations of Se in herbage are generally associated with soils of low total Se content and those derived from acid parent rock.

From a practical farming perspective, Gupta and Wilkinson (1985) reported that the greatest variations in

herbage Se are probably caused by plant rather than by soil factors, particularly by dilution during rapid growth. Evans et al. (1983), conducting a survey in England and Wales, found a very poor correlation between total soil Se and Se levels in conservation-stage herbage. These latter workers also found that soil and herbage values were very poor predictors of blood Se levels in grazing stock.

The ability to detect Se deficiency in animals was aided by the discovery of the Se-containing enzyme glutathione peroxidase (GSH-Px) in blood. The measurement of GSH-Px in ruminant serum is now the standard technique for assessing Se status in livestock, but Halliday (1983) rightly points out that finding a low GSH-Px status in animals is an indicator of likely Se deficiency, rather than an unequivocal indicator of deficiency and consequent production loss.

Clinical Se deficiency is not common in modern agriculture. This is perhaps due to greater awareness (see section below on farmers' knowledge). However, sub-clinical deficiency leading to sub-optimal live weight gain of young beef and lamb stock, and also to reduced fertility of suckler cows, ewes, and dairy cows, if often a problem. Prophylactic treatment is simple and effective, but it should be remembered that, in the UK (which has large areas of acid base rock), Se deficiency is most commonly also associated with deficiencies of Co and Cu (SAC/SARI, 1982). It should therefore be remembered that treating a Se deficiency only will most likely leave a Co and/or Cu deficiency untreated to continue a damaging effect on animal performance.

Selenium toxicity

Poisoning of ruminant stock with Se is uncommon, but can occur on seleniferous soils. 'Alkali' disease and 'blind staggers' are localised names for diseases of animals grazing certain seleniferous areas in the USA. Acute poisoning, which results in death from respiratory failure, can result from sudden exposure to high Se intakes. Chronic forms of the condition include dullness, stiffness of the joints, loss of hair from the tail, and hoof deformities.

Copper

The role of Cu in mammalian metabolism was first reported in 1924 (McDonald et al., 1981). Although not itself a constituent of haemoglobin, Cu is used in its formation. Cu is present in certain other plasma proteins, such as ceruloplasmin, which are concerned with the

release of Fe from cells into plasma. A deficiency of Cu impairs the animal's ability to absorb Fe, mobilise it from the tissues, and utilise it in haemoglobin synthesis.

Copper is also a component of other proteins in blood. One of these, erythrocuprein, occurs in erythrocytes (red blood cells), where it plays a role in oxygen metabolism. The element is also known to play a vital role in many enzyme systems; for example, it is a component of cytochrome oxidase, which is important in oxidative phosphorylation. Copper is also necessary for the normal pigmentation of hair, fur, and wool, and is present in all body cells, being particularly concentrated in the liver, which acts as the main Cu storage organ in the body.

Since Cu performs a variety of functions in the animal body, deficiency symptoms are many and complex. These include anaemia, poor growth, bone disorders, scouring, infertility, de-pigmentation of the hair and wool, gastrointestinal disturbances, and lesions in the brain and spinal cord. The lesions are associated with muscular incoordination, and occur especially in lambs, where the condition in the UK is known as 'swayback'. However, soil and pasture Cu levels are poor predictors and even poor diagnostic tools for Cu deficiency, because, as with Co and vitamin B12, the liver acts as the main storage organ and acts as a buffer to low Cu intake. Thus, swayback is often a reflection of historical Cu intake and can occur on pastures that seem adequate or even 'high' in Cu content.

Copper plays an important role in the production of 'crimp' in wool. The element is present in an enzyme which is responsible for the disulphide bridge in 2 adjacent cysteine molecules. In the absence of the enzyme the protein molecules of the wool do not form this bridge and the wool, which lacks crimp, is referred to as 'stringy' or 'steely'.

Copper toxicity

It has long been known that Cu salts given to animals in excess are toxic. Copper can be considered a cumulative poison and so care is needed in administering Cu to animals in any form. The tolerance to Cu varies between species and breeds, with sheep being the most susceptible of the ruminants. Where Cu in herbage is 10-20 mg kg^{-1} DM and herbage Mo and S contents are low, then Cu toxicity has been reported (McDonald et al., 1981). Therefore, Cu should only be administered to livestock where it is known that a deficiency exists or where there is a regular history of deficiency diseases; prophylactic treatment without this knowledge is unwise.

Copper-molybdenum-sulphur interrelations

Molybdenum (Mo) can exert a depressive effect on Cu absorption and availability in animals. It is now clear that Mo only exerts its effect on Cu in the presence of sulphur (S). Sulphide is formed by ruminal micro-organisms from dietary sulphate or organic S compounds; the sulphide then reacts with Mo to form thiomolybdate, which in turn combines with Cu to form an insoluble copper thiomolybdate (CoMoS$_4$), thereby limiting the absorption of dietary Cu.

Where industrial pollution results in large depositions of S from the atmosphere or where ammonium sulphate is used as a source of nitrogen for livestock farmers (who, with their suppliers, tend to ignore that the fertiliser contained 60% SO$_3$), S induced Cu deficiency can occur where soil Mo is relatively high. However, it should be strongly noted that the level of S being deposited from the atmosphere in western Europe is now only some 10% of what it was in the 1960s, '70s, and '80s (McGrath et al., 2002). Further, the practice of using ammonium sulphate as a nitrogen fertiliser and ignoring the S content has very largely gone. Coupled with the grass growth and nutritional quality benefits that can be obtained where S deficiency exists (Brown, 2000), this means that this negative interaction between Cu, Mo, and S is no longer a widespread threat in developed countries. As a rule, S/Mo induced Cu deficiency will only occur where herbage levels of S are at least 4000 mg kg^{-1} DM (0.4%) and combine with herbage Mo of over 3000 mg kg^{-1} DM (0.3%).

Iodine

Higher animals need very little I and its only known use is in the synthesis of 2 hormones, tri-iodothyronine and tetra-iodothyronine (thyroxine), both produced in the thyroid gland. These hormones accelerate reactions in most organs and tissues, thus increasing basal metabolic rate, accelerating growth, and increasing the oxygen consumption of the whole animal.

Lack of I leads to a reduction in thyroxine. The main indicator of clinical disease is enlargement of the thyroid gland, termed endemic goitre (referred to as 'big neck' on-farm, where animals develop a swollen neck as the gland expands). Reproductive disorders are the most common problem arising from I deficiency, with dams

giving birth to hairless, weak, or dead young. General infertility is the symptom seen in more sub-clinical situations; again a difficult 'symptom' to diagnose and attribute to a micronutrient deficiency.

Goitrogenic compounds can also induce goitre, even where dietary I is sufficient. These substances are present in certain potential ruminant feeds, such as plants from the *Brassica* family (kale, cabbage, and rape) and also soya beans, linseed, peas, and groundnuts. Livestock fed on diets containing significant quantities of these feeds should receive I supplementation.

Iodine toxicity

Toxic effects of excess I intake include depressions of weight gain and feed intake. However, these are rare and a dietary level of 50 mg kg^{-1} DM is necessary to induce toxicity in animals of around 100 kg bodyweight.

Iron

More than 90% of Fe in the body is combined with proteins, the most important being haemoglobin, which contains about 3.4 g kg^{-1} of the element. Iron also occurs in blood serum in a protein called transferrin, which is concerned with the transport of Fe from one part of the body to another. Ferritin, a protein containing up to 200 g kg^{-1} of the element, is present in the spleen, liver, kidney, and bone marrow and provides a form of storage of Fe. Iron is also a component of many enzymes including cytochromes and certain flavoproteins.

Since more than half the Fe in the body occurs as haemoglobin, a dietary deficiency of Fe would clearly be expected to reduce formation of this protein and result in anaemia. Haemoglobin is contained in red blood cells (erythrocytes) that are continually produced in bone marrow and are always being 'turned-over'. However, the Fe from the used red blood cells is recycled in the formation of new ones and, as such, the dietary need for Fe is relatively low compared to how much is contained in the body as a whole. Thus, Fe deficient anaemia is not common in ruminants and is most likely to occur in pregnant dams and rapidly growing young.

Zinc

Zinc has been found in every tissue of the animal body. The element tends to accumulate in bones rather than the liver. High concentrations have been found in skin, hair, and wool (McDonald et al., 1981). The main use of Zn is in enzymes and these include: carbonate dehydratase, pancreatic carboxypeptidases, glutamic dehydrogenase, and a number of pyridine nucleotide dehydrogenases. Zinc also has a role as a co-factor for many other enzymes.

Clinical deficiency symptoms in calves include inflammation of the nose and mouth, stiffness of the joints, swollen feet, and parakeratosis. The response to supplementation is dramatic, with improvements in skin condition being seen within 2 to 3 days. Sub-clinical conditions associated with general 'ill-thrift' are also easily treated with Zn supplementation.

Zinc toxicity

The toxic effects of an excess dietary intake of Zn are depressed feed intake and induced Cu deficiency. However, ruminants have a high tolerance for this element and poisoning will be very rare.

Manganese

The amount of Mn present in the animal body is extremely small. Most tissues contain traces of the element, the highest concentrations being in the bones, liver, kidney, pancreas, and pituitary gland (McDonald et al., 1981). Manganese is important to ruminants as an enzyme activator and resembles magnesium in its ability to activate a number of phosphate transferases and decarboxylases, notably those concerned with the tricarboxylic acid cycle and thus energy acquisition and utilisation.

Clinical Mn deficiency will show in livestock as retarded growth, acute ataxia in newborns, skeletal abnormalities and reproductive failure. Sub-clinical deficiency symptoms in cattle have been recorded as depressed or delayed oestrus and conception, as well as increased abortions.

Boron

The exact action and function of B in ruminants, indeed all animals, is unclear. Indeed, its inclusion as a micronutrient for livestock may still be seen as controversial in a paper such as this. However, there is evidence that B is needed in bone formation (Bergman, 1981). Apparently, low levels of B can cause brittle bones and joint problems. Boron is associated with Ca processes in plant cells and this may also be the case in mammalian bone. The role of B, directly or indirectly, in bone physiology requires further investigation.

Boron toxicity

This subject is again unclear. Reports of excess B leading to reproductive dysfunction await adequate scientific investigation.

Animal requirements

It is important to establish the requirements of animals for micronutrients. It may be that soil and plant supply is low, but is adequate for a relatively low level of animal performance. Alternatively, a soil and plant supply that appears adequate may prove borderline or low in the case of a production system that targets high levels of animal performance. Standard requirements (ARC, 1980; SAC, 1982) and allowances are given in Table 5.

Micronutrients and monogastrics

According to Nielsen (2004), Se deficiency in foals and horses has been observed to result in white muscle disease. In swine, deficiency symptoms include hepatic necrosis, oedema of the colon, lungs and subcutaneous tissues, bilateral paleness, white muscle disease, and dystrophy of the myocardium (heart). Pigs can also suffer from impaired reproduction, reduced lactation, and compromised immune response resulting from sub-clinical and clinical Se deficiency. Se deficient chicks exhibit poor condition, reduced feed intake, and growth and leg weakness; exudative diathesis may result, especially at 3 to 6 weeks of age in housed birds and often leads to death.

Chronic Se toxicity in monogastrics has similar symptoms to those in ruminants. Additionally, swine may exhibit impaired reproductive performance generally and in chickens Se toxicity leads to reduced egg production and hatchability and deformed chicks.

Whilst Co deficiency is not really an issue in monogastrics, as it is involved with ruminant metabolic pathways, the deficiency symptoms and effects of Zn in monogastrics are very similar to those of ruminants. In swine, the skin lesions are most prominent in the extremities; in chickens the effect is seen as severe dermatitis and poor feathering. In the case of B, monogastric animals respond to deficiency as do ruminants, with malformed bones and joints. Indicative allowances of Se and Zn in the diets of pigs and chickens are shown in Table 6.

Diagnosis of micronutrient deficiencies

In terms of the impact of micronutrient deficiency and animal performance, there are some indicative levels of elements in soil, grass, and other forages. These are shown in Tables 7 to 9. However, these are incomplete and should be treated with caution. Measuring levels in herbage is generally more satisfactory than looking at levels in soil (SAC/SARI, 1982). The complex interactions of soil, plant, and animal mean that it is very difficult to predict or diagnose a micronutrient deficiency from analysing soils and plant tissue. This applies more to some elements such as Co (Voss and MacPherson, 1977; Paterson et al., 1991) than to others, but may be taken as a rule of thumb for all elements.

The surest way of diagnosing a deficiency of any micronutrient is to sample the animal (usually blood) and look for the metallo-enzymes that the elements are an essential part of. If a deficiency is suspected, then blood sampling 10% of the animal group should be sufficient to show up problems. If the specific reasons for the deficiency are required in order to understand the route to solving the problem, then soil and herbage analysis

Table 5. Micronutrient allowances for ruminants (mg kg^{-1} of dietary dry matter).

Element	Young calf	Growing bullock	Cows	Lambs	Sheep
Fe	40	35	30	30	40
Cu*	1.2	15	15	5.0	7.0
Co**	0.11	0.11	0.10	0.1-0.2	0.1-0.2
Se	0.1	0.1	0.1	0.1	0.1
I - winter	0.5	0.5	0.5	0.5	0.5
I - summer	0.15	0.15	0.15	0.15	0.15
Mn	25	25	40	25	40
Zn	50	40	40	40	40
B	5	5	5	5	5

* When feeding stuffs contain > 2.0 mg Mo kg^{-1} DM and/or > 3.0 g total S kg^{-1} DM absorption is depressed and additional dietary Cu is required.

** When rations high in concentrates are fed, some experiments have shown that 0.2 mg kg^{-1} gave better results.

Table 6. Selenium and zinc allowances for monogastrics (mg kg^{-1} of dietary dry matter).

Element	Growing pigs	Gestating and lactating sows	Chicks	Laying hens	Broilers
Se	0.15-0.30	0.15	0.10-0.15	0.05-0.08	0.15
Zn	50-100	50	33-38	29-44	40

Table 7. Potentially useful diagnostic categories of micronutrients in soil.

Element (mg l^{-1})	Very Low	Low	Average	High	Very High
Fe	<5	5-10	10-15	15-25	25-50
Cu	<0.3	0.3-0.8	0.8-1.2	1.2-2.5	2.5-10.0
Zn	<0.6	0.6-1.0	1.0-3.0	3.0-8.0	8.0-20.0

Table 8. Potentially useful diagnostic categories of micronutrients in grass.

Element (mg kg^{-1} DM)	Very Low	Low	Average	High	Very High
Fe	<50	50-100	100-150	150-250	250-500
Cu	<5	5-8	8-10	10-12	12-15
Co	<0.05	0.05-0.10	0.10-0.15	0.15-0.20	0.20-0.40
Se	<0.01	0.01-0.10	0.10-0.15	0.15-1.50	>1.50
Mn	<25	25-50	50-100	100-150	150-300
Zn	<15	15-25	25-50	50-75	75-150

Table 9. Typical values of Cu, Co, and Se in grass and forage.

Element	Intensive grazing grass	Extensive grazing grass	Hay	Silage (grass - range)	Silage (whole crop cereals)	Straw (Wheat and Barley)
(mg kg^{-1} DM)						
Cu	8.0	7.0	5.5	3.0-11.0	6.0	3.2
Co	0.10	0.08	0.12	0.05-0.40	0.07	0.04
Se	0.05	0.04	0.04	0.02-0.10	0.06	0.02

should be considered, but in primary diagnosis it is better to 'ask' the animal's themselves.

Approaches to correcting deficiency

There are many ways of correcting a low supply or an imbalance of minerals to livestock. Most of these are effective if carried out carefully. They are itemised in the following scheme:

Treat the soil

- Fertilisers and sprays

Treat the herbage

- Herbage sprays

Treat the animal

- Metered water

- Feeding blocks and licks

- Supplementation through the feed

- Injecting

- Drenching

- Dosing (e.g. of boluses)

The key to deciding which is the most appropriate for any particular situation lies in the following steps:

- Establish through soil/plant/animal investigation that micronutrient supplementation into the system is necessary

- Try to understand why the deficiency exists (low levels in soil; herbage species grown; production level of the animals)

- Establish which elements must be supplemented after taking into account any interactions that may occur

- Establish with the farmer which supplementation route best fits the system (for example, if the problem requires a quick solution, do not leave the farmer with a supplementation route where the elements are relatively unavailable and will take months or years to properly enter the whole of the system)

- Balance the decision on supplementation route with the relative costs of different options

- Implement supplementation and monitor effects

- Change supplementation according to monitored results.

It is important to take a balanced and holistic approach to treating micronutrient deficiencies in practical farm situations. It may be that providing multiple routes to solving a problem is appropriate, such as using fertilisers or feed blocks during the grazing season and feed supplementation over winter, with boluses used to 'top-up' the most at risk (highly productive) animals. However, the farmer must be assured that all the deficiencies are being addressed in the most efficient and cost effective manner. For example, Se deficiency is gaining increased profile in the UK, and rightly so. However, it is rare in the UK that for ruminants a deficiency of Se, Cu, or Co will exist in isolation of the other 2 elements. Therefore, when identifying and treating a Se deficiency, it is probably unwise to focus only on fertilisers, drenches, mineral feed supplements, and boluses that contain only Se and not Cu or Co.

Another issue here is cost. For example, a dairy cow can be supplied with Cu, Co, and Se through glass boluses supplied as 2 per animal per year. The current cost for this would be approximately $20 per cow. The treatment is easily administered and is relatively effective. Therefore, for treating deficiencies in these micronutrients, any fertiliser, spray, water, injection, feed supplement, or other treatment must be equally as effective, must contain the same elements, and must not cost more than $20 per cow.

Response to product use – example with a micronutrient containing fertiliser

It is important to record that animal responses to micronutrient containing products have been demonstrated experimentally and independently. The following example from Phosyn (1992) serves to show the positive impact of micronutrients when used in production systems.

At the ADAS Liscombe Experimental Station in Devon, lamb growth rates, particularly after weaning, were disappointing and it was suggested that micronutrient deficiencies could be the cause. Soil samples taken in the summer of 1990 showed that some fields were 'low' in magnesium, Cu, I, and Zn.

An experiment was conducted where in each of 3 treatment groups the live weight gain in 83 core lambs (249 in total) was recorded every month from 19 June to 22 September. Grazing for each group was kept separate and the grass height in each group paddock was controlled, using extra lambs as necessary, between a height of 6 and 7 cm. Thus, sward morphology was eliminated as a cause of differences in the results.

Three treatments were imposed:

Control – No micronutrients applied

Grasstrac – Fertiliser containing Cu, Zn, Co, I, Se, and sodium applied beginning of June

3. Grasstrac Sheep Special – Fertiliser containing Co, Se, and sodium applied beginning of June.

The results (Tables 10) suggested that the micronutrient treatments had a positive impact on lamb live weight by 11 August, compared to the control treatment, and these lasted through to September (when lambs would begin to be finished and sold). It is not possible to surmise from these data which individual or group of micronutrients were responsible for the response, particularly as there were no statistically significant differences between the 2 micronutrient treatments. However, the results illustrate the large positive responses that are possible to micronutrient supplementation in animals suffering from sub-clinical, or 'marginal' deficiency, with no apparent outward signs.

Farmer knowledge

In a July 2004 survey of just over 200 large UK grassland farmers (average number of dairy or beef cows 343, average number of ewes 387), 95% believed that 'trace element deficiencies in soil affected animal performance', but 33% did not know that particular regions are susceptible to deficiency (Kemira GrowHow, 2004, *pers. comm.*). Of the total sample, 44%, 28%, and 43% believed that their own farms suffered from Cu, Co, and Se deficiencies, respectively.

Table 10. Live weights and growth of lambs receiving micronutrients through fertiliser applications.

	Control	Grasstrac	Grasstrac Sheep Special	± SED	Sig. Diff.
Weight (kg) 19 June	24.8 [a]	25.5 [a]	25.6 [a]	0.60	NS
Weight (kg) 11 August	32.1 [a]	32.7 [a b]	34.3 [b]	0.79	P < 0.05
Final weight (kg)	34.0 [a]	37.0 [b]	37.2 [b]	0.71	P < 0.001
Growth rate (g day^{-1})	108 [a]	135 [b]	144 [b]	7.00	P < 0.001

Data in rows with different letter superscripts showed a statistically significant difference at the level shown.

Most farmers (64%) look to call in experts (such as agronomists and veterinarians) to diagnose and treat deficiencies, but few (under 20%) look for help to, or would trust, distributor representatives and product manufacturers.

Interestingly, of those that treat for deficiencies of micronutrients, most (57%) take advice exclusively from their vet. On average, farmers used 2.4 different means to correct deficiencies in their animals, with the most to least popular means being: licks (73%), boluses (53%), injections (40%), supplementing water (37%), supplementing feed (20%), using fertilisers (16%), and drenching (2%).

It seems from these data that in some livestock farming communities awareness of micronutrient deficiencies is high, vets are trusted and listened to the most, and several means of solution have been tried and are used concurrently. This reiterates the need for cost effective, efficacious techniques that address the micronutrient problems that farmers actually have.

Micronutrients in animal manures

Ruminants excrete most of the elements that they ingest and so manures can be a useful source, particularly when they are imported from areas that do not suffer from deficiencies. Monogastrics are even less efficient at absorbing micronutrients than are ruminants and so species such as pigs and humans may supply a useful source of elements.

There is not a great deal of data on the typical contents of micronutrients in manures, although Eriksson (2001) demonstrated how variable concentrations can be (Table 11).

Table 11. The micronutrient content of biosolids and manures (after Eriksson, 2001).

Source	No. of samples	Mean	Minimum	Maximum
Zn		mg kg^{-1} DM		
Sewage sludge	48	550	230	2300
Pig slurry	4	582	394	680
Pig FYM	4	680	347	821
Cattle slurry	4	154	128	170
Co		mg kg^{-1} DM		
Sewage sludge	48	6.20	1.50	32
Pig slurry	4	0.65	0.47	0.90
Pig FYM	4	1.10	0.47	1.70
Cattle slurry	4	0.85	0.63	1.10
Se		mg kg^{-1} DM		
Sewage sludge	48	1.3	0.50	2.8
Pig slurry	4	1.4	1.4	1.5
Pig FYM	4	0.89	0.19	1.30
Cattle slurry	4	0.56	0.40	0.77

Other work quotes concentrations of B in FYM of 20 mg kg^{-1} DM, 30 g m^{-3} in pig slurry, and 1.5-3.0 g m^{-3} in cattle slurry (Bergman, 1981). Whitehead (2000) reports concentrations from the USA in cattle dung of 1600-2000 mg Fe kg^{-1} DM. Clearly, more work on the levels of micronutrients in manures, and their use, is needed.

Conclusion

What is entirely certain is that deficiencies of micronutrients, particularly in the sub-clinical form, can markedly reduce the performance and profitability of a livestock agricultural system. Farmers require to be more focused on this, but long-term gain can only be attained if the farmers involved have trust in the solutions provided. In order to gain trust then these solutions must be based on efficacious, appropriate, and cost effective means of delivery.

This requires farm advisers to identify problems based on sound scientific knowledge and techniques, starting with the animal first. This information must then be applied in the most appropriate manner to the farming system in question and the situation should be monitored, feedback gained, and further improvements made.

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