

# Common Vitamin and Mineral Abnormalities in Beef Cattle

*Jeffery O. Hall, D.V.M., Ph.D., Diplomate A.B.V.T.*  
Utah Veterinary Diagnostic Laboratory  
Department of Animal, Dairy, and Veterinary Sciences  
Utah State University

January 2021

## **Introduction**

Many minerals and vitamins have been proven in research studies to be essential for optimal growth, physiologic function, and productivity in animals. Data from the analytical section of the Utah Veterinary Diagnostic Laboratory would indicate a significant increase in incidence of vitamin and mineral deficiencies during 2009 and 2010. Much of this increase appeared to be associated with producers decreasing or completely stopping the practice of vitamin-mineral supplementation due to the economy and costs. However, significant deficiencies are still observed throughout the US routinely. A common finding with many of the diagnosed deficiencies is a lack of adequate vitamin-mineral supplementation either long term or due to cost cutting.

Increasing incidence of adverse neonatal health effects, due to vitamin or mineral deficiencies, are commonly encountered at the Veterinary Diagnostic Laboratory. A continued lack of or inadequate supplementation results in depletion of body reserves of minerals in the cow and subsequently in poor calf health.

This paper is directed at the health effect of common vitamin and mineral deficiencies and provides a summarization of the most commonly analyzed tissues and fluids that are used for diagnosing specific deficiencies. The paper also touches on immune system effects and appropriate supplementation.

## **Deficiency Diagnoses**

Historically, testing for deficiencies has been performed on diets and/or dietary components to ensure “adequate” concentrations in the diet. However, general mineral analysis does not identify the chemical forms of these minerals, which can dramatically alter their bioavailability and utilization. In addition, certain dietary factors can alter bioavailability of certain minerals as well.

Although not possible for some of the minerals, the most specific means of diagnosing a mineral deficiency is by testing animals for unique functional deficits or deficiencies of specific mineral containing proteins or enzymes. This type of testing is often impractical from a field perspective, due to individual test costs or rigorous

sample handling requirements. But, when possible, this type of testing eliminates the need to know the specific molecular characteristics of a dietary mineral and the potential for competitive interactions of antagonistic minerals for absorption/utilization. For minerals that do not have identified physiologic indices for which testing can be performed, direct quantification from animal tissues or serum may provide a reliable indication of the overall mineral status of the animal or group.

Testing of adequacy of fat soluble vitamins is commonly achieved by testing serum or liver tissue. But, it is essential that serum be separated from the red blood cells soon after collection. In addition, serum should be maintained frozen and protected from sun light while being shipped to the testing laboratory.

Vitamin and mineral deficiencies can be suggestively diagnosed by the development of clinical disease or by post-mortem identification of tissue lesions. But, proof of deficiencies often requires analytical verification since most do not have very unique clinical signs or lesions. In some instances, circumstantial proof of a deficiency can be provided by positive response to supplementation of a suspected deficient vitamins or minerals. But, positive response may have nothing to do with the supplementation and may be just a time responsive correction of some other clinical condition.

An individual vitamin or mineral may have multiple means of measurement for identification of deficiencies, but most have one that is more specific than the others. For example, dietary concentrations may or may not be reflective of the amount that is bioavailable. Or, an individual tissue concentration may or may not reflect functional availability at the target or functional site.

The age of the animal being tested also is important for proper interpretation of status. For example, fetuses accumulate some minerals at different rates during gestation, necessitating adequate aging of the fetus for interpretation. In addition, some minerals, for which little is provided in milk, accumulate at higher concentrations during gestation in order to provide neonates with adequate body reserves for survival until they begin foraging. This is especially prevalent with copper, iron, selenium, and zinc. Thus, the “normal range” for these minerals in body storage tissues would be higher in early neonates than in an adult animal. One must be careful to make sure that the testing laboratory is interpreting the results based on the age of the animals tested, as some laboratories try to interpret all samples as if they were from adult animals.

When individual animals are tested, the prior health status must be considered in interpreting vitamin and mineral concentration of tissues. Disease states can shift mineral from tissues to serum or serum to tissues. For

example, diarrhea can result in significant loss of sodium, potassium, and calcium from the body. Or, acidosis will cause electrolyte shifts between tissues and circulating blood. It is known that infectious disease, stress, fever, endocrine dysfunction, and trauma can alter both tissue and circulating serum/blood concentrations of certain minerals and electrolytes. Thus, evaluation of multiple animals is much more reflective of mineral status within a group than testing individual animals that are ill or have died from other disease states.

### **Live Animal Sampling**

A variety of samples are available from live animals that can be analyzed for vitamin-mineral content. The most common samples from live animals are serum and whole blood. These samples are adequate for measurement of several minerals, but it must be recognized that some disease states, as well as feeding times, can result in altered or fluctuating serum concentrations. Other samples from live animals that are occasionally used for analyses include liver biopsies, urine, and milk. But, since milk mineral content can vary through lactation, vary across lactations, and be affected by disease it is not typically used to evaluate whole animal mineral status. Furthermore, hydration status significantly affects urinary mineral concentrations, rendering it a poor sample for evaluation of mineral status. For Vitamin A and E, serum is the best sample from live animals.

Serum should be separated from the red/white blood cell clot within the 1 to 2 hours of collection. If the serum sets on the clot for longer periods of time, minerals that have higher intracellular content than serum can leach into the serum and falsely increase the serum content. Minerals for which this commonly occurs include potassium and zinc. In addition, hemolysis from both natural disease and due to collection technique can result in increase serum concentrations of iron, magnesium, manganese, potassium, selenium, and zinc. Vitamin A and E can begin breaking down in serum if not separated from the red blood cells and frozen within 1-2 hours of collection. Serum for vitamin A and E analysis should be stored such that breakdown from sunlight exposure does not occur.

The best type of collection tube for serum or whole blood is royal blue-top vacutainer tubes, as they are trace-metal free. Typical red-top clot tubes will give abnormally increased results for zinc content as a zinc containing lubricant is commonly used on the rubber stoppers. For minerals other than zinc or vitamins A and E, serum samples from the typical red-top clot tubes are adequate. Similarly, serum separator tubes are typically adequate for vitamin-mineral analyses, except for zinc.

Samples should be appropriately stored for adequate sample preservation. Liver biopsies, urine, and serum can be stored frozen long term or refrigerated if mineral analysis is to be completed within a few days. Whole blood and milk should be refrigerated but not frozen, as cell lysis or coagulation of solids, respectively, will result in loss of the overall integrity of the sample.

### **Post-Mortem Animal Sampling**

A variety of post-mortem animal samples are available that can be analyzed for vitamin-mineral content. The most common tissue analyzed for mineral content is liver, as it is the primary storage organ for many of the essential minerals. In addition, bone is used as the primary storage organ for calcium, phosphorous, and magnesium. For Vitamin A and E, liver is the tissue of choice for analysis, but it needs to be relatively fresh. Tissue degradation will correspondingly decrease the vitamin A and E present.

Post mortem samples should be stored frozen until analyzed to prevent tissue degradation. If samples are to be analyzed within 1-2 days, they can be stored under refrigerated conditions.

### **Copper Deficiency**

Copper deficiency is one of the most commonly encountered nutritional problems in ruminants, but copper excess is also commonly encountered, especially in sheep or occasionally in dairy cattle. In contrast, copper deficiency is rare in non-ruminants. Clinical signs of deficiency can present as a large array of adverse effects. Reduced growth rates, decreased feed conversion, abomasal ulcers, lameness, poor immune function, sudden death, achromotrichia, and impaired reproductive performance are commonly encountered with copper deficiency.

Cows will do all they can to ensure adequate copper is in calves when they are born. They will actually deplete their own body reserves to ensure neonatal adequacy. As such, neonates diagnosed with copper deficiencies are proof of maternal deficiencies. With copper being an essential component of the immune function, this maternal deficiency likely results in poor colostrums quality and inadequate neonatal protection even in calves that get adequate volumes of colostrums.

The best method for diagnosing copper status is via analysis of liver tissue, although much testing is performed on serum. Deficiency within a herd will result in some animals that have low serum copper concentrations, but serum content does not fall until liver copper is significantly depleted. In herds that have had livers tested and found a high incidence of deficiency, it is not uncommon for a high percentage of the animals to

have “normal” serum concentrations. At the Utah Veterinary Diagnostic Laboratory, it is commonly recommended that 10% of a herd or a minimum of 5-10 animals be tested in order to have a higher probability of diagnosing a copper deficiency via serum quantification. Even with herd deficiency, low serum copper concentrations may only be seen in 10% or more of the individuals. Herds that may be classified as marginally deficient based on liver testing may have predominantly “normal” serum copper concentrations. Thus, serum copper analysis should be viewed as a screening method only. Another factor that can influence diagnosis of copper deficiency in serum is the presence of high serum molybdenum. As the copper-sulfur-molybdenum complex that forms is not physiologically available for tissue use, “normal” serum copper content in the presence of high serum molybdenum should always be considered suspect. In addition, the form of selenium supplementation can alter the normal range for interpretation of serum copper status, with selenite supplemented cows having a lowered normal range for serum copper.

Excessive supplementation of copper in dairy cattle is a relatively common finding at the Utah Veterinary Diagnostic Laboratory. Liver copper concentrations greater than 200 ppm are routinely identified. But, in recent years, several cases of deficiencies also have been identified, due to cessation of mineral supplementation programs. These have most commonly been in first lactation cows that were not adequately supplemented in the growth, breeding, or pre-lactation period.

The recommended adequate liver copper concentration range in adult cattle is 25 to 100 ppm. In comparison, late term fetal or neonatal liver should have 65 to 150 ppm copper to be considered normal.

### **Manganese Deficiency**

Manganese deficiency in ruminants is associated with impaired reproductive function, skeletal abnormalities, and less than optimal productivity. Cystic ovaries, silent heat, reduced conception rates, and abortions are reported reproductive effects. Neonates that are manganese deficient can be weak, small, and develop enlarged joints or limb deformities. Manganese deficiencies in beef cattle are most commonly seen in areas of highly alkaline soils, due to much poorer plant uptake of manganese, but this is not very commonly identified in beef cattle operations.

Manganese at sub-normal to deficient concentrations, although not reported often in beef cattle, is identified routinely in dairy. Of interest is the fact that most testing of beef cattle (greater than 95%) finds normal

manganese concentrations in liver, blood, and serum, but in these same matrices greater than 50%, 75%, and 95%, respectively, of dairy cattle tested are below recommended normal concentrations (unpublished data, Utah Veterinary Diagnostic Laboratory). This may, in part, be due to high calcium and phosphorous content of dairy rations, which can be antagonistic to the bioavailability of manganese.

Of the samples available, liver is the most indicative of whole body status, followed by whole blood and then serum. As red blood cells have higher manganese content than serum, hemolysis can result in increased serum content. Since the normal serum concentration of manganese is quite low, many laboratories do not offer this analysis because of inadequate sensitivity. Overall, response to supplementation has frequently been used as a means of verifying manganese deficiency, but it is critical that a bioavailable form be utilized. For example, manganese oxide has almost no bioavailability.

### **Selenium Deficiency**

As an essential mineral, selenium is commonly identified as deficient in ruminants, but infrequently in dairy cattle. Selenium deficiency is also identified in many non-ruminant species. Selenium deficiency is associated with reduced growth rates, poor feed efficiency, poor immune function, impaired reproductive performance, and damage to muscle tissues. “White muscle disease”, a necrosis and scarring of cardiac and/or skeletal muscle, is linked to severe selenium deficiency; although, it can be caused by vitamin E deficiency as well.

Cows will do all they can to ensure adequate selenium is in calves when they are born. They will actually deplete their own body reserves to ensure neonatal adequacy. As such, neonates diagnosed with selenium deficiencies are proof of maternal deficiencies. With selenium being an essential component of the immune function, this maternal deficiency likely results in poor colostrums quality and inadequate neonatal protection even in calves that get adequate volumes of colostrums.

Diagnosis of a deficiency can be made by analysis of liver, whole blood, or serum for selenium content or by analysis of whole blood for glutathione peroxidase, a selenium dependent enzyme, activity. The most specific analysis is that of whole blood glutathione peroxidase, as it verifies true functional selenium status. Liver is the optimal tissue to analyze for selenium content as it is a primary storage tissue. With serum and whole blood, the former better reflects recent intake, while the latter better reflects longer term intake status. Since seleno-proteins

are incorporated into the red blood cells when they are made and the cells have a long half-life, selenium content of whole blood is a better reflection of intake over the previous months than serum.

In order to adequately diagnose selenium deficiency, the dietary form of the selenium intake by the animals is important. Natural selenium, predominantly in the form of seleno-methionine is metabolized and incorporated into selenium dependent proteins, but can also be incorporated into non-specific proteins in place of methionine. Inorganic selenium is also metabolized and predominantly incorporated into selenium dependent proteins. Thus, “normal” concentrations in serum and whole blood differ depending on whether the dietary selenium is a natural organic form or an inorganic supplement.

The recommended adequate liver selenium concentration range in adult cattle is 0.25 to 0.50 ppm. In comparison, late term fetal or neonatal liver should have 0.35 to 0.75 ppm selenium to be considered normal.

### **Zinc Deficiency**

Zinc is an essential mineral that is required by all cells in animals. Zinc plays a role in numerous enzymatic reactions. Deficiencies of zinc are associated with reduced growth, poor immune function, diminished reproductive performance, and poor offspring viability, as well as skin lesions in severe cases.

Tissue zinc concentrations do not reflect body status well. Of the common samples tested, liver and serum are the best indicators of zinc status. But, serum and liver zinc can be altered by age, infectious diseases, trauma, fever, and stress. Response to zinc supplementation has shown that some animals having low-end normal liver or serum zinc can still show improvement in some clinical conditions. Thus, liver and serum only verify deficiency when these samples have very low zinc content.

Increased numbers and frequency of low zinc status has been found in samples at the Utah Veterinary diagnostic Laboratory from cattle pastured in areas, during and 1 to 2 years following, of severe drought. This could be due to geochemical changes in the zinc form limiting plant uptake.

### **Vitamin A Deficiency**

Vitamin A is an essential fat soluble vitamin in ruminants. It is essential for all cell replications and is especially important in epithelial integrity. It plays an important role in tight junctions between cells, as well as being an important antioxidant in the body and in mucosal secretions. Vitamin A deficiency is associated with

poor growth rates, poor feed intake, poor immune function, poor reproductive performance, and high incidences of diarrhea in calves. Loss of efficient tight junctions in the epithelial cell lining of the digestive tract allows opportunistic pathogens to invade and cause disease.

Vitamin A is provided in the diet via green growing vegetation or supplementation. Dead, brown forages have relatively no Vitamin A content. Thus, for grazing livestock, they must accumulate enough body reserves of vitamin A to carry them through the winter and have enough left to provide adequate vitamin A to their offspring. Therefore, it is more common to see vitamin A deficiencies in the springs after significant drought years, due to decreased time for body reserve accumulation. Unlike minerals, much of the vitamin A provided to the neonate is via the colostrums and in milk fats. Also, early calving has increased the incidence of neonatal vitamin A deficiencies due to lack of green forage for the cows at the time of parturition.

Vitamin A analysis can be efficiently performed on serum or liver tissue. It is important that samples be stored frozen and protected from light to prevent degradation of the vitamin A.

### **Vitamin E Deficiency**

Vitamin E is an essential fat soluble vitamin in ruminants. It is essential for all cells as an important antioxidant in the body in conjunction with selenium. Vitamin E deficiency is associated with poor growth rates, poor immune function, poor reproductive performance, poor muscle function, poor cardiovascular function, and “white muscle disease”.

Vitamin E is provided in the diet via green growing vegetation or supplementation. Dead, brown forages have relatively no Vitamin E content. Thus, for grazing livestock, they must accumulate enough body reserves of vitamin E to carry them through the winter and have enough left to provide adequate vitamin E to their offspring. Therefore, it is more common to see vitamin E deficiencies in the springs after significant drought years, due to decreased time for body reserve accumulation. Much of the vitamin E provided to the neonate is via the colostrums and in milk fats, although it is also transferred, a very small amount, across the placenta. Also, early calving has increased the incidence of neonatal vitamin E deficiencies due to lack of green forage for the cows at the time of parturition.

Vitamin E analysis can be efficiently performed on serum or liver tissue. It is important that samples be stored frozen and protected from light to prevent degradation of the vitamin E.



### **Effects on Immune Status**

Deficiencies in vitamins and minerals have a two part impact on immune function in neonates. Firstly, since neonates are still developing their immune capabilities, these deficiencies have a direct negative impact on that development. And, indirect immune compromise is via the mother's poor immune function. At the time in which it is essential that the mothers be immune competent in order to produce antibodies for the colostrums, when inadequately supplemented they are often deficient due to depletion from the movement of minerals to the fetus. Additionally, poor immune function at the time of vaccination can result in very poor vaccine response, which in turn results in poor immune memory and antibody production necessary for good quality colostrum. Thus, herd deficiencies would be expected to result in poor colostrums quality. This poor quality equates to a higher incidence of disease in the offspring due to poor maternal protection. Often this is seen as high incidence of neonatal diarrheas and/or high incidence of neonatal/juvenile pneumonias.

Case data review at the Utah Veterinary Diagnostic Laboratory has found that over 90% of the calves diagnosed with "summer pneumonia" or "dust pneumonia" have copper deficiency, selenium deficiency, or both. This later summer time period would coincide with the timing of a calf's depletion of body reserves received from the mother, extremely rapid growth rates, and inadequate dietary intake. These factors can leave the calf deficient with impaired immune function, leading to disease susceptibility.

### **Optimization of Supplementation**

There are three basic time periods in which it is critical that vitamin-mineral status is optimal in the animals. Firstly, since the majority of minerals are transferred to the fetus during the last trimester of gestation, the three months prior to parturition are essential for offspring to be born with adequate body reserves and leave the mothers with enough to still have good immune status for colostrum production. Secondly, as these vitamin-mineral deficiencies play a significant role in reproductive health, the time period from parturition to breeding is critical to ensure that the mother's system is replenished for optimal breed-back efficiency. And, thirdly, any time period in which animals are to be vaccinated, one must ensure adequate vitamin-mineral status in order to maximize response

to vaccines. I routinely suggest that animals be on a well balanced vitamin-mineral supplementation plan for a minimum of 30 days prior to any vaccination.

### **Summary**

A variety of samples can be tested for vitamin-mineral content, but may not provide any indication of the overall mineral status of the animal. Appropriate diagnosis of mineral status involves thorough evaluation of groups of animals. The evaluation should include a thorough health history, feeding history, supplementation history, and analysis of several animals for their mineral status.

Dietary mineral evaluation should only be used to augment the mineral evaluation of animal groups. If minerals are deemed to be adequate in the diet, but the animals are found to be deficient, antagonistic interactive effects of other minerals and true average daily per animal intake of the supplements need to be investigated. As an example, high sulfur or iron can cause deficiencies in copper and selenium even when there are adequate concentrations in the diet. Furthermore, if a supplement is formulated for an average of 4 ounces per head per day intake and they are only averaging 1-2, deficiencies can still be present.

Overall, common vitamin-mineral deficiencies are significant hindrances to profitability in the livestock industry. Poor reproductive performance results in increased incidence of culling open cows. Poorer than optimal feed efficiency and weight gain impact the bottom line in terms of pounds of cattle sales. And, poor calf health results in deaths and disease. The resultant increased disease incidence results in lost income in terms of treatment costs and poorer overall growth rates and gains in affected animals. Herds have been followed where deficiencies were observed, then corrected in which breed-back efficiency has improved by 10% or more and weaning weight averages have improved by as much as 30 to 70 pounds per calf or more. These changes amount to significant improvements in profitability in cattle operations.

In its programs and activities, Utah State University does not discriminate based on race, color, religion, sex, national origin, age, genetic information, sexual orientation or gender identity/expression, disability, status as a protected veteran, or any other status protected by University policy or local, state, or federal law. The following individuals have been designated to handle inquiries regarding non-discrimination policies: Executive Director of the Office of Equity, Alison Adams-Perlac, [alison.adams-perlac@usu.edu](mailto:alison.adams-perlac@usu.edu), Title IX Coordinator, Hilary Renshaw, [hilary.renshaw@usu.edu](mailto:hilary.renshaw@usu.edu), Old Main Rm. 161, 435-797-1266. For further information on notice of non-discrimination: U.S. Department of Education, Office for Civil Rights, 303-844-5695, [OCR.Denver@ed.gov](mailto:OCR.Denver@ed.gov). Issued in furtherance of Cooperative Extension work, acts of May 8 and June 30, 1914, in cooperation with the U.S. Department of Agriculture, Kenneth L. White, Vice President for Extension and Agriculture, Utah State University.