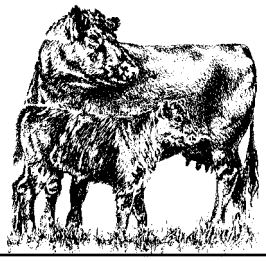

Western Beef Resource Committee *Fourth Edition*

Cattle Producer's Handbook

Animal Health Section 629

Copper: An Essential Micronutrient for Beef Cattle

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Throughout the world and especially within the western United States and western Canada, copper deficiency limits cattle production by reducing growth, reproductive performance, and immune response to infectious diseases. Insufficient copper in the diet is the major cause of this deficiency.

In many geographical areas copper deficiency is caused by an excess of certain other minerals that interfere with copper absorption, function, or both. In these areas copper consumption may be adequate to meet an animal's normal essential needs for copper, but a toxic mineral like molybdenum renders copper unavailable.

Molybdenum or a combination of molybdenum and sulfur are the minerals of greatest concern in interference with copper metabolism. Major relationships between various minerals and organic content in the soil affect the uptake of copper by the plant and the ability of the animal to use its copper. Because of these interrelationships, it is more difficult to manage proper nutritional levels for the beef herd.

Clinical Signs of Copper Deficiency

A variety of conditions can affect the clinical symptoms of copper deficiency. An acute deficiency will cause different clinical symptoms than a long term chronic deficiency. Grazing cattle are more likely to develop a copper deficiency than feedlot cattle. Because of the low copper availability in forages, forage-consuming animals are more likely to become copper deficient. The extent of copper release from forages depends upon forage type and stage of vegetative growth. Cattle grazing mature, low-quality forages during fall and winter or grazing late-harvested hays are more likely to develop a copper deficiency than those grazing in spring and summer.

Clinical signs of copper deficiency include ill thrift and poor growth in the young or loss of body condition in the cow. The haircoat will be rough and faded. The change in hair color is a result of loss of pigment in hair follicles. The hair coat will appear more yellow on a red colored cow or gray on a black colored cow. Some deficient cattle show little change in hair coat color, or change is less evident because of "breed" or breed color.

Another symptom is the immune system becomes compromised, resulting in diarrhea and susceptibility to certain infectious diseases. Lameness, incoordination of movement (ataxia) in newborn calves, spontaneous fractures of bones in mature cattle, and rickets-like condition are frequently observed. Major blood vessels may rupture and hemorrhage.

Muscle tissue of the heart can degenerate because fibrous tissue replaces it. This causes sudden heart failure called "falling disease." Usually less than 5 percent of the cattle herd will die of this sudden heart failure. This condition may become fatal without any prior copper deficiency symptoms.

Cow infertility may be a major problem with copper deficiency. Delayed or depressed estrus or fetal deaths because of fetal hemorrhages and anemia are commonly observed fertility symptoms of a copper deficiency.

These clinical signs may vary with the presence of antagonists or interfering minerals.

Diagnosis of a Copper Deficiency

Patterns of copper deficiency are common to specific geological regions and soil types. Two types of soils have a marked influence on copper uptake by the plant or copper absorption by the animal—muck soils and interference from molybdenum.

Cattle grazing meadows or fed harvested feeds from high-organic-matter and poorly drained soils (muck soils) often develop a copper deficiency. This is especially a problem during late summer and fall, but it can be a problem where cattle are grazing wetland meadows during the spring and summer.

Because of the high organic base, muck soils bind copper, making forage copper levels inadequate for nutritional needs of the cow. Forages along rivers and old lake-bottom areas are prone to copper deficiency. Research has indicated that if you have 25 acres of swampland, even if you have 250 acres of good ground, you will have copper-deficient cattle.

The second soil condition involves the interference from molybdenum. Forage plants may incorporate high levels of molybdenum from high molybdenum soils, especially if the soils are alkaline. Molybdenum can impair the animal's use of copper, especially if the diet is also relatively high in sulfur and iron. A copper-sulfur antagonism lowers the biological availability of copper by forming a copper-molybdenum or copper-molybdenum-sulfur complex in the digestive tract. Forage levels of 3 ppm molybdenum and above will significantly reduce copper availability.

Many soils in the western United States are rich in molybdenum and cause copper deficiency conditions for cattle grazing forages from these lands. See Table 1 for information concerning these minerals in forages and water.

Diagnosis of copper deficiency is inferred from clinical signs, geographical regions, history, serum or liver copper levels, soil type, and plant growing conditions. Definitive diagnosis is based on measured deficient concentrations of copper in either serum or liver.

Other enzymes such as superoxide dismutase, and ceruloplasmin activity will also be reduced in deficiency state. Forage and water analysis for copper, molybdenum, iron, and sulfate content can also help its diagnoses.

Tissue levels of copper are helpful in evaluating the nutritional status for copper, especially liver tissue. Liver is the best measure of current copper status, except in the fetus or newborn calf. The fetus stores copper in its liver at the expense of the dam. In late pregnancy, it is normal for the cow's serum and liver copper levels to decline drastically.

Serum is a more reliable and consistent measure of copper status than is whole blood. Serum copper will not reflect copper stores unless the liver is depleted of its stored copper content. Therefore, low serum copper indicates low copper storage, but normal serum copper levels do not evaluate copper reserves.

To determine copper reserves you need to biopsy liver and analyze it for its copper content. Table 2 presents tissue concentrations of copper for cattle at various nutritional regimes from a severe deficiency to toxicity of copper.

Nutritional Recommendations for Copper

In general, copper is poorly absorbed by the digestive tract. Its absorption is influenced by:

- The age and breed of cattle,
- The amount and chemical form of copper,
- The level of certain minerals such as molybdenum and sulfur in the diet,
- Prior copper status of the cattle, and
- Certain other dietary factors like forage type and stage of vegetative growth, neutral detergent fiber fractions of forages, etc.

Table 2. Tissue copper concentrations for beef cattle under different nutritional regimes.

Nutritional status	Serum copper (ppm)	Liver copper (Wet wt., ppm)
Clinical signs	<0.2	na
Deficient	0.2 to 0.4	0.5 to 10.0
Marginal	0.4 to 0.7	5 to 25
Normal	0.7 to 1.1	25 to 150
Toxic	>1.2	250 to 800

From Wilkes, et al. Diagnosis of copper deficiency in cattle. JAVMA 200:1625-1629, June 1, 1992.

Table 1. Element levels in forages (DM basis) and water.

Nutritional status	Copper (ppm)	Molybdenum (ppm)	Cu:Mo ratio	Sulfur (%)	Iron (ppm)	Sulfur in water (ppm)
Clinical signs	<2		<2			
Deficient	<3 to 5		<2			
Marginal	<10		<3			
Adequate	10	<3	6 to 10			
High	-	>3		>0.2	>300	>500
Toxic	-	>10	>12			

Copper Sources

Choose a copper source having a higher bioavailability. Copper sulfate and certain chelated copper sources are good choices. Table 3 presents the relative bioavailability of various commercial copper sources.

An injectable product (MolyCu) is commercially available. Unfortunately, it causes a moderate to severe tissue reaction at the injection site. Injectable products may provide adequate copper for up to 4 to 6 months.

Another method of supplementation would be through a copper bolus placed directly into the rumen via the esophagus. Companies like Pitman-Moore and Schering-Plough market boluses containing cupric oxide needles. Because of its low bioavailability, cupric oxide slowly releases copper into the rumen content and it will supplement copper for 3 to 6 months.

Table 3. Comparison of commonly used sources of copper in livestock mineral supplements.

Source of copper	% copper in the mineral source	Relative bioavailability (Relative to cupric acetate, %)
Cupric sulfate	25.0	High (89%)
Cupric carbonate	53.0	Intermediate (55%)
Cupric chloride	37.2	Intermediate
Cupric oxide	80.0	Low (0.5%)
Cupric acetate	32.1	High (100%)

Supplementation Recommendations

If the molybdenum content of the diet is less than 1 ppm of the dry matter, then 8 to 10 ppm copper in the diet should be adequate. If the molybdenum content in the diet is greater than 2.5 ppm, 10 ppm may be inadequate and should be increased to 15 ppm or higher. Generally, where molybdenum levels are not toxic, supplements providing 125 to 140 mg copper per head per day are usually adequate.

Some forages like alfalfa accumulate more than 20 ppm molybdenum, which may require a higher copper consumption. Remember, there are major differences in the bioavailability of copper from the various sources of copper. These recommendations generally are based upon using copper sulfate as the copper source.

Remember, copper is a potentially toxic mineral. Many species of animals cannot tolerate high copper consumption, so these recommendations are specific to mature cattle.

Another important consideration concerning copper supplementation is to evaluate the selenium status of the herd. Supplementation with selenium reduces the serum copper concentration and actually increases the amount of copper available to the animal. If selenium is deficient in the cattle herd, supplement both minerals since they interact with each other.

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