Trace Elements - Exploding the Myths

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Introduction
Many issues and factors conspire to make New Zealand’s farm managers extremely susceptible to the notion that the productivity of their farm operation will be improved by mineral supplements. What is the basis for this assertion?

• Trace or micronutrient deficiencies, such as Cu, Co, Se reflect soil forming conditions. Agricultural development has required trace element supplementation in certain regions of New Zealand to achieve optimum stock health. The farming community is therefore “sensitised” to the potential need for supplements.

• In this situation trace element supplements can be an easy sell. Intuitively the manager feels much more comfortable with the notion that the suboptimal stock performance is due to mitigating circumstances - i.e. soil nutrient deficiencies - than a reflection of his management ability.

• Diagnostic criteria are crude: reference ranges are still not sufficiently robust to confirm that response to supplementation will occur. There is a tendency to go for peace of mind insurance.

• Local investment in mineral nutrition research does not reflect the uniqueness of the pastoral systems of New Zealand and the on-farm problems of mineral supplementation. The chemicals themselves are very cheap. In countries in which concentrate supplements are fed routinely they can be added cheaply to an animal’s diet. It doesn’t matter whether they are needed or not because the “insurance cover” is so cheap. In pastoral systems unique delivery systems are needed. The effective ones are generally not cheap to deliver to the animal; many approaches to supplementation are not effective.

• The industry is therefore susceptible to the introduction of research findings from overseas often from feedlot systems where feeding conditions, for example feeds fortified with fatty acids, and exposure to environmental stressors - for example exposure to pathogens - may significantly affect mineral metabolism and demand. Again what is fashionable and, in some cases, appropriate overseas can be “sold” because of lack of local information or critical local evaluation of the overseas data.

• Finally, in this complex situation, the major sources of advice for farmers are usually his veterinarian or local mineral or feed company representatives. I have no grounds
to be critical of the veterinary profession, many of whom I admire greatly and are close colleagues; even they would have to admit a potential conflict of interest as both advisor and potential supplier. They are not helped by the lack of local investment, reliable diagnostic tools and local data.

It is impossible in a short review such as this to deal with every potential mineral problem for every situation. I will attempt to justify what I have said with four issues currently under debate:

- The need to always meet (mineral requirement).
- Some difficulties of diagnosis.
- Strategic supplements to stimulate immunity.
- Supplements to modify acid-base balance and rumen function.

**The need to meet daily mineral requirement**

Under conditions of mineral intake greater than requirement the body regulates concentrations of most minerals in tissues and blood within “normal” well established limits. This is important because excessive accumulation of minerals in the body is generally toxic. With the onset of “deficiency” of daily intake these values are initially maintained, in some cases by increasing absorption, in others by reducing excretion, in other cases by utilizing expendable body pools of the nutrient - e.g. Ca and P from the skeleton, Cu or vitamin B₁₂ from the liver. Ultimately, the blood concentration falls but this still doesn't mean that the animal has insufficient of the mineral for the function, usually a specific enzyme activity, for which the particular mineral is required. This is in stark contrast to the consequence of failure to meet daily energy or protein requirement, which is an immediate (next milking) reduction in milk production or in growth of a young animal. Animals can handle seasonal and other fluctuations in mineral supply provided these are neither too severe or too prolonged. Similarly the concept that one can “build up the reserve of mineral nutrients” is fallacious unless the animal has previously suffered prolonged mineral depletion.

**Lack of diagnostic precision**

Copper will serve as a good example. Copper is important for a whole series of important enzymes whose function is protected during copper deficiency, Figure 1. Only in prolonged and/or extreme deficiency will enzyme levels be affected. Before that both liver (storage) and plasma (transport) Cu pools will have been depleted. Measurement of the enzyme concentrations is too expensive for routine analysis. Cost dictates that the transport pool (blood plasma) is used. The aim is to predict whether enzyme concentrations will fall below acceptable levels for normal metabolism sometime in the future. The problem is that in pastoral situations
dietary copper concentration fluctuates seasonally, varies with forage species and the ability of the animal to absorb copper is affected by other dietary constituents - sulphur, molybdenum and iron, the concentrations of which also fluctuate with season. Your advisor should be attempting to assess whether a low blood copper concentration is important in the context the animals likely future need, the likely future intake of copper and of nutrients which will affect the ability of the animal to absorb that copper. There is a tendency, erroneous in my view, to foster the attitude that we should supplement to maintain the “normal” tissue concentrations seen in animals with greater than adequate intake. The international literature has more reports of supplementation of hypocupraemic animals in which plasma Cu concentrations were restored to “normal” but which were without production response than of reports in which production response occurred. The latter are, in fact extremely rare. This suggests that the animal’s strong homeostatic mechanisms are able to maintain supply of Cu to critical enzymes despite low concentrations of Cu in transport forms. The marginal bands for serum Cu, indicating possible responsiveness, are very wide (3-9 µmol/L); and there are many instances of cattle tolerating mean values in this range (Underwood and Suttle; 2000). In New Zealand, Ellison (1992) describes trials with herds with serum values of 8.2 and 8.5 µmol/L only one of which showed response to supplementation. Small improvements in intake and/or availability on one property may have been the difference but this is often not measured or assessed.

Figure 1: Typical relationships between critical enzyme pools and transport and storage pools of a trace element.
Given these difficulties and uncertainties it is perhaps not surprising that supplementation “just in case” or as insurance-peace of mind is resorted to. An example is the widespread adoption of Cu supplementation of deer in Canterbury. Recent copper supplementation studies using 850 weaners on 10 Canterbury farms, on which copper supplementation was routine practice and on which mean plasma concentrations of only 8.6 µmol/L were observed in unsupplemented animals, has revealed no production responses (A M Nicol, 2001). Is this an example of too much insurance being sold?

About four years ago I was approached by WoolPro and MeatNZ with concern about the “increasing incidence of Co - vitamin B<sub>12</sub> deficiency in sheep in the South Island in sheep.” The diagnosis of a responsive condition was based on the large numbers of blood samples with Vitamin B<sub>12</sub> concentrations in or below the marginal reference range of 185-370 p vitamin B<sub>12</sub> mol/L serum. In reality, this indicates that status is low, but not necessarily that function is impaired. As a consequence only a proportion of cases of low vitamin B<sub>12</sub> status are actually vitamin B<sub>12</sub> or Co responsive conditions (Clark et al, 1989). We have begun to measure changes in the metabolite, methyl malonic acid (MMA) which accumulates when insufficient coenzyme (vitamin B<sub>12</sub>) is available to allow normal conversion of propionate, produced during rumen fermentation, to glucose. We have found many cases of serum vitamin B<sub>12</sub> below the reference range but with no elevation of MMA, indicating a low status but unresponsive condition. Analysis of MMA will be significantly more expensive than the standard vitamin B<sub>12</sub> test but it is suggesting that the existing vitamin B<sub>12</sub> reference ranges have been set conservatively high. The consequence is that the apparent need for supplementation is over-stated which, in the longer term, conditions producers to over-use supplements and divert attention from the real causes of lost production. This test will be a real help for the cattle industries because the vitamin B<sub>12</sub> blood analysis has not even been recommended for cattle.

Confusion from overseas data

Comparisons with overseas reference ranges can be quite misleading because the difference in feeding conditions between the northern hemisphere and NZ can impose quite different nutrient demands. A prime example is the difference in reference ranges for selenium between here and the USA. In this case a complication arises from the synergistic roles of Selenium and vitamin E in protection of cells from oxidant damage. Pastoral animals have a large and very adequate intake of the vitamin which protects lipids from oxidation and the production of toxic free oxygen radicals. The vitamin E content of conserved forages decreases during storage, is low in grains and the practice of lipid enhancement of supplements increases the need for anti-oxidants. Since one of the major roles of selenium is as a component of an oxidant-scavenging enzyme (Glutathione peroxidase - GSH-Px) it is not surprising that need for
selenium may be greater in typical northern hemisphere systems. The overseas reference ranges for whole blood Se are > 1460 nmol/L and for GSH-Px > 20 kU/L @ 25°C, almost 10 times the accepted New Zealand range. Not surprisingly this can lead to confusion and the notion that we perhaps should supplement to overseas levels in case we have missed something. There are clear seasonal patterns of change in blood Se and GPX concentrations in New Zealand, with generally lowest values in dairy cattle in mid-late summer and autumn. However, Whelan et al. (1992) in a comprehensive supplementation trial were unable to record differences in milk production between cattle with GSH-Px levels at 40% (0.7 kU/L @ 25C) or in excess of 200% of the New Zealand reference range, the latter achieved by supplementation. Another comprehensive study (Wichtel et al., 1994) is difficult to interpret. Small increases (~5%) in milk fat and protein were observed when cows in two herds were given 2 Se pellets. Cows given 4 pellets showed no response. Blood GSH-Px were increased similarly from below (0.7 and 1.5 kU/L) to in excess of 7 times the reference range irrespective of number of pellets. Convincing evidence of a lower requirement of lactating than growing cattle was, however, shown in growth rate responses of replacements in the herd with the lowest GSH-Px concentrations. The need for supplementation may differ with the particular production function of the animal. A lack of confidence that our lower reference ranges are in fact sound and confusing reports of responses of clinical conditions to supplementation in very different systems overseas, may have contributed to a perception that lactating cattle be responsive.

Minerals and immunity

That trace elements should be involved in the immune responses of animals should be no surprise, given their essentiality as components of enzymes and co-factors involved in the metabolism and in some cases the protection of cells. For example, both copper and selenium are components of enzymes involved in protecting tissues from oxidant stressors which are formed as part of the normal metabolism of cells. They are produced in increased quantities during exercise, high performance, in response to infection, and in handling toxic plant secondary compounds, which increase oxidant stress, such as occur in brassicas. However, although trace elements have this function, as described earlier, it is highly conserved even in prolonged mineral deficiency. Many of the demonstrations of requirement for normal cell function have been demonstrated in cell or tissue culture media rather than in the animal proper as a result of being able to achieve extremely low concentrations (Suttle and Jones, 1981). Overseas results have suggested that low Se status is associated with poor udder health (Weiss et al., 1997) usually measured in terms of somatic cell counts (SCC). To my knowledge only one trial in New Zealand has reported similar findings (Witchel, 1994) and these were inconsistent. Somatic cell counts were 235, 199 and 139 (Herd A) and 235, 112 and 191 (Herd B) when
supplemented with 0, 2 or 430 g intra-ruminal Permasal pellets which resulted in mean GSH-Px of 0.7, 15.0 and 29 kU/L (Herd A) and 1.5, 22.2 and 23.6 kU/L (Herd B). Only the effect of four pellets was significant in Herd A, and the effect of two pellets (Herd B) despite the fact that large numbers of animals were involved. An alternative conclusion would be that despite very low Se status the average SCC of these two herds was normal. This is also consistent with a general lack of abnormality of reproductive parameters in cattle in New Zealand with very low Se status or of response to supplementation (Wichtel, 1994; Clark et al, 1992) in contrast to overseas data. The potential importance of differences in feeding regimes for selenium requirement has been mentioned earlier, but the close and extended confinement practices in northern hemisphere systems undoubtedly expose the udder to both pathogens and increased risk of physical damage.

Addition of chromium to numeral supplements is a recent development. The supposed evidence for production benefit of chromium supplementation comes almost entirely from the work of one group in Canada. Quite large increases (up to 30%) in growth rates of weaner calves entering feedlot systems have been reported (Chang and Mowat, 1992; Moonsie-Shageer and Mowat, 1993). However, despite the use of large numbers of animals these differences have not been statistically significant, though this tends to be ignored in the discussion. These cattle are “heavily stressed,” often having been in transit for over two days. They are susceptible to a range of shipping related diseases including pneumonia, bovine viral diarrhoea, parainfluenza (PI3), infectious rhino tracheitis and usually receive multiple vaccines on arrival. Treatment with long acting antibiotics has shown more regular and reliable responses (Chang and Mowat, 1992). The situation should be monitored, however, because evidence is accumulating to suggest that chromium supplements may change the sensitivity of cells to insulin and cortisol, though whether this requires chromium to be given in organic forms is still unresolved (Kegley, et al, 1997). This is important in a New Zealand context because herbages are naturally rich sources of chromium.

Milk production responses (3 and 6% in two experiments) have been claimed for the addition of organic chromium (0.5 mg/kg feed DM) to the diets of dairy cows in late pregnancy and early lactation (Yang et al, 1996). Closer examination of the work shows that the responses “occurred” only in primiparous and not in multiparous cattle. Since there were only 6-9 animals in each group the improvements in performance could equally be explained by problems in randomization of animals to treatment. There is insufficient evidence to support a need for Cr supplements in New Zealand.

**Manipulation of acid-base balance**
During the last 30 years much attention has been focussed in the Northern Hemisphere on the need to provide pH balancing nutrients. The concept was developed in the poultry industry as dietary electrolyte balance (DEB).

The issues really fall into two sections:
- prevention of milk fever
- promotion of growth and productivity.

Of these, the only situation worthy of detailed examination is the prevention of milk fever.

The literature considers that acidic diets (diets which predispose to acidic rather than the conventional alkaline pH of ruminant urine) are those in which the dietary cation-anion balance (DCAB) measured as (potassium (K\(^+\)) + sodium (Na\(^+\)) – (chloride (Cl\(^-\)) + sulphate (SO\(_{4}\)\(^{2-}\))), in milli-equivalents (atomic weight ÷ valency)/ kg DM is below 100, and which produce urine with pH below 7.5.

The practice was initiated from the observations of Ender (1971) that incidence of milk fever decreased markedly in Europe with the switch from feeding hay to acid-preserved silages. Subsequently, a significant literature has accumulated (Figure 2) which has shown increasing incidence of milk fever as the DCAD balance shifts from negative to positive. In this work 50-80% incidence of milk fever has been observed at DAD values in excess of 250 m-equiv/kgDM.

These findings are backed by numerous studies (Block, 1994) which have shown that acid diets reduce incidence of milk fever by enhancing calving calcium absorption and bone calcium resorption, probably mediated by increases in production of calcium regulating hormones (Abn Damir et al. 1994).

The major contributor to the DCAD calculation is dietary K. In New Zealand's pastoral conditions during early spring herbage K concentrations are such that DCAD values in excess of 600 m-equiv/kgDM can be anticipated. There are two trials (one an uncontrolled farm trial) in the New Zealand literature, the results of which are shown in Figure 2. Wilson (1996) reduced the incidence of milk fever from 5% to zero by reducing DCAD of a pasture/maize silage diet from 250 to 165. The trial, however, used only 20 cows/treatment so one case made the difference. In the other trial pasture spraying, calculated to have reduced DCAD from 626 to 247m-equiv/kgDM, was associated with a reduction from 10.8% to 2.3% in incidence of milk fever cases (Todd, 1994). This was a farm trial and did not have any controls. The most striking aspect of these comparisons, however, is the comparatively low incidence of milk fever despite very high DCAD values in New Zealand pasture. There are clearly other very significant factors at work - possibly the excessive steaming up, and higher initial milk production and therefore higher Ca demand of northern hemisphere cattle. The work in New Zealand showing benefit is
based on extremely small numbers (Wilson, 1996). More recent overseas studies suggest there to be benefit in using Ca salts such as (CaCl$_2$) in gel or emulsion form because acidification *per se* may be ineffective if the diet is low in calcium (Goff & Horst, 1993; 1994; Oetzel, 1988). Pasture has an abundant supply of Ca; one simple approach may be to avoid the use of potassic fertilizers in an area in which cows are held during the calving period.

The concern about DCAD outside the calving period is much less defensible. A major protagonist for the practice of cation-anion balancing (Block, 1994) argues that “it appears logical to keep the DCAB highly positive (anionic) for lactating cows because these cows have a high metabolic rate and the cellular environment (therefore) appears to be acidic, necessitating higher dietary levels of Na$^+$ and K$^+$ relative to Cl$^-$.” It is perhaps relevant that Wilson (1996) found no significant effects on milk production, a conclusion generally obtained overseas.

![Figure 2: Relationship between dietary cation-anion balance of diets and incidence of milk fever. x, * represent New Zealand data and the remainder from conserved forage-grain based diets.](image)

Finally, rumen modifying agents such as sodium bicarbonate have been advocated to counteract (buffer against) low rumen pH and a “low milk fat” syndrome. This syndrome and practice is characteristic of animals offered diets comprising high levels of readily digestible carbohydrate. The normal fermentation of sugars and starches in the rumen yields short chain fatty acids which are absorbed and used as energy-yielding nutrients. When large quantities are rapidly fermented the acids temporarily depress rumen pH. The activity of rumen microorganisms which digest the cellulose in the forage component of the diet is sensitive to pH and falls when pH falls from the normal 6.5-7 to below 6.0. In these circumstances forage intake
can be depressed. The results of feeding bicarbonate supplements, even in high concentrate systems, is extremely variable. Carbonate supplements have little buffering capacity. Such improvements in fat yields, as have been observed, may have been due more to increased flow of digesta through the rumen so that more starch is digested post-ruminally with resultant change in the balance of fatty acids to glucose absorbed. Similar responses have been obtained with NaCl (Erdmann 1988). While responses to feeding NaHCO₃ have been reported in cows at pasture, this has been when these have been fed with significant levels of concentrate supplements (Chiy et al. 1993a). The case for such rumen modifiers in New Zealand conditions has not, in my view, been made.

Conclusions

- Hypo-mineralaemia (low mineral concentration in blood or tissue) does not mean that the animal is performing sub-optimally.
- An animal is only “at risk” if low intake persists. Will it?
- Direct transfer of information from overseas systems can be misleading for pastoral systems.
- Our diagnostic tools are still crude and in need of New Zealand-specific research.
- Many mineral nutrition “problems” vanish when animals are well fed and managed.
- Mineral supplementation is not a cure-all; it should be based on quantified responses.

References


Workshop summary

- If you don’t feed an animal their ME requirements on a daily basis you will have an immediate effect on production, and almost as quickly with a protein deficiency. This is not so with minerals - animals can store and regulate the mineral status in the body.
- Low mineral status in cattle does not mean that the animal is performing sub-optimally.
- The animal is only “at risk” if low intakes persist. Will it?
- Direct transfer of information from overseas systems can be misleading for the New Zealand pastoral systems.
- Our diagnostic tools are still crude and in need of New Zealand specific research.
- Many mineral nutrition “problems” vanish when animals are well fed and managed.
- Mineral supplementation not a cure-all, it should be based on quantified responses.
- However, mineral are cheap, so if feeding out supplements add mineral as an insurance policy if unsure - but be careful not to over-supplement as toxicity can occur.

Discussion summary

- At this stage there are no other diagnostic tools, other than bloods and lover samples out there to give accurate trace element status. It is more important however to monitor your whole system over the season and determine when you think “something” is happening. Bloods only give a “glimpse” of what is happening that day.
- There are a lot of mineral products on the market - organic and inorganic varieties. Both are adequate forms of mineral supplementation, although the organic form may be more efficiently absorbed. However, you will only get a response if there is a deficiency.
- It is important to work out your own mineral supplementation for your own farm - don’t rely on the sales person. Analyse the evidence they give you.
- Trace elements are a part of the overall supplementation system and should not be considered as a separate issue.
- There are a number of Canterbury farmers interested in carrying out trials on their own farms.

Take home message

Minerals are easy to sell. We need to be more critical of the “scientific data” used to promote these products. We also need to be smarter in diagnosing trace elements problems and become more efficient at supplementing minerals.

Future directives

We need accurate scientific data on trace element problems in New Zealand. Ideally there trails should be set up on-farm as it needs to be done on a large scale…any volunteers?