

Understanding Milking Cow Mineral Requirements

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Your committee set the scene for this paper by asking me to address the following questions:

- 1 What does a high producing cow really need?
- 2 Realistic costs of minerals.
- 3 Are chelated products really necessary?
- 4 Testing regime. How often and how much should we spend?
- 5 Do young stock require anything different?

I'm going to start by answering questions 1 + 5 together, and simply. Of course, the needs of the high yielding cow, in terms of g or mg of mineral/day, are much greater than those of the calf, but the dietary concentrations needed in their diet are essentially the very similar. This is because the much greater requirement for energy and protein of the high yielding dairy cow means that it has a much larger DM intake which carries in more mineral. Remember, mineral intake is dry matter (DM) intake x mineral concentration in DM, so that the higher mineral demands of the high producing cow are provided by the greater DM intake, provided she is well fed. Calculations from nutritional principles of the minimum dietary requirements for a range of minerals are given in Table 1 for a 200 kg calf growing at 1 kg/d and for a 500 kg dairy cow producing either 10 or 30 litres milk/d together with the range of concentrations observed in herbage in New Zealand. The data are taken directly from Grace, Knowles & Sykes (2010) and show remarkably consistent mineral requirement when expressed as dietary concentration. So neither the rapidly growing calf nor the very high yielding cow have particular dietary requirements when fully fed.

It is impossible to discuss mineral nutrition and requirements in general terms because each mineral has specific functions and regulation by the animal. The major minerals calcium and magnesium are probably of major interest to this audience because of the spectacular effect of failure of the animal to maintain tissue concentrations.

Calcium

There is ample Ca in ryegrass white clover (see Table 1). However, ruminants use a sophisticated endocrine system based on vitamin D metabolism to regulate Ca absorption to adjust the amount required for maintenance and milk production and to maintain Ca

concentrations in body fluids. When demand for Ca increases rapidly at calving this mechanism sometimes doesn't respond quickly enough, the concentration in body fluid falls, and you find the comatose cow. It is a temporary failure and for most the experience is that when detected early an intravenous and/or subcutaneous injection of calcium restores the cow and its own system usually very rapidly adjusts.

We do know, however, that there is a Mg requirement for components of the Ca control system and clinical evidence suggests that, as a prophylactic measure, whole herd Mg supplementation by pasture dusting or fertilizer application is considered beneficial.

So – the problem is not inadequate diet but an inadequate cow – temporary failure of its physiological regulatory mechanisms. Several approaches have been explored overseas to try to overcome this.

- (a) Feeding of ionic salts to reduce body pH and 'assist' Ca mobilisation from the skeleton, otherwise described as the DCAD (dietary cation-anion balance) dietary manipulation. While achieving some success in feedlot systems in the northern hemisphere, I have seen no evidence for its value in NZ. In fact, prediction of the incidence of hypocalcaemia on our spring pastures from the DCAD values of NZ pastures – from relationships seen in the northern hemisphere feedlot systems- suggest we should actually expect a much greater incidence than is the case in NZ. The fact that we don't suggests that other environmental factors are operating in its systems which predispose to hypocalcaemia which don't operate here. What it really shows up is the need to be cautious about extrapolating overseas data on mineral nutrition from overseas to Nz pastoral systems, a point I will return to later.
- (b) Feeding low Ca forages until just before calving to set up the vitamin D system; the problem is that NZ pastures have adequate Ca, with the possible exception of green-feed oats, but this approach is logistically difficult.
- (c) Large injections of Vitamin D; needs to be timed close to calving and is logistically difficult.
- (d) Pasture dusting as a prophylactic treatment during calving.

Magnesium

Hypomagnesaemic tetany is another dairy industry problem with spectacular and obvious clinical symptoms. In this case it occurs despite the fact that pasture, particularly pasture with high clover content, has ample Mg for the high yielding dairy cow Table 1. The problem is interference with absorption of dietary Mg by the high concentrations of K which are characteristic of highly digestible rapidly growing plant leaf material. As the plant matures and develops structural material and K concentrations fall and Mg absorption increases and risk of

hypomagnesaemic tetany recedes. As we get better at managing to maintain high pasture quality into the summer period this problem has probably been extended from very early lactation into mid-lactation and even into high quality autumn regrowths. Although Mg can be provided on a regular basis in drinking water this has not proved effective during early lactation because cows don't drink much at that time. A high yielding cow consuming 15-18 kg DM/d of high quality pasture will have an intake of 100-120 L water/d, well in excess of its requirement for milk production and rarely approaches the trough. The only safe way, unless Mg salts can be included in concentrate feeds, is the dusting of pasture with calcined magnesite or causmag (MgO or MgCO₃ heated with lime at high temperature) at 500g/cow/week. Individual drenching with 10-15 g/Mg/d as the magnesium sulphate or chloride is a possibility, but labour intensive. Preventative treatment is by far the best approach, and can require supplementation from 3-4 weeks before to 12 weeks after calving. Blood testing is not a useful approach though a periodic blood test of older cows can be helpful in deciding when to stop treatment: an average of 0.6 mmol Mg/L blood would indicate safety to cease supplementation.

Trace elements

While deficiencies of the major elements, calcium and magnesium, are universal, trace element deficiencies are generally specific to soil type. For example cobalt deficiency is only likely on soils derived from acid igneous rocks such as granite; copper deficiency on coastal sands and silts and on peaty soils; selenium deficiency can be anticipated on soils of volcanic origin with high rainfall and high pH. Not surprisingly therefore examples of these deficiencies have historically been seen in New Zealand and were originally diagnosed and treatments developed here. However, the situation is more complex than this. The ability to accumulate trace elements varies with plant species. Reports of cobalt and copper deficiencies are a feature of the early literature of agricultural development in New Zealand and severe deficiencies were recorded. However, during land development less productive grass species such as *Agrostis tenuis* (browntop), were replaced with ryegrass-white clover species which are better accumulators of both copper and cobalt and occurrence of deficiencies has reduced. As a consequence the historical record can be misleading and the evidence is that deficiencies of these elements are less likely than the early record suggests. The opposite is the case for selenium; in this case the less productive browntop is a much better accumulator of selenium than ryegrass – white clover. Land development in this case lead to increase rather than decrease an occurrence of the deficiency which was really only diagnosed and effectively treated relatively recently (1960's) and, as a consequence is still probably the most significant trace element problem today.

Can some minerals limit the uptake of others?

The question needs to be broadened because whereas uptake of minerals can be limited by other minerals, significant interactions between minerals and other nutrients can occur during metabolism as well as during absorption.

Mineral – mineral interactions on absorption

The most important antagonistic effects of minerals on mineral absorption relate to the absorption of magnesium and copper. We have discussed the case of the depressing effect of potassium on magnesium absorption previously: fortunately the antagonism is not complete and can be overcome by heavy dusting of pasture with magnesium. In the case of copper high levels of dietary sulphur (S) and molybdenum (as molybdate-MoO₄) lead to the formation of thio-molybdates (MoS¹⁻⁴) in the rumen which combine with copper making it unavailable for absorption. Increasing dietary copper orally is unhelpful because the copper supplement suffers the same fate as dietary copper and is complexed in the rumen and unavailable for absorption: under these circumstances use of orally administered Cu-oxide-wire particles, which lodge in the acid-secreting fourth stomach and release Cu beyond the rumen, or Cu injections are the only way to overcome deficiency. Copper shares transport mechanisms with Fe and Zn. Fe intake with soil can reduce Cu status as has also been shown during use of ZnO for control of facial eczema. In this case choice of an injectable Cu supplement will overcome this problem.

Mineral – other nutrient interactions

The number of potential interactions between minerals as a consequence of their involvement in essential metabolic activities of cells is huge. An example is the involvement of copper, selenium and vitamin E in providing 'anti-oxidant' capabilities which prevent the normal metabolic activities of cells from causing self-destruction. This is especially so with certain cells of the immune response which generate oxidising peroxides to destroy foreign bodies, but which would destroy the cell itself if excess is not rapidly removed, or is simply generated by cells with high metabolic rate such as muscle cells. Copper- and selenium-dependent peroxidases deal with these waste products while vitamin E, because of its solubility in lipids provides similar protection in the lipid cell membrane. The most significant interaction is that between Se (in the enzyme glutathione peroxidase (GSH-Px)) and vitamin E; in New Zealand pasture-based animals have a high intake of vitamin E and can maintain normal productivity with relatively low levels of GSH-Px in cells. In systems in the USA which used conserved forages and concentrates vitamin E levels are often much lower unless the diet is fortified. In addition, fats are often added to diets to increase energy intake which further increases the oxidant load on the animal. Recommended blood levels of GSH-Px for diagnostic

purposes are 10x higher in USA than those at which responses have been observed in NZ. There has been confusion in New Zealand as a consequence. The message has to be that, given the potential for such nutrient interactions diagnostic tools and recommendations for supplementation must be appropriate for local conditions.

Are chelated products necessary?

The simple answer is, emphatically NO. In the most recent authoritative review of the subject, Suttle (2010) states ‘There is a popular misconception, sustained and encouraged by aggressive marketing, that (cheaper) inorganic trace mineral supplements are intrinsically of less value to livestock (and man) than the natural forms in which elements are available in feeds, and are less available than synthetic organic complexes containing those elements Evidence of consistently superior value in a robust test of availability of organic complexes has yet to appear in a reputable, peer-reviewed journal, with the singular exception of iron in non-ruminants’. This is a view I strongly endorse. There are reports which suggest the bioavailability of organic supplements to be greater than that of simple mineral supplements (for example selenised yeast (Juniper et al 2006)) but there are none which suggest that inorganic supplements are less effective in correcting deficiency. Remember, mineral supplements are high margin products which have been marketed very successfully. The ability to add “superior” availability to the generally bland claims such as “overcoming low levels can assist with general good thrift, improved milk production, improved appetite, improved fertility” is as much a marketing ploy as a genuine advance in ability to correct mineral deficiency.

If the question was asked ‘which are the most effective mineral supplements?’ the answer would be those which provide an economic response to supplementation which implies those which deliver sufficient mineral to overcome a diagnosed mineral deficiency. In some cases the use of a complexed product is the best way to provide sustained provision of a mineral. But this issue is now part of the next question.

Testing regime. How often and how much should we spend?

For the major elements – Ca and Mg – a testing regime to prevent hypomagnesaemia or hypocalcaemia around calving is a waste of time. The assumption has to be that there will be some susceptible animals in the herd (~ 5% under NZ conditions - Roche & Berry, 2006): observation and treatment will be needed but such preventative measures as are reasonably available in pasture dusting with magnesium salts is still the best approach. Heavy use of N or K fertilizers are factors to consider when assessing risk. Fortnightly testing for of plasma magnesium of 20 herd members as indicated earlier would be useful to determine safety to stop

magnesium dusting, for example when all samples are > 0.6 mM Mg or a mean of 0.7 mM Mg, but cow behaviour and previous farm history are probably sufficient.

The trace element testing regimes are valuable where soil series and plant communities suggest deficiency may be present. There are, however, some issues that need to be more clearly understood. Minerals are needed by animals either as co-factors or components of enzyme systems and their specific essentiality derives from the fact that they have very powerful chemical attributes. These attributes which power metabolism in cells are, like the nuclear power plants in Japan, benign and useful when under control, but dangerous (toxic) when out of control or present in excessive quantities. Because of this animals have evolved sophisticated mechanisms to control the amount of mineral in the body. How this is achieved varies with trace element but which determines the best testing regime and interpretation of data. Consider three possible pools of the trace-element in the body.

1 *Enzyme pool*

This is the critical pool. Only when this pool is depleted do loss of function and therefore performance occur. It is an enzyme-associated pool within the cells intimately involved in metabolic reactions and, while not impossible to measure, is too costly and difficult to use in routine diagnosis. When analysis shows it is depleted it generally also the case that supplementation is too late to avoid loss of productivity.

2 *Transport pool*

This is the pool of mineral in the blood which enables transport of the element around the body – from site of absorption or storage in the body to the critical enzyme pool. This is the easiest and cheapest to sample: the problem is that there is considerable redundancy; critical enzyme pool levels can be maintained over a wide range of blood concentrations. There has been a fashion by the industry to equate the maximum concentrations observed as the ‘normal’ concentration, when really they really represent the maximum concentration permitted by the animal’s regulatory control systems. Of course this suits the mineral sales industry to suggest you top up your animals to these levels but it doesn’t necessarily translate to a production response when supplements are applied. Examination of the literature for copper, selenium, cobalt and iodine supplementation reveals much greater success in lifting blood concentrations back to ‘maximum’ levels than in achieving economically positive production responses.

3 *Storage pools*

Several elements are stored in the body and can be depleted by the animal during a period of dietary deficiency to maintain the transport and enzyme pools. As a consequence the animal will often manage a seasonal period of low intake without problems provided it is not too severe or persistent. The most extreme case is that of Iodine for which more than

a year's supply can be stored in the thyroid gland; for copper liver copper storage can cover a deficiency of 3-6 months and for cobalt and selenium up to 3 months. For copper, and to a lesser extent cobalt measurement of liver concentration allows estimation of the size of these stores and gives a better indicator of status of body reserves than plasma. Selenium is incorporated into the enzyme glutathione peroxidase (GSH-Px) at the time of cell formation. Measurement of GSH-Px gives a better indicator of the history and status of selenium than measuring selenium itself.

What does this mean?

It really means that the only way to establish the economic value of trace element supplementation on your property is to consider:

- 1 is your property on a soil series on which deficiency can be anticipated?
- 2 is your choice of forage plants likely to enhance or mitigate against deficiency?
- 3 If these indicate reason for concern, have appropriate animal testing carried out at critical times of the year – e.g. for copper, selenium and cobalt during late spring when tissue reserves would be expected to be lowest. If the results indicate of less than “normal” status-
- 4 design an effective supplementation regime and run it for at least 12 months i.e. treat half the herd with an effective supplement and leave the remainder as controls. Because our industries do not use concentrate supplements, which would allow us to carry in minerals at negligible, the most effective supplements are those given directly to the animal in sustained release form – for example selenium as barium selenate; copper as oral copper oxide needles or calcium copper edetate or copper glycinate injections; cobalt as, as injections of sustained release microencapsulated vitamin B₁₂ or intra-ruminal cobalt oxide bolus; iodine as iodised oil injection. While these preparations are more expensive than oral supplements they have effectiveness, and will ensure an effective long term supply. Their big advantage is that they allow a simple to manage but true evaluation of need on farm, by allowing treated and untreated animals to graze together. Details are provided in Grace, Knowles & Sykes (2010) and veterinary advice should be sought. If production responses are observed a true cost benefit can then be determined. For some, continuation with individual treatment will be needed, but, for whole herd treatment, cheaper alternatives such as topdressing with fertiliser application, for example selenium prills, can be the routine. In most cases it will provide peace of mind that trace element status is consistent with optimum productivity and you can have an informed dialogue with the trace element salesman.

Realistic cost of mineral supplements

Mineral salesmen rely on farmers buying minerals in much the same way as people buy vitamin pills. You think you need them and feel they do some good, and they don't seem to do any harm. If you stop taking them maybe you'll go down with a cold, or something worse. So the few cents a day on the vitamin pill seems like a good investment. Any supplement used which isn't necessary is an expensive supplement. Short term use of an expensive supplement, as described above, to provide you with the evidence that you don't need to use a cheap supplement will prove to be a good investment which saves money in the long run. The answer, do the analysis of soil type, consider the limitations of the plant species you are using and, if in doubt, demonstrate need for supplementation. You are then in a position to answer this last question.

References

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- Juniper D T, Phipps R H, Jones A K and Bertin G. 2006. Selenium supplementation of lactating dairy cows: effect on selenium concentration in blood, milk, urine, and faeces. *Journal of Dairy Science* 89:3544-3551.
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Table 1: Recommended dietary mineral concentrations for a 200 kg calf growing at 1 kg/d and a 500 kg cow producing 30 L milk/d (from Grace, Knowles and Sykes, (2010)).

Element	Calf	10 L/d	30 L/d	NZ pasture
Ca (g/kg DM)	4.4	3.1	3.2	3.7-14.2
Mg (g/kg DM)	0.4-0.9	0.75-1.4	0.8-1.4	1.0-6.0
P (g/kg DM)	2.5	2.5	2.3	4.0-5.0
Cu (mg/kg DM)	6.5	9.2	9.2	3.5-18.0
Co (mg/kg DM)	0.06	0.06	0.06	0.11-0.24
Se (mg/kg DM)	0.032	0.037	0.035	0.005-0.070
I (mg/kg DM)	0.18?	0.18?	0.18?	0.1-0.8