Trace Mineral Nutrition in Cattle

D. G. Pugh, *DMV, MS, Dipl ACT, Dipl ACVN*
Food Animal Section
Department of Large Animal Surgery and Medicine
College of Veterinary Medicine
Auburn University, Alabama 36849-5522

Trace minerals are needed in only trace amounts, and are measured in either parts per million (ppm) or mg/kg. Fifteen of these mineral elements have been identified (arsenic, cobalt, copper, chromium, fluoride, iodine, iron, manganese, molybdenum, nickel, selenium, silicon, tin, vanadium, and zinc), but this discussion will concern only copper, cobalt, iodine, selenium, and zinc.[1] These minerals are required in specific biochemical reactions as part of metalloenzyme systems or as cofactors for enzymematic functions. Trace mineral deficiencies usually occur slowly over time. The clinician who is confronted with a problem where trace minerals are suspected should always obtain a detailed herd history, perform a complete physical examination on affected animals, evaluate the total diet, including pastures, and harvest tissues needed to evaluate the specific mineral concentrations in the body.[1]

Salt is commonly used as a carrier to ensure trace mineral intake, as cattle have a clear cut drive for NaCl. If the clinician and rancher elect to use a salt containing mineral mixture to insure mineral intake it should be remembered individual animal intakes may vary drastically.[1] Further, improperly prepared salt mixtures, feed supplements or blocks, liquid feeds, or some types of contamination can alter nutrient absorption and produce conditional deficiencies.

Copper

Copper (Cu) deficiency may occur outright due to low Cu concentrations in roughages of certain geographic regions. Deficiencies can also be secondary or conditional[2] due to high concentrations of molybdenum (Mo) or sulfur (S) in feedstuffs. In the rumen Cu, Mo and S may form thiomolybdates which reduce Cu availability. Other factors which alter Cu absorption are high concentrations of dietary cadmium, iron, selenium, zinc, Vitamin C, and alkaline soils.[1] Roughages grown on "improved pastures" (eg limed, fertilizer) will more likely result in Cu deficiency because fresh forages have lowered available Cu; liming reduces Cu uptake by the plant and many fertilizers contain Mo.[9] There appears to be breed alterations in Cu availability with Angus being more susceptible and Brahman appearing to be less susceptible to deficiencies.

Good quality lush grass forages will have lower available Cu than most hays. Legumes have more available Cu than do most grasses.[1] Signs of Cu deficiency in cattle include anemia, depressed milk production, lighter or faded hair coat color, heart failure, infertility, increased susceptibility to disease, slowed growth, enlarged joints, lameness, gastric ulcers, and diarrhea.[1,2,3,5,7,8,9] Signs of Cu deficiency appear to be more severe than with primary Cu deficiencies with a lowered Cu:Mo ratio, but may still take up to 170 days before signs occur.[2] Calves are more susceptible to Cu deficiency as they are continuing to grow and may have high requirements; milk is usually deficient in Cu while Mo is concentrated in milk.[9] Calves will have poor growth rate, lightened hair coats and diarrhea.[2,3,4,9]

When suspecting Cu deficiency both the Cu and Mo concentrations of the entire diet should be determined. In order to help to confirm copper deficiency, the clinician should measure body tissue concentration. Serum Cu is commonly utilized to determine body Cu status, but much of the Cu will be bound in the clot, thus making plasma a more reliable indicator of body Cu status.[3] Unfortunately, from a body assessment standpoint, blood Cu concentrations may be falsely increased by stress or disease, with resultant difficulty in interpreting the values unless they are deficient. Generally speaking, if serum Cu concentrations are used for assessment and Cu concentrations fall within normal ranges, additional Cu supplementation will probably be of little or no value unless dietary Mo is high[3] or the Cu to Mo ratio is <5:1. All blood samples should be collected in royal blue top or trace mineral tubes. Liver concentrations are perhaps the best tissue to help determine Cu status,[3] but has limitations as it is a poor indicator of short term Cu balance.[9]

A liver biopsy can be performed on the left side of a restrained, standing cow on a line from the point of the elbow to the tuber coxae at the 10th intercostal space.[3,4] Cows in advanced pregnancy may need to be sampled in the 9th intercostal space.[4] An area is clipped and aseptically prepared with only isopropyl alcohol (disinfectants may adulterate samples).[4] After anesthetizing the skin, a stab incision through the skin with a 14 gauge, 6 inch Tru-Cut® needle is advanced in a very slightly ventral-cranial direction, while feeling the needle move through the body wall and diaphragm. After passing through the diaphragm the needle is then advanced an additional cm and the sample is carefully harvested.[4] The clinician should be familiar with the working of the biopsy needle prior to its use. If the biopsy chamber is completely filled with liver tissue, only one biopsy will be needed.[4] Liver Cu may be low if plasma Cu is very deficient. If liver Cu is marginal yet plasma Cu is in normal ranges, the animal may have a favorable response to Cu supplementation.[9] Biopsies can be mailed to: Animal Health Diagnostic Health Laboratory, Veterinary Medical Center, Wilson Road, Michigan State University, East Lansing, Michigan 48824. Any laboratory chosen to perform the analysis should be consulted for their preferred tissue handling and shipping practices.

Copper concentrations in hair are reflective of the concentration of Cu in the plasma during the growth phase of the hair and is therefore of limited value. A recent survey suggests that the traditional procedure of testing the blood of 10% of a suspect herd may be inappropriate and more animals may be sampled.[6] In certain instances it may be advisable to sample not only a cross section of ages and production status, but also as many animals who are showing clinical signs as is possible. Brown paper may be contaminated with Cu, therefore care should be exercised in order to avoid its use if Cu deficiency is suspected.[1] Dietary Cu can range between 4 to 10 ppm[5] and the Cu:Mo ratio should be maintained between 6:1 and 10:1. In extremely deficient areas, copper needles can be administered orally. These needles will be trapped in the abomasum thus bypassing rumenal reactions which suppress Cu availability.[1] In most situations trace mineral salt mixes with 0.5 to 1.9% Cu sulfate[2] or 0.2 to 6% Cu[9] will usually prevent deficiency.[1] The form of Cu used in salt mixes should be examined, particularly in areas of Cu deficiency or Mo excess. Copper oxide is less absorbable than copper sulfate, while copper lysinate has still greater absorbability. Dietary Cu concentration above 115ppm[5] may be toxic. This is a very real potential in some broiler litter fed diets.[7]

Iodine

Iodine (I) deficiency is more common in certain geographic regions of North America, particularly the northern tier states. Iodine availability is depressed by methylthiouracil, nitrates, perchlorates, soybean meal, and thiocynates.[1] Minerals which interfere with I absorption are rubidium, arsenic, fluorine, calcium, and potassium.[5] Iodine appears to be most available for use by the body during winter months or during lactation.[1] There appears to be breed effects as Jersey cattle absorb I more readily than Holsteins.[1] The state in which I exists in the feed alters availability as iodates are absorbed more readily than are iodides.[1] Signs of I deficiency include goiter, poor growth, depressed milk yield, and possibly ketosis, reproductive abnormalities, (eg. abortion, stillbirth, retained placentas, irregular estrus, infertility, depressed libido, birth of small, weak, or hairless calves).[1,5]

After a thorough examination of the diet if I deficiency is still suspected, serum or plasma thyroxine can be used to assess the body status, and will be lowered in deficient states.[1] Iodine is readily absorbed so most sources will work well in salt mineral mixtures or feed supplements.[2] A salt mineral mixture containing 0.01% I will usually prevent deficiency.[5] Other sources of supplementation, calcium iodate, EDDI, potassium or sodium iodine are useful unless they are to be exposed to very humid, hot or rainy conditions, which promote leaching.[2,5]

Cobalt

Cobalt (Co) is utilized by rumen bacteria in the formation of Vitamin B_{12}. It may be deficient in some highly organic or poorly drained soils, resulting in Vitamin B_{12} deficiencies of unsupplemented cattle grazing forages grown on those soils. Signs of Co deficiency include anemia, rough hair coat, poor growth, reproductive abnormalities, (eg. anestrus, infertility, abortion, weak calves), muscle wasting, and depressed internal parasite resistance.[5,9]

In order to determine if Co deficiency exists, the entire diet should be evaluated. Where available, assays for serum or urinary methylmalonic acid may be performed, and are increased with Co deficiency.[1] From a practical standpoint, it is difficulty to find this assay being performed commercially. Serum Vitamin B_{12} concentration may be available and can help identify a deficient animal.[5] Liver samples can aid in the diagnosis of deficiency Co. Unfortunately due to tissue concentration of Co, diagnosis may be difficult.[2] A diet with a Co concentration of 0.1ppm is adequate in most instances but dietary levels below 0.06ppm should be considered deficient. If a frank deficiency exists, a Co supplemented trace mineral mixture fed free choice or the addition of 120g of Cobalt sulfate per ton of "salt lick" may be effective.[5]

Selenium

Selenium (Se) has received much interest and discussion in recent years, and even though volumes of research have been published, Se deficiency-associated clinical diseases still exist. The absorption of Se from the small intestine is enhanced by adequate dietary levels of Vitamins E and A and histidine.[1] Large dietary quantities of arsenic, calcium, Vitamin C, Cu, nitrates, sulfates, and unsaturated fats all inhibit Se absorption.[1] Forages grown on rock-derived acidic soils are more likely to be deficient in Se.[1] Legumes are usually better sources of Se than grasses, which are in turn superior to cereals.

The signs of Se deficiency include nutritional muscular dystrophy, particularly the skeletal and cardiac muscles of fast growing young calves, and retained placentas; while signs only associated with insufficient Se include poor growth, weak or premature calves, and depressed immune function, increased cases of early lactational mastitis, increased susceptibility to metritis.

Liver is the preferred tissue for analysis, but many clinicians prefer to collect either serum or whole blood. Glutathione peroxidase activity is also occasionally used, but availability of this assay may limit its use. Serum Se concentrations are difficult to interpret as they may reflect the dietary intake in the recent 2 to 4 weeks. Whole blood Se is more reflective of dietary Se intake over the past + 100 days. Milk concentrations of Se may also be of value in diagnosis.

Diets containing 0.2ppm are usually protective for most classes of cattle. The clinician should always inspect all trace mineral supplements as many are devoid of Se. Mineral salt mixes should contain between 24 and 90ppm Se. In cases of frank deficiency, injectable Vitamin E and Se preparations may be given, particularly prior to times of expected stress, such as calving. Sustained release boluses of Se have been used successfully in the recent past.[2]

Zinc

The availability of zinc (Zn) is positively affected by the presence of sufficient quantities of Vitamin C, lactose, or citrate.[1] Oxalates, phytates, large dietary concentrations of calcium, cadmium, iron, molybdenum, orthophosphate,[1] and folates[2] all depress Zn availability.[7] Zinc concentrations are usually higher in legumes than in grasses, but are less available from cereal grains.[1] Calves appear to absorb Zn better than adults.[5]

The signs of Zn deficiency include dermatitis and parakeratosis, depressed milk production, impaired appetite, poor feed utilization, slowed growth, increased susceptibility to footrot, swollen joints, poor growth, impaired Vitamin A metabolism, and increased Vitamin E requirements.[2] When Zn deficiency is suspected, the clinician should carefully sample all constituents of the diet. If zinc deficiency is still considered, serum or plasma should be properly collected into tubes specifically designed for trace mineral analysis (eg royal blue top tubes). The butyl rubber in the stoppers of many tubes or syringes are rich in Zn and will lead to sample contamination.[1] Hemolysis will alter the accuracy of either service or plasma samples, as RBCs have large Zn concentrations.[1] Both polystrene containers and brown paper bags are contaminated with Zn, and caution should be used to minimize their use for sample collection where Zn is suspected. Liver samples give the most reproducible results.[1] Diets containing 30-40ppm Zn are usually sufficient. Trace mineral salt mixes with 0.5 to 2% zinc are usually protective of deficiency.

Conclusions

The mineral mixture of 50% dicalcium phosphate and 50% trace mineral salt with Se added in deficient areas has been used successfully in most scenarios if cows can eat about 2oz daily. Obviously, if the trace mineral salt is diluted with dicalcium phosphate, the trace minerals per ounce are reduced. Since the mineral intake is due to the need or drive for salt, this system may still be adequate. Unfortunately in times of stress, if production demands increase, or if the trace mineral mix is further diluted with unpalatable constituents (eg. MgO, etc), this form of supplementation may not be adequate. The palatability and physical characteristics of the mineral supplement affect its intake. In these cases the clinician should use custom blended trace mineral salt mixes or mineral fortified protein or energy supplements to ensure intake.

With higher production demands for cattle, deficiency are more likely to occur. Whenever deficiency is suspected, it should be confirmed by proper sampling of as many affected animals as possible, and appropriate dietary modification performed.

References

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