INTERRELATIONSHIP BETWEEN COPPER AND BOVINE HEALTH

R. L. Larson, J. D. Arthington, and L. R. Corah

Summary

Trace mineral nutrition is important to production efficiency and animal health. Trace mineral imbalances may be the roots of many diagnosed or undiagnosed problems in a herd. The low cost of a complete mineral analysis when compared to production losses encourages its use in the evaluation of any bovine herd in which trace mineral imbalances are suspected.

A systematic gathering of information on mineral intake, antagonist intake, and serum and tissue values is necessary in order to make a diagnosis and a rational treatment decision. It is important to understand the complex interactions between minerals so that supplementation with one element does not make a complicated situation worse. Because of the many interactions between nutrients and the cost of mineral supplements, recommending use of higher levels of trace minerals in a ration or supplement without a complete diagnosis is economically and nutritionally unjustified.

(Key Words: Copper, Trace Minerals, Immune Function.)

Introduction

The effects of trace mineral imbalances on production and health are becoming better understood, but the information is still far from complete. Because, as their name implies, trace minerals are needed only in small amounts, dietary supplies are usually adequate. But direct or induced trace mineral deficiencies do occur and may be primary underlying problems in infectious or metabolic diseases. Suspected trace mineral problems should be investigated not as a single element problem, but as an imbalance problem involving several minerals. Generally, the trace elements that may be deficient are: cobalt, copper, iodine, manganese, selenium, and zinc. This article will focus on copper deficiencies and the manifestations of this deficiency.

Copper Functions and Signs of Deficiency

Copper is essential for hemoglobin formation and for iron movement. Copper is the rate-limiting element in ceruloplasmin synthesis. Ceruloplasmin is necessary for the oxidation of iron, permitting it to bind with the iron transport protein, transferrin. Ceruloplasmin levels have been shown to be reliable indicators of copper status in cattle. At blood copper levels as low as .3 ppm, ceruloplasmin is virtually absent. Copper is essential in many enzyme systems. By knowing the function of these enzyme systems, the clinical signs seen with copper deficiency are readily explained. The involvement of copper in immune function is well recognized. It is an integral part of the Cu/Zn - superoxide dismutase (SOD) enzyme. This enzyme is the primary scavenger of toxic oxygen radicals, which are produced as a result of normal cellular respiration. Cu/Zn - SOD reduces these radicals to hydrogen peroxide, protecting the body against oxidative damage. Indications of reduced Cu/Zn - SOD activity from copper deficiency may be most evident in cattle under shipping, weather, or handling stress.

Copper deficiencies can be manifested in a variety of disease conditions. One may see reduced fertility in cows and heifers; decreased conception rate, lack of estrus, and fetal re-

sorption. Bulls may have poor quality semen. Iron deficiency anemia and cardiovascular problems occasionally occur in copper-deficient individuals. Ataxia, inability to suckle, incoordination, stiff gait, and other central nervous system signs have been reported in young calves when dams were copper deficient. Slow growth rate or low milk production are commonly seen in copper-deficient animals. Connective tissue pathologies can include improper bone development resulting in fractures, heel cracks, sole abscesses, or foot rot. The most consistent physical finding we have encountered is abnormal hair pigmentation because of decreased tyrosinase activity, resulting in poor conversion of tyrosine to melanin. Red cattle become yellow and black cattle become grey or red tinged, particularly around the eyes, on the tips of the ears, and on the flank. Impaired immune response, manifesting itself as poor response to vaccination, severe parasitism and failure to respond to treatment, has been reported in cattle diagnosed as copper deficient.

Causes of Copper Deficiencies

The causes of trace mineral imbalances are often complex. Among the factors to consider are breed, frame size, and growth rate of the animals and their sources of feed and water. Simmentals may require twice as much copper as Angus. Probably more important than breed is production potential. Cattle of any breed that are heavy milking or fast growing may require higher levels of trace minerals than animals not pushing their genetic potential.

Many times, copper deficiencies are not caused by a primary shortage of copper, but by antagonists to copper absorption or utilization. Probably the best documented copper antagonist is molybdenum. High molybdenum is usually associated with alkaline soil. Liming the soil increases molybdenum uptake by plants, and legumes generally accumulate more molybdenum than grasses. Pasture molybdenum levels are lowest in winter, rising in the spring and peaking in the early fall. More important than actual copper and molybdenum levels is the ratio of these two elements. The dietary Cu:Mo ratio should ideally be between 6:1 and 10:1. Borderline levels are 2 to 3:1 and toxic levels of molybdenum are below 2:1.

High (20 to 30%) protein feeds can reduce copper availability through binding of sulfur-containing amino acids. Sulfur levels of 500 ppm can decrease copper availability by up to 50 percent. This problem is most evident in cows grazing lush spring pastures. It is suggested that for each .05% unit increase of sulfur above the .2% baseline, one should add 5 ppm of copper to the already existing requirement of 8 ppm of copper in the diet. According to the literature, copper deficiencies are most likely in these dietary circumstances: (1) low Cu:Mo ratio, 2:1 or less; (2) Cu deficiency, below 5 ppm; (3) high protein, 20-30% protein in fresh forage; and (4) in some areas, high iron concentrations in forages and water.

Diagnosing Copper Deficiencies

The diagnosis of trace mineral imbalances requires a systematic accumulation of information pertinent to trace mineral interactions and availability.

(1) Water samples should be taken from all sources, and special attention should be paid to copper, nitrate, and sulfate-sulfur levels. Also check iron, calcium, and zinc levels because of their interactions with copper.

(2) Feed samples should be taken, and phosphate, nitrate, copper, molybdenum, iron, and zinc levels evaluated. Total dietary protein should be evaluated for excesses. Forages containing < 3 ppm of copper (DM basis) are considered deficient, whereas 3 to 6 ppm is considered marginal. Feeds containing 8 ppm or more are considered adequate.

(3) Plasma samples for enzyme activity determination should be taken in dark blue top vacutainer tubes with heparin.

(4) Liver copper appears to be a reliable indicator of copper status. Take a softballsized sample at necropsy or slaughter. Biopsies taken from at least 10 head with a Tru-Cut biopsy needle are appropriate for live animals.

(5) Ceruloplasmin synthesis responds to copper availability. Copper content of blood can be estimated by the oxidase activity of ceruloplasmin. A mean value of 69 µg/100 ml of ceruloplasmin-bound copper is reported in cattle.

Dietary Requirement and Treatment

The minimum recommended level of copper is 8 to 10 ppm (DM basis) total diet. In a free-choice trace mineral or salt mix, a level of .1 to .5% copper is required, assuming consumption of 40 g/head/day.

Production levels must be considered when evaluating NRC recommendations for trace mineral supplementation. In situations of high milk production, rapid growth or stress, NRC recommendations are probably not adequate and must be increased. Many minerals, nitrate, sulfate, protein, and plant estrogens are known to reduce copper utilization and must also be accounted for.

Four routes of copper supplementation are available: injection, mineral mix, water source and copper needles in a bolus. Injections raise copper levels quickly and bypass the effect of other elements in the gut. Copper glycinate and copper EDTA are two common injectables. However, injectable products can cause injection site reactions, toxicities, and hemolysis.

Copper supplementation in salt can be in the form of sulfate, chloride, carbonate, acetate, or oxide. These forms vary in the amount of inorganic copper they contain and also in the copper's bioavailability. The mineral mix route is convenient but cannot assure adequate or uniform consumption by individual animals.

Metering devices can be utilized to place deficient elements into the water supply. This route can be effective, if it is the only source of water.

Copper oxide needles in a gelatin capsule are placed in the reticulum where they release of water.

Copper oxide needles in a gelatin capsule are placed in the reticulum where they release copper to be stored in the liver. Serum copper levels will not rise to high levels following bolus administration, but liver levels rise significantly and the effect is a long residual one (> 6 months).

Work at Washington State University indicates that no single level of supplementation was adequate in all copper-deficient herds. In some herds, an annual injection and in others, three or more injections per year were required to maintain copper levels. With copper supplementation in salt, .5% copper sulfate produces adequate copper in some herds, and in others, 3% was necessary to maintain copper levels.

Trace minerals chelated to amino acids are being touted as an answer to trace mineral deficiencies. The standard chelate involves the combination of a mineral with two or more amino acids, forming a stable, neutrally charged, biochemical ring compound. Because of their neutral charge, it is claimed that chelated minerals are not bonded into insoluble forms in the digestive tract. However, very little research has shown a positive effect in cattle production. Chelated mineral supplements are much more expensive than their inorganic counterparts and any benefits must be weighed against the cost.

Before any treatment of trace mineral imbalances is undertaken, one should understand the interactions with other minerals and be careful not to replace an existing problem with another deficiency or toxicity. Identifying and removing, if possible, any antagonists are also necessary for treatment of mineral imbalances.