

## **Copper Balance in Bison - Are your Bison Getting Enough?**

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First described in the early 1930's, and since recognised worldwide, copper (Cu) deficiency is a well known nutritional problem in domestic ruminants (e.g. cattle, sheep) and deer species. However, it has not been given the attention it deserves in the literature on farmed bison. Every few weeks, I receive a nutrition related telephone call from a bison producer or their veterinarian, and most of these are about probable mineral imbalance or deficiency issues. The signs of Cu deficiency are usually vague and non specific. Chronic diarrhea in spite of testing and treatment for intestinal parasites and bacterial disease is a good example. Many producers want to believe that a stubborn virus or bacteria is at fault, but all too frequently an imbalanced or inadequate mineral intake is at the root of the problem.

Many of the facts written below about Cu metabolism and deficiency come from studies on cattle and sheep and we must make the assumption that these facts are true for bison as well. However, it has been my experience that bison are at least as sensitive as cattle to Cu imbalance, and perhaps more so. Once confined on a farm, bison are not as easy to feed as many people think and nutritional problems, especially mineral imbalances, are not uncommon.

### *Causes of Cu deficiency*

Copper deficiency can be primary or secondary to other factors. Both types result in the same problems for the affected animal, but there is a difference in the approach to correcting either type. Primary Cu deficiency occurs when dietary Cu levels are insufficient to meet metabolic demand. This can happen on diets having low Cu-to-molybdenum ratios. In this type, there simply isn't enough Cu in the food provided for the bison. Diets very high in protein, for instance 20-30 % in fresh forage, are often Cu deficient diets.

Secondary Cu deficiency develops when Cu absorption or metabolism is inadequate. This type may result when dietary factors inhibit the absorption of Cu from the digestive tract. For example, high levels of molybdenum (Mo) in the diet can bind with Cu in the reticulo-rumen creating an insoluble (won't dissolve) Cu molybdate complex. Sulphur compounds bind Cu, forming a non-absorbable Cu sulphate complex, and Cu also reacts with iron on one of the steps to the creation of insoluble complexes of Cu sulphide. These chemical reactions reduce absorption of Cu in the intestinal tract and effectively make Cu unavailable to the animal. Even under normal circumstances only a small fraction (5%) of the Cu eaten by a bison is absorbed by the intestinal tract and, if they persist, high levels of iron, molybdenum, and sulphur create a secondary state of Cu deficiency.

### *Signs of Cu deficiency*

The signs of Cu deficiency are often vague, and appear in various body organs and systems. Many of the body's growth and metabolic processes use the same Cu dependent enzymes, which become ineffective without an adequate supply of Cu. The physical signs connected with a deficiency are due to its effect on enzyme related activities in various body

systems. The enzymes involved have names like lysyl oxidase, cytochrome oxidase, glutathione oxidase, ceruloplasmin, iron transferrin, ferrochelatase, and tyrosinase. Which particular organ system shows signs of failure is determined by which Cu dependent enzymes are affected in a particular animal. Symptoms in ruminants (presumably bison too) can be observed in the gastrointestinal (diarrhea), reproductive (infertility), hematopoetic (blood forming - anemia), skeletal (lameness) and cardiovascular (heart enlargement) systems.

Signs can be dramatic such as in nervous system disorders of calves, or as non-specific as poor growth rates, poor body condition, and changes in hair or coat color. The severity of signs in bison is not a generally good indicator of the degree of deficiency. A faded, scruffy hair coat might be as important as an obviously lame calf, when looking for Cu problems. Moreover, not every animal in a herd will show signs or the same severity of signs. The diagnosis of Cu deficiency is impossible to make without testing a few animals, the feed and water, or both, for Cu and other mineral levels.

An example of Cu dependent enzyme activity can be seen with arthritis and lameness resulting from Cu deficiency. Copper is essential for normal elastin and collagen (connective tissue) production. A lysyl oxidase deficiency from a lack of Cu causes defects in the normal production of cartilage and subchondral (under cartilage) bone. Skeletal abnormalities and lameness resulting from these defects have been documented in young bison.

Similar examples can be found in altered bone marrow production of red blood cells and hemoglobin, and changes to cardiac function and blood vessel formation. Without Cu containing enzymes there is inadequate maintenance of the intestinal tract lining, and disruption of intestinal tract functions. Copper deficiency results in poor semen quality in bison bulls. In cows it causes decreased conception rates, anestrus (no cycling), and decreased productivity. When conception does occur there can be fetal absorption and retarded growth of offspring. Poor calving rates in apparently healthy bison with adequate protein and energy in their diet might simply be from Cu deficiency, although infertility usually has many factors involved and is unlikely to respond to an increase in Cu alone. Pale, scruffy, reddish hair coats from Cu deficiency in bison are actually because of defective enzymes controlling hair growth, keratinisation and pigment production resulting in lighter and weaker hair.

### *Copper and the immune response*

One of the reasons that Cu imbalance can be difficult to identify in bison herds is the fact that marginal or deficient Cu metabolism indirectly results in so many non-specific production problems such as poor weight gain, and what bison legend Doc Throlson calls ADR disease (Ain't Doin' Right) in bison. Thanks to widespread information about AIDS in humans, most readers will be familiar with the concept that serious and complex health problems result from depression of the immune system. Copper deficiency isn't the only cause of inadequate immune function in ruminants, but it is an important one.

Copper plays a role in the effective function of immune system cells called lymphocytes, neutrophils, and macrophages. These cells are involved in non-specific immune functions such as the phagocytosis (eating) and chemical killing of infectious bacteria, and specific immune functions such as the production of antibodies (humoral immunity). Research has shown that neutrophils and macrophages of Cu deficient cattle have a reduced capacity to eat and destroy bacteria. This results from the reduced activity in those enzymes involved in recycling the chemicals necessary to kill bacteria in these cells. Furthermore, problems of this kind in bison may be most evident following stressful situations. In Cu deficient animals there is also

depressed production of lymphocytes in response to immunity stimulating compounds in bacteria and viruses, thereby reducing antibody production.

It is easy to see that bison spend a lot of energy defending themselves against bacteria, viruses and parasites, and that Cu deficient animals are prone to infections that prevent them from fully participating in growth, reproduction, milk production for calves and other important biological activities. Also, diseases like pneumonia, pinkeye, and scours are perhaps more likely to affect Cu deficient animals than others.

### *Dietary requirements for copper*

Cattle diet requirements for Cu have been determined to be a minimum of 8-10 mg/kg (or parts per million, ppm) of feed. This is only the minimum requirement, and the need for Cu can be affected by increased metabolic demand for such things like milk production, growth, and stress. The availability of Cu to meet these demands can be affected by interactions with other minerals, nitrates, sulphates, and protein. The truth is that we don't actually know the minimum requirements for bison. They have never been determined, but we must assume that they are at least that of cattle under the same conditions.

Some average Cu concentrations in feed are: grass hay 10 mg/kg (ppm), legume hay (alfalfa) 9.5 mg/kg, cereal silage 10 mg/kg, oats 3 mg/kg, and barley 5mg/kg. It should be obvious that under most management systems Cu levels in the feed are only just enough, and when mixed rations are fed, mineral supplementation is required. If the feed or water contains molybdenum, iron, or sulphates, then Cu supplements become very important.

A recent case in Saskatchewan resulted in joint abnormalities in yearling bison. The problem was traced to the water supply. Excessive levels of sulphates in the well water, which was the only source of water for the calves, and the pathology findings suggested a diagnosis of secondary Cu deficiency. Absorption of dietary Cu in these cases was impaired by the formation of Cu sulphate by the interaction of Cu in the feed and sulphates in the drinking water. The adult animals on the farm had a similar diet but did not suffer a deficiency because they were free-ranging and used snow as their water source. However, under such conditions dietary intake of Cu could be ensured by way of trace mineral supplementation to a level of 25 mg of Cu per kg of total feed intake.

### *Diagnosis*

A diagnosis of Cu imbalance is made on the basis of history, clinical signs, testing of feed, water, and tissue samples. **Test your feed!** There is no other way to know what minerals, and in what amounts, the feed is providing to your bison. Feed should be evaluated whenever new stocks are brought in until the feed source is predictable. ***You can't tell the actual contents of the feed by just looking at it!*** Yes, this is expensive, but so are infertile or slow growing animals. When analysing feed, the amount of Cu, molybdenum, and sulphur compounds are important. Look for excess phosphorous, nitrate, iron, zinc, and dietary protein. Forages with less than 3 ppm (mg/kg) of Cu are deficient, and those with 3-6 ppm are marginal. Anything greater than 8 ppm might be adequate, if the amount of Cu is greater than 4.5 times that of molybdenum.

Water testing is always indicated when there are signs of Cu deficiency but feed levels are normal. Rule out the possibility of high nitrate or sulphate levels acting as Cu inhibitors. Levels of 6000 ppm (mg/litre) of nitrate and 1000 ppm of sulphate are considered high, and ingestion of water containing 600 mg of sulphate per litre of water (600 ppm) has been reported to induce Cu deficiency in Saskatchewan beef cattle.

Blood testing should include a complete mineral profile. Serum levels are not generally a good indicator of the true Cu status and testing for Cu alone will not provide much information. One has a better opportunity to diagnose herd problems, and more useful information can be obtained, if 10% (or at least more than 10) of the animals in the herd are tested. Be warned that serum concentrations can be normal in spite of obvious clinical symptoms and a Cu deficiency. Serum Cu concentrations of 0.4 to 0.7 ppm (micrograms/ml) are marginal and hard to interpret but anything less than 0.6 ppm might be considered deficient. Serum levels generally won't fall until liver Cu reserves have been used up.

The best tissue to test for an indication of Cu status in the bison is the liver. Liver can be obtained at the time of slaughter of market animals, or from animals dying on the farm in the event of an immediate problem. Save some liver from slaughtered animals to monitor mineral status in your herd. Samples can be frozen and submitted in batches when convenient. Literature on Cu deficiency in cattle and sheep encourage liver biopsies but because of bison behaviour when closely restrained, this is sometimes easier said than done. Having said that, liver biopsy is routinely practised to monitor Cu levels in farmed NZ red deer, and at one time that was thought to be impossible. The stores of Cu in the liver maintain blood concentrations until deficiency is long standing or severe. In cattle, and presumably bison, Cu deficiency occurs at levels less than 40 mg/kg of liver (40 ppm).

### *Treatment*

Injectable Cu compounds may be indicated in severe cases where immediate results are needed. Injectable Cu is Cu glycinate or Cu edetate which is best given according to the manufacturers directions either sub cutaneously or intramuscularly. Injectable Cu has the disadvantage of injection site reactions, toxicity and rupture of red blood cells from overdoses; also, rapid deaths have been reported in cattle.

In most instances, Cu supplementation is best accomplished by mouth. Gelatine capsules full of Cu oxide "needles", which look like a pencil lead, can be given by mouth using a balling gun. This could be a challenge with adult bison, and would require a good bison chute and some courage. Once inside the animal, these Cu oxide "needles" sink into the reticulum chamber of the ruminant stomach and slowly release Cu, providing adequate and uniform supplementation. In cattle, they raise the liver Cu concentration significantly and are relatively long lasting (6 mos).

Otherwise, Cu can be provided in mineral or salt mixes, or through water sources. Water supplementation is effective if minerals are added to the sole water source and there is no alternative for the bison to drink but this can be complicated if the forages being used also have a high mineral content. Feed mineral or salt mixes generally provide inorganic Cu in a variety of forms. Organic sources may have increased bioavailability, but are expensive. These are fed free choice or are added to the total feed intake in the form of formulated supplements. Both ways are convenient but there is no assurance of adequate or uniform consumption, and this can be a problem especially, it seems, with bison. Producers have complained that salt mixes added to feeds such as grain, either blow off in the wind, or remain at the bottom of the feeder after the bison have cleaned up the food. As with salt blocks, some bison will eat it, others won't touch it. It is one thing to provide the minerals but quite another to ensure that they actually eat enough to make a difference. For this reason many producers add Cu supplements in the form of pre-mix containing pellets, or add Cu salts to sweet feed or molasses.

Recommended Cu levels provided in publications can be stated in two different ways; levels in the supplement, and levels in the total ration. A supplement might have Cu at 250 ppm

(250 mg/kg of supplement) but this is not given to the bison directly. The supplement becomes part of the total ration which is mixed so that Cu is present at the currently recommended dose of 25 ppm (25 mg/kg of feed). Recipes for making Cu salts and supplements are beyond the scope of this article, but a qualified animal nutritionist should be able to help you with the arithmetic if you want to mix your own using copper sulphate ( $\text{CuSO}_4$ ) or copper oxide ( $\text{CuO}$ ). The easiest way to formulate a ration that, when consumed in adequate amounts by a bison will provide an adequate daily intake of Cu is to have a feed company do it for you.

In summary, Cu deficiency is not usually about a lack of Cu but an imbalance of Cu and other minerals in the diet. Bison generally need to be supplemented with minerals and failure to do so may inhibit their potential to produce and can lead to illness. Diagnostic testing is expensive, but so are production losses. Remember to routinely have your feed tested for nutritional value. Your hay may look excellent this year, but that is no guarantee that it meets any nutritional standard. Problems are usually not associated with providing supplemental Cu, but with getting bison to eat enough of it.