DETERMINING THE MINERAL REQUIREMENT OF DAIRY CATTLE

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These notes will not deal with the physiological function of the minerals but will deal with meeting the dietary requirements. Certain aspects unique to dairy will be discussed as well as physiological factors affecting absorption.

DIETARY REQUIREMENTS - THE FACTORIAL APPROACH

When possible the requirement of the tissue for each mineral will be discussed. The dietary requirement is dependent on the amount of dietary mineral that is absorbed into the tissues. This means that if we can determine the requirement for maintenance (endogenous fecal loss and urine loss), pregnancy (fetus plus placenta), lactation (milk content), and growth (mineral content of the tissue gained) we can determine the tissue needs. If we know the fraction of dietary mineral absorbed from the diet the requirement of the cow can be described by:

$$\text{Dietary Requirement} = \frac{\text{Maintenance + Pregnancy + Growth + Lactation}}{\text{Absorption co-efficient}}$$

The values used for maintenance, pregnancy, growth, and lactation are those used in either the 1980 Agricultural Research Council's Nutrient Requirements of Ruminant Livestock or the 1989 National Research Council's Nutrient Requirements of Dairy Cattle. These values vary little and really are readily measured and pretty reliable.

The real challenge is to determine the co-efficient of absorption for the minerals across a wide variety of diet and/ or supplemental sources. Those factors affecting this number will be described as much as possible so that you can use reasonable judgement to decide when you are dealing with a situation that calls for more or less mineral than standard texts on mineral nutrition would suggest. An excellent reference to assist in deciding relative bioavailability of minerals from feedstuffs and supplements is Bioavailability of nutrients for animals, edited by C. Ammerman, D. Baker and A. Lewis.

A section follows this on diagnostic aids to help you decide when a mineral problem exists. An excellent resource for diagnostic evaluation of mineral deficiencies is Mineral Levels in Animal Health, by R. Puls.

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CALCIUM (Ca)

Since Ca is so essential for life vertebrates have evolved an elaborate system to maintain Ca homeostasis. This system attempts to maintain extracellular Ca concentration constant by increasing Ca entry into the extracellular fluids whenever there is a loss of Ca from the extracellular compartment. When Ca loss exceeds entry hypocalcemia can occur. If Ca enters the extracellular compartment faster than it leaves hypercalcemia can occur which can lead to soft tissue deposition of Ca.

Ca leaves the extracellular fluids during bone formation, as digestive secretions, sweat, and urine. An especially large loss of Ca to milk occurs during lactation. Ca lost via these routes can be replaced from dietary Ca, from resorption of Ca stored in bone, or by resorbing a larger portion of the Ca filtered across the renal glomerulus, ie. reducing urinary Ca loss.

Ultimately dietary Ca must enter the extracellular fluids to permit optimal performance of the animal. Ca absorption can occur by passive transport between epithelial cells across any portion of the digestive tract whenever ionized Ca in the digestive fluids directly over the mucosa exceeds 6 mM. These concentrations are commonly reached when young animals are fed milk. In non-ruminant species, studies suggest that as much as 50% of dietary Ca absorption can be passive. It is unknown how much passive absorption of Ca occurs from the diets typically fed ruminants but the diluting effect of the rumen would likely reduce the degree to which passive Ca absorption would occur.

Active transport of Ca is the second route for Ca absorption, and is especially important when diets are not high in Ca. Active transport of Ca is controlled by 1,25-dihydroxyvitamin D, the hormone derived from vitamin D. Vitamin D, produced within the skin or provided in the diet, is converted to 25-hydroxyvitamin D in the liver and can be further metabolized to 1,25-dihydroxyvitamin D in the kidneys. Parathyroid hormone indirectly stimulates intestinal Ca absorption because it is the primary regulator of renal production of 1,25-dihydroxyvitamin D

DIETARY Ca REQUIREMENTS

Maintenance: For non-lactating cattle the absorbed Ca required is .0154 g / kg live wt. For lactating animals the maintenance requirement is increased to .031 g / kg Lwt
-increased dry matter intake increases intestinal Ca secretion during digestion.

Growth: 17 g Ca/ kg live wt gained if live wt is less than 200 kg, 13 g Ca if live wt is between 200 and 300 kg, 8 g Ca if live wt is between 300 and 450 kg, and 5 g Ca if live weight is greater than 450 kg.

Pregnancy - Fetal skeletal calcification is especially great in the last weeks before parturition. The absorbed Ca required to meet the demands of the uterus and conceptus increases exponentially from 1 g / day at day 190 of gestation to about 10 g Ca/d at parturition.

Lactation - The absorbed Ca / kg 4% FCM is 1.22 g for Holstein, 1.45 g for Jersey, and 1.37 g for other breeds.
Colostrum requires 2.1 g absorbed Ca / kg produced.

ABSORPTION COEFFICIENT

To truly determine the availability of Ca from a feedstuff, the animals being tested should be fed less total dietary Ca than the amount of absorbed Ca required to meet their needs. This will ensure that intestinal Ca absorption mechanisms are fully activated so that the animal will absorb all the Ca from the feedstuff that it possibly can. Few studies fulfill this requirement, thus it is likely that the published studies have underestimated the availability of Ca in many cases.

Previous NRC publications have determined a single efficiency of absorption of dietary Ca irregardless of the source of Ca or the physiologic state of the animal. This absorption coefficient was .38 in the 1989 dairy NRC and .45 in the 1978 NRC based on the average proportion of Ca absorbed during a variety of trials. The decision to utilize .38 as the Ca absorption coefficient was based largely on a summary of 11 experiments with lactating dairy cows in which the average percentage of dietary Ca absorbed was 38. In the majority of these 11 experiments the cows were fed diets supplying Ca well in excess of their needs placing the cows in positive Ca balance by as much as 20 - 40 g Ca / day. In 3 of the experiments the cows were in negative Ca balance and the percentage of dietary Ca absorbed was still below 40%. In those experiments alfalfa and/or brome hay were supplying the dietary Ca.

The 1980 ARC chose 0.68 as the coefficient of absorption for Ca; a coefficient considerably higher than the estimate of other committees that had examined dietary Ca requirements of cattle but one that may be closer to the truth.

A SINGLE COEFFICIENT MAY NOT BE APPROPRIATE

Forage Ca availability - around 30% available for absorption judging the literature on alfalfa Ca availability Martz et al, 1990). Oxalate in forages seems to be the culprit.

Concentrate Ca availability - around 60% available for absorption

Ca within mineral supplements is generally more available than Ca in forages and common feedstuffs. Theoretically the factor limiting mineral Ca availability is the solubility of the Ca in the mineral source. Most mineral sources of calcium are at least 70% available for absorption (Hansard, et al., 1957).

In early lactation nearly all cows are in negative Ca balance. As feed intake increases and Ca intake increases most cows go into positive Ca balance about 6-8 wks into lactation. Cows in the first 10 days of lactation are at greatest risk of being in negative Ca balance and some are subclinically hypocalcemic throughout this period. There is no evidence to demonstrate that negative Ca balance in early lactation was detrimental to the cow provided plasma Ca concentration remained normal, ie. bone was being used to ensure adequate entry of Ca into the extracellular Ca pool. The bone would be replaced in later lactation.

The effects of Ca:Phos ratio on absorption of Ca and Phos was once felt important but many recent studies suggest that Ca: Phos ratio is not critical, unless the ratio is greater than 7:1 or less than 1:1 .

Some studies suggest that high fat diets increase the dietary Ca requirement through the formation of Ca soaps (Oltjen 1975) however the available data do not justify a factor to increase dietary Ca when fat is added to the diet.

SYNDROMES OF SPECIAL CONCERN

Milk fever in dairy cows.

An important determinant of milk fever risk is the acid-base status of the cow at the time of parturition. Metabolic alkalosis impairs the physiological activity of PTH so that bone resorption and production of 1,25-dihydroxyvitamin D is impaired reducing the ability to successfully adjust to the Ca demands of lactation. Evidence suggests that metabolic alkalosis induces conformational changes in the PTH receptor which prevent tight binding of PTH to its receptor. Cows fed diets that are relatively high in K or Na are in a relative state of metabolic alkalosis which increases the likelihood that they will not successfully adapt to the Ca demands of lactation and will develop milk fever. These cows exhibit a temporary pseudohypoparathyroidism at parturition. The parathyroid glands recognize the onset of hypocalcemia and secrete adequate PTH. However the tissues respond only poorly to the PTH, leading to inadequate osteoclastic bone resorption and renal 1,25-dihydroxyvitamin D production.

Since metabolic alkalosis is an important factor in the etiology of milk fever it is important to prevent metabolic alkalosis. Dry cow diets that are high in K and / or Na alkalinize the cow's blood and increase the susceptibility to milk fever. Adding Ca to practical prepartal diets does not increase the incidence of milk fever. The landmark studies of Ender et al. 1967 demonstrated that addition of anions to the prepartal diet could prevent milk fever. Ammonium, Ca and Mg salts of Cl and sulfate have been successfully used as acidifying anion sources. Cl salts are more acidogenic than sulfate salts. Hydrochloric acid has also been successfully utilized as a source of anions for prevention of milk fever and is the most potent of the anion sources available

A second common cause of hypocalcemia and milk fever in the periparturient cow is hypomagnesemia. Low blood Mg can reduce PTH secretion from the

parathyroid glands causing temporary hypoparathyroidism and can alter the responsiveness of tissues to PTH by inducing conformational changes in the PTH receptor, again causing temporary pseudohypoparathyroidism.

PHOSPHORUS (PHOS)

Phos is primarily absorbed in the small intestine via an active transport process that is responsive to 1,25-dihydroxyvitamin D. Intestinal Phos absorption efficiency can be upregulated during periods of Phos deficiency as renal production of 1,25-dihydroxyvitamin D is directly stimulated by very low plasma Phos. Plasma Phos concentrations are well correlated with dietary Phos absorption. Phos absorbed in excess of needs is excreted in urine and saliva.

Parathyroid hormone, secreted during periods of Ca stress, increases renal and salivary excretion of Phos, which can be detrimental to maintenance of normal, blood Phos concentrations. This is one reason that hypocalcemic animals tend to become hypophosphatemic. Parathyroid hormone could conceivably increase blood Phos concentration since it stimulates bone mineral resorption. However, parathyroid hormone is secreted in response to hypocalcemia, not hypophosphatemia. This means that Phos homeostasis and Ca homeostasis are sometimes at odds.

Salivary secretions remove between 30 and 90 g Phos from the extracellular Phos pool each day depending, with higher amounts secreted when dietay Phos is high. Salivary Phos secretions supply rumen microbes with a readily available source of Phos which appears necessary for cellulose digestion. Most, but not all, of the salivary Phos secreted is recovered by intestinal absorption.

Rumen microbes are able to digest phytic acid so that much of the phytate-bound Phos, the major form of Phos in plants, is available for absorption in ruminants.

FACTORIAL PHOS REQUIREMENT

Maintenance: 1989 NRC- 1.43 g / 100 kg Bwt = 8.6 g for 600 kg cow
Rest of the world - uses 1.2 g P / g DM intake
- for a cow eating 20 kg DM / day = 24 g / day.

Pregnancy: increases exponentially
- from 1.5 g / day at Day 190 to 6 g / day just before calving.

Growth: Young animals require more Phos / kg BWt gained than older animals as skeleton forms.
Varies from 9 when young to 6 when nearly an adult g Phos /kg Live Wt gained.

Lactation: = 0.9 g Phos / kg 4% FCM milk

Coefficient of Absorption for Phos:

1989 NRC - assumes 50% of dietary P absorbed
1978 NRC - assumed 65% of dietary P absorbed
Rest of the world- between 60 and 75% of diet P absorbed
Recent literature suggests about 60% of feedstuff P is absorbed and about 75-80% of mineral P is absorbed.

Are we over-feeding P (Wu and Satter, 1998)? Who cares? Environmentalists do and ultimately the farmer will. We can do a better job.

PHOS DEFICIENCY

Moderate chronic hypophosphatemia with plasma Phos between 0.64 and 1.3 mmol/L or 2 and 4 mg/dl. is generally recognized only as animals that perform poorly. Growth and fertility are impaired. With more severe hypophosphatemia, the performance of the animals becomes very poor and feed intake of the animals is depressed. The reduction in feed intake is often accompanied by pica with a particular desire for soil, flesh, and bones. Pica can cause problems for Phos deficient animals. Outbreaks of botulism in cattle in South Africa and other parts of the world, where phosporus deficiency is endemic, have been traced to consumption of carcasses of wild animals that had died on the veldt and contained toxin as a result of the growth of Clostridium botulinum during putrefaction.

Severe - Recumbency and paresis observed if plasma Phos concentrations decrease below 0.3 mmol/L or 1 mg/dl.

RICKETS AND OSTEOMALACIA

Acute Hypophosphatemia in ruminants (Downer cows).

Beef cows fed a diet marginal in Phos will have a chronic hypophosphatemia of 0.6 - 1.1 mmol/L or 2-3.5 mg/dl. In late gestation plasma Phos can decline precipitously as the growth of the fetus accelerates and removes substantial amounts of Phos from the maternal circulation. These animals often become recumbent and are unable to rise, though they appear fairly alert and will eat feed placed in front of them. Cows carrying twins are most often affected. Plasma Phos concentration in these recumbent animals is often less than 0.3 mmol/L or 1 mg/dl. The disease is usually complicated by concurrent hypocalcemia, hypomagnesemia, and in some cases hypoglycemia (see Pregnancy Toxemia).

At the onset of lactation the production of colostrum and milk draws large amounts of Phos out of the extracellular Phos pools. This alone will often cause an acute decline in plasma Phos levels. In addition if the animal is also developing hypocalcemia, parathyroid hormone will be secreted in large amounts, which increases urinary and salivary loss of Phos. Cortisol, secreted around parturition, may further

depress plasma Phos concentrations. In dairy cows, plasma Phos concentrations routinely fall below the normal range at parturition and in cows with milk fever plasma Phos concentrations are often between 0.3 and 0.6 mmol/L or 1 and 2 mg/dl. Plasma Phos concentrations usually increase rapidly following treatment of the hypocalcemic cow with intravenous Ca solutions. This rapid recovery is due to reduction in parathyroid hormone secretion reducing urinary and salivary loss of Phos, and resumption of gastrointestinal motility accompanied by increased plasma concentrations of 1,25-dihydroxyvitamin D which allows absorption of dietary Phos and reabsorption of salivary Phos secretions (Goff, 1998).

Some animals developing acute hypophosphatemia do not recover normal plasma Phos concentration. This is sometimes the case in cows that are classified as "downer cows". This syndrome often begins as milk fever but unlike the typical milk fever cow, plasma Phos remains low in some of these cows despite successful treatment of the hypocalcemia. Protracted hypophosphatemia in these cows appears to be an important factor in the inability of these animals to rise to their feet, but why plasma Phos remains low is unclear.

MAGNESIUM (Mg)

Despite the importance of Mg there is no hormonal mechanism concerned principally and directly with Mg homeostasis. The kidneys play a key role in maintaining Mg homeostasis, but only under conditions of hypermagnesemia. If dietary Mg is absorbed in excess of needs plasma Mg concentration rises above the renal threshold for reabsorption of Mg and the excess is excreted into the urine. The renal threshold for Mg, ie. the plasma Mg concentration at which all Mg filtered across the glomerulus is reabsorbed is 0.75 -0.90 mmol/L or 1.8 - 2.2 mg/100 ml.

Plasma Mg concentrations below these levels indicate that dietary Mg absorption is not sufficient and little or no Mg will be detected in urine. Parathyroid hormone, released in response to hypocalcemia, raises the renal threshold for both Ca and Mg. The result is that during hypocalcemia plasma Mg concentrations will increase if dietary Mg absorption is adequate. This is often the case in cows suffering from milk fever. If plasma Mg is below 0.75 mmol/L or 1.8 mg/dl (suggesting inadequate dietary Mg absorption) raising the renal threshold further will not increase plasma Mg (Goff, 1998).

Bone is not a significant source of Mg that can be utilized in times of Mg deficit, as bone resorption occurs in response to Ca homeostasis, not Mg status. Maintenance of normal plasma Mg concentration is nearly totally dependent on a constant supply of dietary Mg.

Mg is absorbed primarily from the ileum and colon of monogastric animals and young ruminants. Mg absorption is by passive absorption and is therefore dependent on the concentration of Mg ions in the digesta. As the rumen and reticulum develop these organs become the main, and perhaps the only, site for Mg absorption in adult ruminants. In adult ruminants the small intestine is a site of net secretion of Mg.

HYPOMAGNESEMIC SYNDROMES OF CATTLE AND EWES

Hypomagnesemic tetany is most often associated with beef cows and ewes in early lactation grazing lush pastures high in K and nitrogen and low in Mg and Na. This is the most common situation and it is often referred to as Grass Tetany, Spring Tetany, Grass Staggers or Lactation Tetany. Mg deficiency occurs most often in spring or fall when pastures are growing at maximal rates, and is most common in grazing lactating ruminants as milk production removes 0.15 g Mg from the blood for each liter of milk produced. Ewes suckling more than one lamb and higher producing cows are at greatest risk. Mg must be constantly ingested, as it cannot be mobilized from body tissues to maintain normal plasma Mg concentrations. Conditions associated with hypomagnesemia as a result of feed restriction include transport over long distance (Transport Tetany) or sudden exposure to inclement weather. Cows can also develop hypomagnesemia in late gestation that is often associated with and complicated by inadequate energy intake. This syndrome is sometimes referred to as winter tetany and is seen in animals turned out in winter to feed on crop residues such as corn stalks or straw. Animals grazing wheat pasture (Wheat Pasture Tetany) or other early growth cereal forages can develop hypomagnesemia with concurrent severe hypocalcemia resulting in a clinical picture that more closely resembles milk fever. Hypomagnesemia can also occur in calves, especially if fed only milk or milk replacer beyond the first two months of age (Milk Tetany).

DIETARY MG REQUIREMENT - FACTORIAL APPROACH

Maintenance:	Fecal endogenous Mg loss = 3 mg/kg BW for adult cattle and 2 mg / kg BW for calves < 100 kg Obligate urinary loss of Mg is negligible.
Growth:	Tissue Mg content is 0.45 g / kg
Pregnancy:	about 0.33 g / day.
Lactation:	Colostrum contains about 0.4 g Mg /kg Milk contains about 0.12-0.15 g Mg / kg

COEFFICIENT FOR ABSORPTION OF MG

Mg deficiency is a common problem in ruminants, therefore some detail on Mg metabolism in ruminants is presented. Mg absorption from the rumen is dependent on the concentration of Mg in solution in the rumen fluid and the integrity of the Mg transport mechanism that is a Na-linked active transport process (Martens and Gabel 1986).

The average coefficient for absorption of Mg from a wide variety of natural feedstuffs fed to ruminants averaged .294 with a Standard deviation of .135.

The soluble concentration of Mg in rumen fluid is dependent on:

1. Dietary Mg content. Low Mg forages and inadequate supplementation will keep soluble Mg content low. Cool weather, common in spring and fall when pastures are growing rapidly reduces plant tissue uptake of Mg as does K fertilization of pastures.

2. The pH of the rumen fluid greatly affects Mg solubility. Mg solubility declines sharply as rumen pH rises above 6.5. Grazing animals tend to have higher rumen pH because of the high K content of pasture and the stimulation of salivary buffer secretion associated with grazing. Heavily fertilized, lush pastures are often high in non-protein nitrogen and relatively low in readily fermentable carbohydrates. The ability of the rumen microbes to incorporate the non-protein nitrogen into microbial protein is exceeded and ammonia and ammonium ion build up in the rumen increasing rumen pH. When high grain rations are fed rumen fluid pH is often below pH 6.5 and Mg solubility is generally adequate.

3. Forage can often contain 100-200 mmol/kg of unsaturated palmitic, linoleic, and linolenic acids which can form insoluble Mg salts. Plants also can contain trans-aconitic acid or citric acid. A metabolite of trans-aconitic acid, tricarballylate can complex Mg and is resistant to rumen degradation - but its role in hypomagnesemic tetany is unclear.

MG TRANSPORT ACROSS THE RUMEN EPITHELIUM

The major factor affecting Mg transport across the rumen epithelium is high dietary K which can reduce the absorption of Mg. High K concentrations in the rumen fluid cause the apical membrane of the rumen epithelium to depolarize, reducing the transepithelial membrane electrical potential responsible for propelling rumen fluid Mg into the blood (Martens, 1998).

Mg oxide is the most widely used inorganic source of Mg for ruminant diets. The coefficient of absorption for Mg from inorganic sources should be .50 based on Mg oxide.

Mg in magnesite and dolomitic limestone should be considered unavailable when formulating dairy rations. Mineral sources of Mg such as Mg oxide are poorly soluble at normal rumen pH. Mg sulfate and Mg Cl are much more soluble and available for absorption!

Because K can have such a large effect on Mg absorption the coefficient for absorption of Mg should be decreased when high K is present in the diet. Greene et al (1983) working with sheep found that the apparent absorption of Mg decreased linearly by 5.7% for every percentage increase in dietary K above 0.6% K.

SODIUM (Na)

Deficiency symptoms: Plants contain only small amounts of Na, leaving herbivores at risk of becoming Na deficient if salt is not added to the diet. Na deficient animals develop an intense craving for salt leading to pica with licking and chewing of various objects. Prolonged Na deficiency leads to an unthrifty animal with rough haircoat, haggard appearance, and poor growth and productivity. Cows will produce less milk.

Toxicity symptoms: Animals can tolerate very high levels of dietary salt if water is provided and the kidneys are functioning. They simply excrete the excess via the kidneys. High dietary salt (Na Cl) will reduce feed intake in animals. Grain intake in ad lib fed cattle can be limited by including 4-5% salt into the ration.

CHLORIDE (Cl)

Dietary Cl is absorbed with at least 80% and closer to 100% efficiency. The kidneys excrete Cl that is in excess of that needed to produce stomach acid and intestinal secretions, sweat, and to maintain acid-base balance in the animal. Often Cl anions accompany the movement of Na rations.

Deficiency symptoms: Cl deficiency can result in metabolic alkalosis and hypovolemia. Less severe deficiency can cause lethargy and poor performance. It rarely occurs if salt is fed. If salt is not fed Na deficiency will generally occur long before Cl deficiency.

Toxicity symptoms: Feeding high amounts of Cl that are unaccompanied by Na or K (for example, feeding ammonium Cl or Ca Cl) will induce metabolic acidosis which can be life threatening. Fed in high amounts it can have the same toxic effects as Na in terms of increasing the osmolarity of the blood and cerebrospinal fluids.

POTASSIUM (K)

Nearly all of the dietary K is absorbed. The kidneys excrete the excess absorbed K. High blood K concentration (hyperkalemia) can directly stimulate secretion of aldosterone by the adrenal enhancing renal secretion of K.

Dietary K can enter the extracellular fluids very rapidly following a meal, while the kidney will take several hours to excrete the excess K. Gastrointestinal secretion of K can work with the kidneys to help prevent hyperkalemia but it is the intracellular uptake of K following a meal that helps buffer blood K concentration. This intracellular uptake of K is mediated by insulin, which is secreted in response to hyperglycemia induced by a meal or in response to elevated plasma K concentration. Insulin increases the activity of the Na-K ATPase pump, particularly in the liver and skeletal muscle increasing uptake of K by these cells. Under most conditions hypokalemia is corrected by reducing aldosterone secretion. However if dietary K is inadequate hypokalemia may not be corrected.

Deficiency: When total body K content is below normal the animal is suffering from K depletion. When plasma K concentration is below normal the animal is hypokalemic. Hypokalemic animals are not always K depleted, and animals with low total body K stores can have normal plasma K concentrations.

Total body depletion of K leads to generalized muscle weakness. Hypokalemia, whether produced by redistribution or total body depletion, is more life threatening. Hypokalemia adversely affects the heart - slow repolarization of the ventricles. Hypokalemia also interferes with insulin secretion upsetting carbohydrate metabolism.

Excessive K: Milk fever of dairy cattle - The K absorbed from the diet induces a mild metabolic alkalosis which interferes with the ability of the tissues to recognize parathyroid hormone interfering with Ca homeostasis.

Grass tetany and other hypomagnesemic disorders of cattle - High K content of forages and pastures coupled with low dietary Mg content prevents absorption of Mg from the rumen. High rumen K concentrations depolarize the apical membrane of the rumen epithelium reducing the electromotive force that normally allows Mg to be absorbed across the rumen wall. Ruminants do not absorb Mg very well from the intestines as do monogastrics.

Dietary Requirement for Na, Cl, and K: The factorial approach does not work!! It predicts the requirement is much lower than practice dictates the requirement should be. Perhaps a requirement for acid- base balance is not taken into account with the factorial approach!

SULFUR (S)

Methionine, thiamin, and biotin cannot be synthesized by mammalian tissues. These nutrients must either be supplied in the diet. When provided with adequate substrates; nitrogen, energy and S; rumen microbial synthesis of methionine, thiamin, and biotin can supply enough of these compounds to the ruminant to meet daily requirements with the possible exception of very high producing cows. Therefore only ruminants can be said to have a dietary requirement for S.
Rumen microbes need .2 -.22 % S diets to operate efficiently. Corn silage diets are the primary type ration benefiting from sulfate supplementation.

Deficiency: The body has no real requirement for S (or sulfate), the form that will actually be in the body. A "S deficiency" is a deficiency in the S containing amino acids, thiamine, or biotin which are discussed elsewhere.

Toxicity: Excessive dietary S can interfere with absorption of other elements, particularly Cu and Se.

Acute S toxicity causes neurological changes, including blindness, coma, muscle twitches and recumbency. Post-mortem examination reveals severe enteritis,

peritoneal effusion and petechial hemorrhages in many organs, especially kidneys. Often the breath will smell of hydrogen sulfide - which is likely the toxic form of S. Sulfates are less toxic, though they can cause an osmotic diarrhea as the sulfate is only poorly abosorbed. Excess sulfate added to rations can reduce feed intake and performance. Water containing more than 5000 mg S/kg. reduces feed and water intake. Recent observations in beef cattle have determined that a polioencephalomalacia-like syndrome can be induced with diets containing 0.5% S using sulfate salts as supplemental S sources or from drinking water high in sulfates. The strong reducing environment within the rumen can reduce dietary sulfate, sulfite and thiosulfate to sulfide within the rumen.

COBALT

Cobalt is a component of vitamin B_{12} (cobalamin) which is a cofactor for 2 major enzymes; methylmalonyl coenzyme A mutase necessary for conversion of propionate to succinate, and tetrahydrofolate methyl transferase which catalyzes transfer of methyl groups from 5-methyltetrahydrofolate to homocysteine to form methionine and tetrahydrofolate. Microbes are the only natural source of vitamin B_{12}. Rumen microbes can produce the entire vitamin B_{12} required by ruminants provided adequate available cobalt is in the diet.

Rumen microbes need 0.11 % Cobalt rations to perform efficiently.

Cobalt Cl and nitrate, and cobaltous carbonate and sulfate all appear to be suitable sources for cobalt in ruminants. Cobaltous oxide, being much less soluble, is somewhat less available (Henry, 1995). Cobaltous oxide pellets and controlled release glass pellets containing cobalt which remain in the rumen-reticulum have been used successfully to supply cobalt over extended periods of time to ruminants on pasture, though regurgitation can cause loss of some types of pellets.

Deficiency:

Ruminants appear to be more sensitive to vitamin B_{12} deficiency than non-ruminants, largely because they are so dependent on gluconeogenesis for meeting tissue glucose needs. A breakdown in propionate metabolism at the point where methylmalonyl-CoA is converted to succinyl-CoA may be a primary defect arising from vitamin B_{12} deficiency. The appearance of methylmalonic acid in urine may be used as an indicator of vitamin B_{12} deficiency. Vitamin B_{12} deficiency may limit methionine production and limit nitrogen retention.

Without cobalt in the diet, rumen production of vitamin B_{12} rapidly (within days) declines. Vitamin B_{12} stores in the liver of adult ruminants are usually sufficient to last several months when they are placed on a cobalt deficient diet. Young animals are more sensitive to dietary cobalt insufficiency because they have lower liver vitamin B_{12} reserves. Early signs of cobalt deficiency include failure to grow, unthriftiness, and weight loss. More severe signs include fatty degeneration of the liver, anemia with pale

mucous membranes, and reduced resistance to infection as a result of impaired neutrophil function.

While the cow may have adequate stores of vitamin B_{12} to last several months, the rumen microbes apparently do not. Within a few days of a switch to a cobalt deficient diet rumen concentrations of succinate rise, either as a result of inability of rumen microbes to convert succinate to propionate or a shift in rumen bacterial populations toward succinate rather than propionate production.

COPPER (Cu)

The trace mineral where deficiency is common and toxicity is also common!

Cu is a component of enzymes such as cytochrome oxidase necessary for electron transport during aerobic respiration, lysyl oxidase which catalyzes formation of desmosine cross links in collagen and elastin necessary for strong bone and connective tissues, ceruloplasmin which is esential for absorption and transport of Fe necessary for hemoglobin synthesis, tyrosinase necessary for production of melanin pigment from tyrosine, and superoxide dismutase which protects cells from the toxic effects of oxygen metabolites which is particularly important to phagocytic cell function.

Dietary Cu Requirement - factorial analysis:

Maintenance: Endogenous losses of copper are approximately -7.1 µg / kg body weight.

Growth: Copper content of growing tissues is about 1.15 mg/kg weight gained.

Lactation: Colostrum copper content is about 0.6 mg/kg colostrum.
Milk copper content is about 0.2 mg/kg on a well supplemented ration.

Pregnancy: In early gestation (<100 days) about 0.5 mg copper is incorporated into fetal, placental and uterine tissue growth each day , increasing to between 1.5 and 2 mg /day during the last month of gestation.

Cu AVAILABILITY FROM DIETS

Between 1 and 5% of dietary Cu will be absorbed by adult cattle. 4% is a good default value.

In monogastrics and pre-ruminants 40 to 70% of dietary Cu is absorbed. However, with the development of the rumen there is a tremendous decrease in Cu absorption Rumen chelators?

A small proportion of dietary Cu is absorbed across the wall of the stomach however Cu is absorbed primarily by mucosa cells of the small intestine. Cu absorption is a 2 stage process; first soluble Cu can diffuse across the brush border of enterocytes. Cu then crosses the basolateral membrane to enter the blood by facilitated diffusion which may involve co-transport with certain amino acids. The biggest deterrent to Cu transport out of the enterocyte is the Cu binding protein, metallothionein, which is produced within the enterocytes. Metallothionein will sequester the Cu within the cytosol of the enterocyte. The bound Cu is eventually lost to the feces upon desquamation of the intestinal epithelial cells. High concentrations of metallothionein helps prevent Cu toxicity by reducing the amount of dietary Cu absorbed. High intracellular Cu can induce intestinal metallothionein which in theory permits regulation of Cu metabolism.

Unfortunately a primary regulator of enterocyte metallothionein concentration is the Zn status of the animal. A diet high in Zn can induce high intestinal metallothionein concentrations which blocks Cu absorption leading to Cu deficiency. From a practical standpoint the research suggests diet zinc only interferes with copper absorption when dietary zinc is greater than 1000 mg/kg.

The availability of dietary Cu is reduced by the presence of S and molybdenum in the diet. S and molybdenum form tetrathiomolybdate in the solid phase of the rumen digesta. Tetrathiomolybdate binds Cu to form a highly insoluble complex that renders the Cu unavailable for absorption. Molybdenum can reduce monogastric Cu absorption as well but the effect is not as pronounced (1980 ARC).

High dietary Fe and water containing high amounts of Fe has also been implicated as a cause of Cu deficiency. It is suggested to increase Cu content of diet by 15% for every 200 ppm Fe in ration above 400 ppm.

Ingestion of soil decreases Cu absorption. Animals at pasture consume 10% of DM as soil. This effectively reduces copper availability by half.

Cu DEFICIENCY

Cu deficiency can interfere with melanin production leading to loss of hair color. An early classical sign of Cu deficiency in cattle is loss of hair pigmentation, particularly around the eyes. Scours is a clinical sign of Cu deficiency that seems to be unique to ruminants, though the pathogenesis of this lesion is not understood. Anemia (hypochromic macrocytic), fragile bones and osteoporosis, cardiac failure, poor growth and reproductive inefficiency characterized by depressed estrus are also observed in Cu deficiency.

An effect of Cu deficiency that is not easily observed is a loss of immune function. Neutrophils have a reduced ability to kill invading microbes leading to increased susceptibility to infections. The dietary Cu required for optimal immune

function may exceed the requirement to prevent the more classical signs of Cu deficiency.

Cu TOXICITY

While Cu toxicity can occur in any species ruminants are much more susceptible than monogastrics. While horses can tolerate 800 mg Cu / kg diet, sheep can be killed by diets containing as little as 20 mg Cu / kg diet! Cattle can generally tolerate as much as 100 mg Cu / kg diet. Cattle seem to have a greater capacity to eliminate Cu from the body by way of bile than do sheep. Goats tolerate more Cu than sheep but not as much as cattle. Cu toxicosis can occur in ruminants that consume excessive amounts of supplemental Cu or feeds meant for monogastrics or that have been contaminated with Cu compounds used for other agricultural or industrial purposes.

Breed differences exist in sheep and cattle that increase the susceptibility to Cu toxicity. Jersey cattle fed the same diet as Holstein cattle accumulate more liver Cu than do Holstein cattle (Du, 1996). It is not clear whether this reflects differences in feed intake, efficiency of Cu absorption, or biliary excretion of Cu.

When ruminants consume excessive Cu, they may accumulate extremely large amounts of the mineral in the liver before toxicosis becomes evident. Stress or other factors may result in the sudden liberation of large amounts of Cu from the liver to the blood, causing a hemolytic crisis. Such crises are characterized by considerable hemolysis, jaundice, methemoglobinemia, hemoglobinuria, generalized icterus, widespread necrosis, and often death

SYNDROMES OF SPECIAL CONCERN

Poultry Manure fed to ruminants can cause Cu toxicity.

Adding Cu at 10 to 50 times the concentrations required to meet requirements can substantially improve the rate of growth of swine and poultry and is a common practice.

NEONATAL ENZOOTIC ATAXIA (SWAYBACK) OF LAMBS

Ewes with chronic Cu deficiency can give birth to lambs that are weak and ataxic. The disease is characterized by symmetrical demyelination of the cerbrum, and degeneration of the motor tracts within the spinal cord. Unfortunately the lesions are permanent and Cu supplementation will not be of aid to these lambs. The disease has also been observed rarely in goats and cattle.

SELENIUM (Se)

Se is a necessary component of glutathione peroxidase, an enzyme which plays a major role in protecting tissues against oxidative damage from free radicals.

Glutathione peroxidase levels in serum are fairly well correlated with dietary Se concentrations. Vitamin E also can neutralize peroxides, but the action of vitamin E is limited to cell membranes. Vitamin E can replace some of the antiperoxidant function of Se, and Se can spare vitamin E by scavenging free radicals before they get to cell membranes.

Se is also critical to thyroid hormone metabolism because the enzyme, iodothyronine 5'-deiodinase, is a Se containing protein. A selenoprotein also seems to be important in muscle, though it is not yet identified. In Se replete animals a selenoprotein can be isolated from the muscle, but it is not present in animals that are Se deficient (White Muscle Disease).

DIETARY REQUIREMENT FOR Se

Research suggests most cattle will do fine when diets contain 0.1 ppm selenium. Experience in the field suggests this is not enough. Legally you can only add 0.3 ppm. The duodenum is the major site for Se absorption. Se absorption is not regulated and homeostasis of Se is regulated by controlling urinary excretion of Se. When dietary Se is in great excess of requirements Se is also expired in the breath as dimethylselenide.

Factors affecting absorption of Se: High dietary or water S seems to interfere with Se absorption. Other factors are unidentified. There seem to be many field situations where 0.3 ppm Se added to feed (legal limit) is inadequate (fails to elevate blood Se). Difficult to duplicate in research conditions. Injection of Se may be an option - READ THE LABEL CAREFULLY!

Deficiency: The soil in large areas of the United States is too low in Se and will not provide adequate Se to meet the needs of animals fed crops grown on those soils. The states bordering the Great Lakes, the Pacific Northwest, and the Eastern shore areas are all considered areas where Se deficiency is likely to occur. Se deficiency causes infertility and poor growth in most species. Se deficiency has some species specific affects as well. Some of these affects can be reduced by vitamin E supplementation. Most animals require about 0.1 -0.3 mg Se / kg diet.

Lambs and calves: White muscle disease - a nutritional muscular dystrophy causing necrotic changes in the striated muscles of the body which is most common in lambs and calves, but also occurs in pigs, foals, and poultry as well. The name is derived from the white striations observed in many of the muscles of the body, particularly those in the thigh and shoulder. Lesions are bilaterally symmetrical and serum aspartic aminotransferase activity (SGOT) will be greatly elevated.

Dairy cows: Se deficiency is associated with an increased risk of retained placenta and perhaps mastitis. It is thought that Se deficiency reduces the immune response in the cow. The mechanisms are largely unknown.

Toxicity: In his travels across Asia Minor, Marco Polo related that horses ingesting certain plants would lose their mane, and tail hair and slough their hooves. Toxicity from Se occurs in two forms acute and chronic. Acute Se poisoning is associated with hepatic and renal damage and can include hemorrhagic exudate in the lungs and ascites is common. Blindness and stumbling are also common. Gastroenteritis may be present. Chronic Se poisoning in horses and catle is associated with lameness and loss of hair and hoof malformations. Animals at pasture eventually die from starvation due to impaired mobility. Se can be toxic at 8-10 mg/ kg diet.

Blind Staggers - cattle and horses: Certain plant species such as princesplume, woody aster and (Stanleya, Xylorrhiza, and Astragalus species) found in pockets of the American upper great plains and deserts are Se accumulators and can contain several hundred to a thousand mg Se / kg. They are generally unpalatable but animals consuming these plants can develop Se toxicity. If consumed in high amounts the animal can exhibit acute poisoning - blind staggers. More commonly in periods of drought the animals on pasture may be hungry enough to occasionally eat a few of these Se accumulator plants.

Alkali disease: The soil (usually alkaline soils) may have high enough Se that forage plants grown in these areas provide more than 10 mg Se / kg pasture may and over time the animals develop lameness and emaciation- alkali disease. Profitable ranching is nearly impossible in these particular areas of the country.

MOLYBDENUM

Molybdenum is a component of xanthine oxidase, sulfide oxidase, and aldehyde oxidase; enzymes found in milk and many tissues. Milk and plasma molybdenum concentration increases as dietary molybdenum increases.
Deficiency - From a practical standpoint this is not a concern

Toxicity: Dietary molybdenum becomes a practical concern because it antagonizes the absorption of Cu (and to a lesser extent Phos). Molybdenum toxicosis signs are essentially those associated with Cu deficiency. Molybdenum and sulfate interact within the digestive tract to form a thiomolybbdate complex which has a high affinity for Cu. Cu bound to this molybbdate is unavailable for absorption (see section on Cu). The toxicity of molybdenum can be overcome by increased Cu supplementation and Cu toxicity can be reduced by molybdenum supplementation. The critical ratio of dietary Cu: dietary molybdenum needed to avoid Cu deficiency ranges from 2:1 in reports from Canada to 4:1 on pastures in England with a high molybdenum content (20-100 mg molybdenum / kg forage DM). In the United States molybdenum is a significant problem in the western states and around the Everglades in Florida.

IODINE

Iodine is necessary for the synthesis of the thyroid hormones thyroxine and triiodothyronine that regulate energy metabolism. Thyroid hormone production is also

increased during colder weather to stimulate an increase in basal metabolic rate as the animal attempts to remain warm.

Most iodine sources are readily available and the iodides of Na, K and Ca are commonly used. K iodide tends to be easily oxidized and volatilizes away before the animal can ingest it. PentaCa orthoperiodate and ethylenediamine dihydroiodine (EDDI) are more stable and less soluble and commonly used in mineral blocks and salt licks exposed to the weather.

Forage iodine concentrations are extremely variable and depend on soil iodine content. Soil near the oceans tends to provide adequate iodine to plants. However in the Great Lakes regions and Northwest USA iodine concentrations in forages are generally low enough to result in iodine deficiency unless supplemented. Iodine deficiency remains a common problem in many parts of the world.

Dietary Iodine Requirement: Miller (1988) calculated the dietary iodine requirement as 0.6 mg iodine per 100 kg body wt. This analysis assumed a daily thyroxine secretion rate of 0.2 - 0.3 mg /100 kg body wt. which would require about 2.1 mg Iodine. If the thyroid binds 30% of dietary iodine the dietary requirement would be 0.7 mg Iodine / 100 kg Bwt. However, 15% of daily thyroxine iodine needs should be met by recycling of iodine released during metabolism of previously produced thyroid hormones, which reduces the dietary iodine requirement to 0.6 mg iodine / 100 kg Bwt / day. Depending on diet DM intake this would correspond to a dietary requirement of about 0.25 - 0.5 mg iodine / kg D.M.

Deficiency Symptoms: Iodine deficiency reduces thyroid hormone production slowing the rate of oxidation of all cells. Often the first indication of iodine deficiency is enlargement of the thyroid (goiter) of newborn animals. Animals may be born hairless, weak, or dead. Fetal death can occur at any stage of gestation. Often the mothers will appear normal. Under conditions of marginal or deficient dietary iodine the maternal thyroid gland becomes extremely efficient in uptake of iodine from the circulation and recycling thyroid hormone iodine. Unfortunately this leaves little iodine for the fetal thyroid gland and the fetus becomes hypothyroid. The goiter condition is the hyperplastic response of the thyroid gland to increased pituitary Thyroid-stimulating hormone production.

Adult animals with iodine deficiency are unthrifty and often infertile.

FACTORS AFFECTING IODINE REQUIREMENT

Goitrogens are compounds that interfere with the synthesis or secretion of thyroid hormones and cause hypothyroidism. Goitrogens fall into two main categories. Cyanogenic goitrogens impair iodide uptake by the thyroid gland. Cyanogenic glucosides can be found in many feeds, including raw soybeans, beet pulp, corn, sweet potato, white clover, and millet and once ingested are metabolized to thiocyanate and isothiocyanate. These compounds alter iodide transport across the thyroid follicular cell

membrane, reducing iodide retention. This effect is easily overcome by increasing supplemental iodine.

Progoitrins and goitrins found in cruciferous plants (rape, kale, cabbage, turnips, mustard) and aliphatic disulfides found in onions inhibit thyroperoxidase preventing formation of mono- and diiodotyrosine. With goitrins, especially those of the thiouracil type, hormone synthesis may not be readily restored to normal by dietary iodine supplementation and the offending feedstuff needs to be reduced or removed from the diet.

Dietary iodine required to overcome the goitrin effects could result in milk with excessive iodine content.

IRON (Fe)

Fe primarily functions as a component of heme found in hemoglobin and myoglobin. Enzymes of the electron transport chain, cytochrome oxidase, ferredoxin, myeloperoxidase, catalase, and the cytochrome P-450 enzymes also require Fe as cofactors.

Absorption: During absorption the Fe binds to specific non-heme Fe-binding receptors within the brush border of the enterocyte and is transported into the cell. Once inside the cell the Fe can be transported to the basolateral membrane and becomes bound to transferrin for transport within the blood. If Fe status of the body is adequate the Fe entering the enterocyte is not transported to the basolateral membrane but is instead bound by ferritin, a protein produced by the enterocytes when Fe is not needed by the body. Once bound to ferritin the Fe is excreted with the feces when the enterocyte dies and is sloughed. The amount of dietary Fe absorbed can be controlled by up-regulation or down-regulation of enterocyte ferritin content. How enterocyte ferritin concentrations are regulated by Fe status of somatic cells is unknown.

Fe Deficiency - a problem of young only: Hypochromic microcytic anemia due to failure to produce hemoglobin. Light colored veal is due to low muscle myoglobin levels as a result of restricted dietary Fe. Anemic animals are listless and have poor feed intake and weight gain. Another important aspect of Fe deficiency is greater morbidity and mortality associated with depressed immune responses. Increased morbidity may be observed before the effect of Fe deficiency on packed red blood cell volume.

Fe Toxicity: Excessive dietary Fe is of concern for two reasons:

1. Fe interferes with the absorption of other minerals, primarily Cu and Zn. As little as 250 - 500 mg Fe/kg diet DM has been implicated as a cause of Cu depletion in cattle.

2. If absorbed dietary Fe exceeds the binding capacity of transferrin and lactoferrin in blood and tissues free Fe levels may increase in tissues. Free

Fe is very reactive and can cause generation of reactive oxygen species, lipid peroxidation, and free radical production leading to Òoxidative stressÓ, increasing anti-oxidant requirements of the animal. Free Fe is also required by bacteria for their growth and excessive dietary Fe could contribute to bacterial infection. The body can produce substances such as lactoferrin which binds free Fe making it unavailable for bacterial growth and preventing bacterial infection.

Fe toxicity is associated with diarrhea, reduced feed intake and weight gain. The NRC (1980) recommends dietary Fe not exceed 1000 mg/kg DM (Council, 1980). This may be too high!

CHROMIUM

Function: Chromium is primarily found in tissues as an organometallic molecule composed of Cr+3, nicotinic acid, glutamic acid, glycine, and cysteine known as glucose tolerance factor. Without Cr +3 the glucose tolerance factor is inactive. Glucose tolerance factor can potentiate the effect of insulin on tissues, either by stabilizing the insulin molecule, or by facilitating the interaction of insulin with its receptor in tissues.

The essentiality of chromium as a required element necessary for normal glucose metabolism in the diet of humans is well accepted and it is recommended that the diet of adult humans supply from 50 - 200 µg chromium / day. The role of chromium in animal nutrition was recently reviewed by the National Research Council (NRC,1997). It has been used at 5- 10 mg /cow / d (0.5 mg / kg DM) in dairy cow studies with some positive effects. Unfortunately the amount of chromium required in the diet for optimal performance is unclear and the literature does not support a general recommendation for chromium supplementation of typical diets. Additional research on the level of bioavailable chromium contained in common feedstuffs and the effects of chromium deficient diets on performance of animals will be required, including appropriate titration studies, before a minimum dietary chromium requirement can be established.

Zn

Zn is a component of many metalloenzymes such as Cu-Zn superoxide dismutase, carbonic anhydrase, alcohol dehydrogenase, carboxypeptidase, alkaline phosphatase and RNA polymerase, which affects metabolism of carbohydrates, proteins, lipids, and nucleic acids. Zn regulates calmodulin, protein kinase C, thyroid hormone binding, and inositol phosphate synthesis. Zn deficiency alters prostaglandin synthesis which may affect luteal function. Zn is a component of thymosin, a hormone produced by thymic cells which regulates cell-mediated immunity.

Absorption: Intestinal Zn absorption occurs primarily in the small intestine. In animals that are Zn deficient Zn readily enters the enterocytes and is transported across the cell by a cysteine rich intestinal protein (CRIP) and released into the portal circulation to be

carried primarily by transferrin and albumen. In animals that are Zn replete, metallothionein, a second cysteine rich protein, is found in the mucosal cells and this metallothionein competes with the cysteine rich protein for Zn coming across the brush border membrane. Zn bound to metalothionein will remain in the enterocyte and be excreted with the feces when the enterocyte dies and is sloughed. By up-regulating or down-regulating mucosal enterocyte metallothionein content the amount of dietary Zn that is absorbed can be regulated. How Zn status regulates intestinal metallothionein concentration is unknown, but it seems to take weeks to change metallothionein concentration in the intestine to adjust to a low Zn diet.

Factorial determination of dietary Zinc Requirement:

Maintenance: endogenous fecal loss is approximately 0.033 mg zinc/kg BW and the bligate urinary loss of zinc has been estimated as 0.012 mg zinc/kg BW or a total zinc maintenance requirement of 0.045 mg zinc/kg body wt.

Pregnancy: about 12 mg zinc/day between day 200 of gestation and the end of gestation.

Lactation: Milk zinc content is about 4 mg zinc/kg milk

Growth: 24 mg zinc/kg live weight gain

Absorption: Coeffcient of absorption for dietary zinc is estimated to be 0.15.

Two major dietary factors that can modify the efficiency of absorption of dietary Zn are interactions of Zn with other metal ions and the presence of organic chelating agents in the diet.

1. Zn and Cu are antagonistic to one another. In most cases Zn interferes with Cu absorption to cause Cu deficiency but when dietary Cu:Zn ratios are very high (50:1) Cu can interfere with Zn absorption. Excessive dietary Fe can interfere with Zn absorption in man and other species. In rats Fe deficiency enhanced both Fe and Zn absorption suggesting Fe and Zn share a common absorption mechanism. In man the effect of excess Fe is evident when the Fe:Zn ratio is greater than 2:1. No data exists on the interaction between dietary Fe and Zn absorption in ruminants. Under practical conditions dietary Fe content is often well in excess of Fe requirements of herbivores.

Cadmium is antagonistic to the absorption of both Zn and Cu and also interferes with tissue metabolism of Zn and Cu in the liver and kidneys. Lead competitively inhibits Zn absorption and also interferes with Zn function during heme synthesis.

High dietary Ca interferes with Zn absorption in non-ruminants (see parakeratosis of swine). The mechanism is not well understood though it appears to be of greater concern when diets are high in phytate.

2. Organic chelators of Zn can increase or decrease bioavailability of Zn.

Those that interfere with absorption tend to form insoluble complexes with Zn. One such chelator is phytate (phytic acid) which is also an important chelator of phosphate. Phytate commonly binds Zn in plant sources of Zn and greatly diminishes the availability of Zn for absorption in monogastric and pre-ruminant animals. However, rumen microbes metabolize most of the dietary phytate so it is not a factor affecting Zn absorption in ruminating animals. Others remain unidentified.

Some naturally occurring Zn chelators improve Zn bioavailability. Scott and Ziegler (1963) demonstrated with chicks that adding distillers dried solubles and liver extract to soybean protein based diets improved the availability of the dietary Zn, though the factor remained unknown. Peptides and amino acids can form complexes with Zn and both cysteine and histidine bind Zn strongly and improve bioavailability of Zn in chicks (Nielsen et al., 1966) (Hortin et al., 1991). At the alkaline pH found in the intestine it is likely that little free Zn cation exists in solution. One action of beneficial chelates is to form Zn complexes that are soluble within the small intestine permitting soluble Zn to reach the brush border membrane for absorption.

Deficiency: Zn deficient animals quickly exhibit reduced feed intake, and reduced growth rate. With more prolonged deficiency the animals exhibit reduced growth of testes, weak hoof horn and perakeratosis of the skin on the legs, head (especially nostrils), and neck. On necropsy thymic atrophy and lymphoid depletion of the spleen and lymph nodes are evident.

Toxicity: High dietary Zn is fairly well tolerated by cattle, however Zn toxicity was observed in cattle fed 900 mg Zn / kg diet. High levels of Zn have a very negative effect on Cu absorption and metabolism and it is for this reason primarily that dietary Zn content should be limited. The maximal tolerable level of dietary Zn is suggested to be 300- 1000mg/ kg diet.

Genetic Zn deficiency of cattle: A genetic defect that greatly reduces Zn absorption has been identified in Black Pied and Dutch-Friesan cattle. These animals become severely Zn deficient unless fed extremely high levels of dietary Zn. Calves appear normal at birth but develop scaly thickened skin over the neck and shoulders within a few months. They also grow slowly and are very susceptible to infection due to their inability to mount an immune response.

MANGANESE (Mn)

Mn is a required cofactor for several enzymes necessary for production of bone collagen and cartilage. Mn super oxide dismutase works in concert with other anti-oxidants to minimize accumulation of reactive forms of oxygen which could damage cells.
Dietary Mn Requirement:

Maintenance:	than .002 mg / kg BW or about 1 mg in a 500 kg cow.
Pregnancy:	0.3 mg Mn / d from Day 190 til parturition
Lactation:	0.03 mg / kg milk
Growth:	0.7 mg manganese / kg gain

The proportion of Mn absorbed from the diet generally between 1 and 0.5% . A good assumption is 0.75% will be absorbed. A mechanism to enhance the efficiency of Mn absorption during Mn deficiency does not appear to exist. Mn accumulates in the liver in direct proportion to dietary Mn providing a more precise index of Mn status.

Factors: High dietary calcium, potassium or phosphorus increase manganese excretion in the feces, presumably by reducing manganese absorption
Excessive dietary iron depresses manganese retention in calves.

Deficiency: Mn deficiency can cause impaired growth, skeletal abnormalities (shortened and deformed), disturbed or depressed reproduction, and abnormalities of the newborn (including ataxia-due to failure of the inner ear to develop).

The skeletal changes are related to loss of galactotransferase and glycosyltransferase enzymes that are vital to production of cartilage and bone ground substance mucopolysaccharides and glycoproteins.

Calf deformitiesassociated with Mn deficient dams included weak legs and pasterns, enlarged joints, stiffness, twisted legs, general weakness, and reduced bone strength. Heifers and cows that are fed low-Mn diets are slower to exhibit estrus, are more likely to have "silent heats," and have a lower conception rate than cows with sufficient Mn in their diet.

Toxicity: Mn toxicity is unlikely to occur, and there are few documented incidences with adverse effects limited to reduced feed intake and growth . These negative effects began to appear when dietary Mn exceeded 1000 mg /kg.

LITERATURE CITED

Agriculture Research Council. 1980. The Nutrient Requirements of Ruminant Livestock, Commonwealth Agricultural Bureaux, Farnham Royal, Slough, U.K.

Ender, F., I. W. Dishington, et al. (1971). "Calcium balance studies in dairy cows under experimental induction and prevention of hypocalcaemic paresis puerperalis. The solution of the aetiology and the prevention of milk fever by dietary means." Zeitschrift fuer Tierphysiologie Tierernaehrung und Futtermittelkunde 28: 233-256.

Goff, J. P. 1998. Phosphorus deficiency. In: Current Veterinary Therapy 4: Food Animal Practice. J. L. Howard, and R. A. Smith, Editors. W. B. Saunders, Co., Philadelphia, p. 218-220.

Goff, J. P. 1998. Ruminant Hypomagnesemic Tetanies. Page 215 in Current Veterinary Therapy 4: Food Animal Practice. 4th W.B. Saunders Co., Philadelphia.

Greene, L. W., J. P. Fontenot, et al. (1983). "Site of magnesium and other macromineral absorption in steers fed high levels of potassium." J. Anim. Sci. 57(2): 503-10.

Hansard, S., H. Crowder, et al. (1957). "The biological availability of calcium in feeds for cattle." J. Anim. Sci. 16: 437-443.

Hortin, A. E., Bechtel, P. J. and Baker, D. H., (1991). Efficacy of pork loin as a source of zinc and effect of added cysteine on zinc bioavailability. J. Food Sci. 56, 1505.

Martens, H., (1983). "Saturation kinetics of magnesium efflux across the rumen wall in heifers." Br. J. Nutr. 49(1): 153-8.

Martens, H. and G. Gabel, (1986). "[Pathogenesis and prevention of grass tetany from the physiologic viewpoint]." DTW Dtsch Tierarztl Wochenschr 93(4): 170-7.

Martz, F. A., A. T. Belo, et al., (1990). "True absorption of calcium and phosphorus from alfalfa and corn silage when fed to lactating cows." J. Dairy Sci. 73: 1288-1295.

Miller, J., Ramsey, N. and Madsen, F., (1988). The trace elements. In: "The Ruminant Animal: Digestive Physiology and Nutrition" (D. C. Church, ed.), pp. 342-400. Prentice-Hall Inc, Englewood Cliffs, NJ.

National Research Council. 1980. Mineral Tolerance of Domestic Animals. Natl. Acad. Sci., Washington, DC.

National Research Council. 1989. Nutrient Requirements of Dairy Cattle. 6th rev. ed. Natl. Acad. Sci., Washington, DC.

National Research Council, (1997). "The Role of Chromium in Animal Nutrition". National Academy Press, Washington D.C.

Nielsen, F. H., Sunde, M. L., and Hoekstra, W. G., (1966). Effect of dietary synthetic and natural chelating agents on the zinc-deficiency syndrome in the chick. J. Nutr 89, 35-42.

Puls, R., 1994. Mineral Levels in Animal Health. 2nd Sherpa International, Clearbrook, B.C.

Scott, M. L. and Ziegler, T. R., (1963). Evidence for natural chelates which aid in the utilization of zinc by chicks. J. Agric. Food Chem. 11, 123.

Soares, J., (1995). Calcium bioavailability. Bioavailability of nutrients for animals. C. Ammerman, D. Baker and A. Lewis. San Diego, Ca, Academic Press, Inc.: 95-113.

Wu, Z., L. D. Satter, R. Sojo, and A. Blohowiak., 1998b. Phosphorus balance of dairy cows in early lactation at three levels of dietary phosphorus. J. Dairy Sci. 81 (Suppl.1):358.

DIAGNOSING MACRO-MINERAL INSUFFICIENCY OR IMBALANCE

Mineral	Best Tissue to Take	Normal	Subclinical	Clinical
Ca (acute)	Serum (mg/dL)- total Ca	8.0 - 10.5	5.5 - 7.5	< 5.5
	Rib Ash (% Ca)	30 - 38%	25 - 30%	< 25%
	Urine (mg/dL)	2.0 - 50.0		< 0.1
Phos	Serum (mg/dL)	4.5 – 6	3.5 - 4.5	< 3.0
	Rib ash (% P)	17 - 20%	15 - 17%	< 12%
Mg	Serum (mgdL)	1.9 - 2.3	1.5 - 1.85	< 1.5
	Vitreous humor (mg/dL)	1.9 - 2.5	1.2 1.8	< 1.0
	Urine	10 – 25	2 – 5	< 0.5
	cerebrospinal fluid (mg/dL)	2 - 2.5		< 0.5
Sulfur	Diet only	adequate = 0.22%		
Chloride	Urine (meq/L)	20 – 150	2 – 6	< 2
	Serum (meq/L)	98 – 110		< 90
Sodium	Urine (meq/L)	10 – 50		< 1.0
	Serum (meq/L)	135 – 152	130- 135	< 125
Potassium	Serum (meq/L)	4 - 5.5		< 2.5 ??

DIAGNOSING TRACE MINERAL DEFICIENCY IN ADULT CATTLE

Mineral	Tissue	Normal	Subclinical	Clinical
Cobalt	Serum Vit. B_{12} (ng/ml)	> 0.4	.25 - .4	< 0.2
	Serum methylmalonic acid (µmol/L)	< 1.5	2.5 – 5	>5
Copper	*Liver (ppm DM)	100 – 400	40 – 100	< 40
	Serum Ceruloplasmin (IU/L)	40 – 50	10 – 30	< 5
	RBC - Cu-Zn superoxide dismutase (mg/g hemoglobin)	> 0.3		< 0.3
Iodine	Serum (µg/dL)	10 – 50	5 – 10	< 5
	Serum thyroxine (ng/ml)	20 – 100	< 10	
	* Milk (µg/L)	30 – 300	< 10	
Iron	Blood PCV	33 %	< 25	< 20
	Liver (ppm DM)	200–1500		< 150
Manganese	Liver (ppm DM)	10 – 25		< 4
Selenium	*EDTA whole blood (µg/ml)	0.2 – 1	0.1 - 0.2	< 0.1
	Serum (µg/ml) = poor test	0.08 - 0.2	0.04 - 0.06	< 0.03
	Blood GSH-Peroxidase (µmoles/min/mg Hemoglobin)	20 – 35	10 – 20	< 10
	* Liver (ppm DM)	0.25 - 0.60		< 0.15
Zinc	Liver (ppm DM) poor, but best	100 – 400	50 - 150?	< 50

*** = good test**
Tissue DM is about 25% of wet weight