

# Central Washington Animal Agriculture Team



Fact Sheet # 1012-2003

## Trace Element Deficiencies in Ruminants

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Copper deficiency and selenium deficiency are the two major problems in Washington State. We have found no evidence of cobalt or zinc deficiencies in this region in our field studies.

### **SELENIUM DEFICIENCY**

A number of diseases or production inefficiency syndromes are associated with selenium and/or vitamin E deficiency. In most of these syndromes selenium and vitamin E appear to be complementary but in terms of recognized deficiencies and response to treatment selenium is the most important.

### **SOIL ASSOCIATIONS**

The selenium content of most soils ranges from 0.1 to 2 parts per million. But in general soil selenium content by itself is not a good measure for the potential for occurrence of selenium deficiency in livestock grazing or consuming forages produced from it. Soil pH has a marked effect on the form of selenium present and its availability to the plant. In alkaline, well-aerated soils with low rainfall selenium forms selenates and organic selenium compounds with good availability to plants. In acid soils ferric iron-selenite complexes are formed with are poorly available to plants. On such soils plants may have adequate or deficient levels of selenium. In soils with very low selenium levels the plants produced are likely to be selenium deficient and the more acid soil the more

likely the deficiency. Selenium deficiency occurs in almost all areas of the Pacific Northwest.

Water, in general, is not a significant source of selenium as selenites are precipitated out with oxides of metals such as iron.

### **PLANT ASSOCIATIONS**

In general grasses contain more selenium than do clovers or alfalfa but on low selenium soils the differences may not be consistent or substantial. Seasonal conditions influence the amount of selenium in plants and when climate favors a lush forage growth deficiencies can occur. Consequently pasture levels tend to be lowest in the spring when the pastures are growing the fastest. Heavy grazing of pastures may also deplete selenium in the plant. Selenium deficiency can be induced by fertilization practices. In particular sulfur containing fertilizers depress selenium uptake by plants. Plant selenium concentrations of less than 0.1 parts per million may be associated with selenium deficiency and concentrations of less than 0.05 have a higher probability. If pasture samples are to be analyzed for selenium content the variation with pasture species and with season must be taken into account. Soil contamination may result in false high levels.

Selenium in grains is variable and depends upon the area of origin. There is no consistent association between grains.

## **VITAMIN E**

Vitamin E has its highest concentration in young green pastures but falls with maturity and can be present in negligible levels in dry summer and autumn feeds. Grains are generally low in vitamin E and levels decline with storage time. In particular levels may be negligible in high moisture grains and feedstuffs preserved with propionic acid or sodium hydroxide. A vitamin E responsive myopathy not associated with selenium deficiency has been reported in sheep on dry summer/autumn pastures in the United States, Australia, and Europe.

## **SYNDROMES ASSOCIATED WITH SELENIUM DEFICIENCY**

### **Nutritional Muscular Dystrophy - White Muscle Disease.**

Congenital white muscle disease occurs more commonly in lambs than in calves. Lambs may be stillborn or show signs within the first few days of life. Where skeletal muscles are involved the syndrome is similar to that described below. Other syndromes may be seen and where cardiac muscle is involved there is frequently sudden death or signs of acute heart failure. Respiratory distress also occurs where there is involvement of the diaphragm and involvement of the muscles associated with sucking can result in death from starvation or outbreaks of apparent aspiration pneumonia.

More typically the disease involves lambs and calves of an older age and the majority of signs are associated with skeletal muscle involvement. Affected animals are reluctant to arise and are frequently found down. If forced they stand with an arched back, move with a stiff gait and frequently show muscle trembling. The muscles of the thigh and back may feel hard and somewhat swollen on palpation. Severe cases may show red urine. White muscle disease in young growing animals frequently follows exertional activity such as movement from winter housing to pastures but may occur spontaneously. Myopathies

in selenium deficient weaners, yearlings and older animals may also occur following excessive exertion.

### **Ill-Thrift.**

A selenium responsive depression in growth rate is recorded in weaner sheep and in sucking calves and weaned calves. The syndrome may be accompanied by diarrhea in a varying proportion of the animals. In sheep there may also be a depression in wool growth. This selenium responsive syndrome usually occurs in the absence of any evidence of clinical or subclinical muscle disease.

### **Retained Placenta.**

Selenium supplementation of dairy cows and beef cows in selenium deficient areas has reduced the incidence of retained placenta and uterine infection.

### **Reproductive Inefficiency.**

A selenium responsive infertility has been recorded in sheep and appears to be associated with a reduction in early embryonic death. Selenium has also been shown to improve sub-optimal reproductive performance in cattle in some areas through an improvement in conceptions to first service.

### **Mastitis.**

Studies in both the United States and Australia have indicated a possible relationship between selenium deficiency and mastitis. In controlled trials selenium supplementation has been shown to reduce the incidence of new infections and to result in a decreased incidence of clinical mastitis and a reduction in the duration of clinical disease.

### **Immunologic Deficiency.**

Neutrophils, peritoneal macrophages and pulmonary alveolar macrophages from selenium deficient animals have low amounts of glutathione peroxidase activity and decreased bactericidal activity. The phagocytic capability of the cells is not diminished but the ability of the cells to destroy the ingested bacteria is

compromised. Selenium has also been reported to enhance antibody synthesis. Impaired immunologic responsiveness may underlie the syndromes above including that of ill-thrift. The possible association of trace element deficiencies with increased disease susceptibility requires further study.

### **DIAGNOSIS OF SELENIUM RESPONSIVE DISEASE**

The diagnosis of selenium deficiency can only really be established by animal tissue analysis and the effect of this deficiency can only really be established by conducting a response trial.

#### **Blood.**

Blood is the most convenient animal material for examination. Analysis can be made for selenium or for the selenium containing enzyme glutathione peroxidase. Liver and kidney can be used for analysis of selenium status but unlike copper deficiency probably offer no advantage to blood analysis.

Vitamin E analysis is not readily available from most laboratories and is expensive. Critical plasma concentrations are 1 mg/ml.

#### **Feed Analysis.**

Feed analysis cannot be used by itself to establish a diagnosis of selenium deficiency. It is of value to establish the level of selenium intake and the possible extent of the problem once a diagnosis has been established by tissue analysis. Seasonal and possible pasture species variations should be taken into account when pasture analysis is attempted. Complete mixed rations analyses are simpler. The minimum requirement for selenium for ruminants is usually placed at 0.1 to 0.2 parts per million of the total ration on a dry matter basis. These should be considered minimal values, as deficiency states can exist at this level of intake.

### **SELENIUM TREATMENT AND SUPPLEMENTATION**

The methods for alleviation of

selenium deficiency include the use of injectable products, the incorporation of selenium and vitamin E into the diet or the use of selenium supplemented salt/mineral mixtures.

Injectable selenium products are available which contain selenium in concentrations which vary from 0.25 mg selenium/ml to 5 mg selenium/ml all products contain 50 mg of D-a-tocopherol/ml. The recommended dose is 2.5 to 3.0 mg selenium/45 kg body weight given intramuscularly or subcutaneously. The amount of vitamin E that is administered at these dose rates is generally insufficient but dose rates should not be increased above the recommended levels because of the risk of selenium toxicity. One injection will give adequate selenium for a period of 30 to 60 days in most instances. The injections can be repeated. The withdrawal period is 30 days.

Selenium can be compounded into the total ration to an added level of 0.3 parts per million on a dry matter basis according to current federal regulations. This form of supplementation is ideal for housed cattle but has obvious limitations in pastured cattle.

Current federal regulations allow selenium to be added to the salt/mineral mix at a level of 120 parts per million for cattle and 90 parts per million for sheep. Supplementation of the salt/mineral mix is a more convenient method of selenium supplementation for cattle on pasture than repeat selenium injections. However, salt intake can be quite variable within a group of animals and there can be considerable variation in the degree of correction obtained. In the Pacific Northwest salt supplementation at this recommended level frequently failed to correct the selenium deficiency.

With all methods of supplementation the efficacy of the level of supplementation, and in the case of injections the duration, should be monitored by whole blood selenium or glutathione peroxidase analysis. It should not be assumed that

because supplementation has been instituted the deficiency has been corrected.

### **SELENIUM TOXICITY**

The toxic dose of selenium by injection for both cattle and sheep is in the region of 1 mg/kg. Acute selenium poisoning may be produced in lambs given a single injection of 5 mg of selenium. This can occur when the wrong selenium preparation is used for injection and with over generous administration. Signs of acute selenium toxicity include blindness, abdominal pain, excessive salivation, paralysis and death after 1 to 7 days. Post mortem examination reveals excessive fluid in the lungs and pleural cavity.

### **COPPER DEFICIENCY**

Copper deficiency may occur as a simple deficiency where the concentrations of copper in the diet are markedly deficient. Copper deficiency can also occur as a conditioned deficiency and in this situation copper concentrations in the diet may be marginal to normal but other constituents of the diet interfere with absorption and utilization of the ingested copper. Prominent amongst these are molybdenum, sulfur and iron. Copper deficiency most commonly occurs, as a conditioned deficiency in Washington State and this is probably true for other areas of the western United States. The clinical syndromes associated with copper deficiency vary from area to area and this variation probably reflects differences in the type of deficiency and the influence of the conditioning factors. Conditions that lead to copper deficiency can be widespread throughout an area or localized to certain fields.

### **SOIL ASSOCIATIONS**

Copper exists in soil in many chemical forms some of which are available to the plant and others not. Total copper in the soil is of little value as a primary diagnostic guide to animal disorders and the usefulness of available copper is also limited.

Severely leached soils, sandy soils, calcareous or alkaline soils, soils with high water tables for a significant portion of the year, and peat and muck soils are soils that are commonly associated with either primary or secondary (conditioned) copper deficiency and should be suspect. In this state the main soil associations have been with alkaline silt loams, soils with high water tables and with peat and muck soils.

Although soil testing is of limited value knowledge of soil type does have important value. We have found that when copper deficiency has been confirmed on a given farm its occurrence on other farms can be predicted (and subsequently confirmed) by the use of soil maps. This has resulted in the detection and correction of copper deficiency problems on farms that were unaware that they had a problem.

### **PLANT ASSOCIATIONS**

Plant associations with copper deficiency are complex because of the interaction between copper and other conditioning substances on the availability and absorption of copper by the ruminant. Of the conditioning factors molybdenum and sulfate are most important. These interact within the gut to form thiomolybdates which complex copper and make it unavailable for utilization.

#### **Copper.**

In general the copper content in grasses ranges from 4-9 parts per million DM. Legumes generally have higher concentrations of copper than grasses under similar growing conditions. Pastures with less than 5 parts per million of copper may be associated with copper deficiency especially if there are significant levels of molybdenum or sulfur. Pastures with less than 2 parts per million copper may be associated with primary copper deficiency. The availability of copper to the animal varies with the type of plant and the stage of maturity. Availability is highest in the mature pastures and in hay and lowest in rapidly growing young pastures. Silages are intermediate.

### Molybdenum.

High molybdenum intakes in the diet can induce copper deficiency even when the copper content of the pasture is quite high. Pasture molybdenum concentrations of greater than 5 parts per million are commonly associated with secondary copper deficiency and lower concentrations (1-4 parts per million) may well be associated if copper values are low and there is also a concurrent intake of sulfur. Molybdenum uptake by plants is higher under high moisture conditions and under soils with high water tables for significant portions of the year. Molybdenum uptake by plants increases with increasing soil pH and can be significantly influenced by fertilization practices such as liming. Legumes uptake more molybdenum than do grasses.

### Sulfur.

The sulfur content of plants is highest during the early growth stages and falls with maturity. Sulfur content of improved pastures is generally higher than that of native or unimproved pastures and copper deficiency can be induced by pasture improvement practices in copper marginal areas. Sulfur concentrations greater than 0.2% may have a risk for copper deficiency.

### **COPPER MOLYBDENUM SULFATE INTERACTIONS**

Experimental work has shown that an increment of 4 mg/kg in dietary molybdenum is sufficient to reduce the availability of dietary copper by 50%. An increment of 1 g sulfur/kg diet has a similar effect on copper availability. Copper deficiency may occur as a primary deficiency when pasture copper levels are low or may occur as a secondary deficiency when molybdenum and/or sulfur levels are high. The copper to molybdenum ratio provides some indication of the potential of the pasture to induce a conditioned copper deficiency. If the Cu:Mo ratio is less than 2 there is a high probability for a conditioned copper deficiency. If it is

between 2 and 5 there is a possibility of deficiency if the copper concentrations are also low or if sulfate is high. Molybdenum and sulfur intakes in surface and well water may need to be taken into consideration.

### Iron.

An intake of 800 parts per million of iron in the diet has been shown to markedly reduce copper availability under experimental circumstances and diets with greater than 400 parts per million of iron may contribute to the limitation of copper availability. Soil ingestion may account for 2-10% of the dry matter intake during certain periods of the year and can result in high iron intakes. Similarly soil contamination of silage can be associated with high iron intakes.

### **SYNDROMES ASSOCIATED WITH COPPER DEFICIENCY**

Depressed growth rate is probably the most common manifestation of copper deficiency and occurs in both sucking and weaned calves and lambs and in yearling cattle. In cattle the effects are most severe in cattle under one year of age but also occur in yearlings. In severe deficiency areas in Washington State we have measured growth depressions as high as 60 to 100 lbs for the grazing season although most were less. The syndrome is frequently accompanied by diarrhea in a variable proportion of the group. Diarrhea is common where high molybdenum pasture concentrations cause a conditioned copper deficiency.

Changes in the coat color of cattle are often cited as early signs of copper deficiency and certainly occur in some copper deficient cattle. In Washington State we have observed these changes in some areas but have not found them in others. The coat of Hereford cattle develops a yellow dun color and Angus cattle show gray discoloration particularly around the eyes. Changes in wool character can be one of the earliest signs of deficiency in sheep. The wool lacks character, develops crimps 3 to 4 times

normal width and has reduced tensile strength. These characteristics are more easily seen in fine wool breeds than Down breeds.

Adult cattle may show no signs of copper deficiency except in severe deficiency states. A proportion may show diarrhea and some in the group may have lowered body condition. Abortion, infertility and lowered milk yield have been associated with copper deficiency in some areas of the world but we have not seen these associations.

The presenting history in some copper deficient cattle herds has been an increased fracture rate particularly involving the scapula, humerus and femur. Young lambs with copper deficiency are frequently osteoporosis.

Anemia has not been a feature of copper deficient cattle in our area although it is recorded as a feature in other area studies. Anemia is common in copper deficient sheep. Swayback-like disease is recorded in calves in North America but is rare.

Increased disease susceptibility may be a feature of copper deficiency. Neutrophils and pulmonary macrophages from copper deficient cattle have been shown to have impairment of bactericidal activity. This impairment occurs relatively early in the development of copper deficiency. Our own field studies and reports from other areas suggest a higher disease prevalence in copper deficient animals but the full importance of copper deficiency to increased disease susceptibility remains to be determined.

### **DIAGNOSIS OF COPPER DEFICIENCY**

The diagnosis of copper deficiency and the establishment of the copper status of the herd must be made by animal tissue analysis (blood, liver). Pasture analysis and knowledge of factors that may affect subsequent copper intake and availability are of value in predicting the possible course of this status. The liver is the main store of copper in the body. Under

conditions of copper deficiency liver concentration of copper will start to decrease before blood concentrations fall. When liver copper concentrations fall to a level of approximately 40 parts per million blood concentrations will start to fall. Cattle can tolerate a period of hypocupremia but if the deficiency persists then clinical hypocuprosis will occur.

### **Blood Analysis.**

The advantage of a blood analysis is that it is the easiest to collect and the cheapest in total cost. Blood concentrations only fall after there has been significant depletion of liver reserves.

### **Hair analysis**

Hair analysis poses problems because of the potential for contamination. Also hair growth takes place over several months. A value of less than 5 mg copper/kg dry matter in a clean recently grown hair sample may indicate an increased risk of hypocuprosis. In our hands hair analysis has proved of limited value as a test for copper deficiency.

### **Liver Copper.**

Liver copper is the best indicator of copper adequacy. It provides a direct measure of the main copper storage area of the body. Its disadvantage is that it requires surgical biopsy. Liver samples have proved of value in monitoring situations where the owner is requested to collect a liver sample from cattle or sheep that have died from any cause. These are stored in the freezer until collected for analysis.

### **FEED ANALYSIS FOR DIAGNOSIS**

In our experience pasture sampling cannot be used to diagnose the occurrence of copper deficiency in cattle. Pasture analysis can be used to determine the nature of the copper deficiency, for example whether it is a primary deficiency or secondary due to high molybdenum and/or sulfate intakes. This information can be of considerable value in determining the level

and frequency of corrective copper supplementation or treatment that is required to alleviate the syndrome. A composite clover sample is probably of greatest value for this purpose; if clovers are not present in the pasture then a composite grass sample taken from several areas in the field is adequate.

Pasture analysis can also be of value in determining the likelihood of copper deficiency but this analysis must be followed by animal tissue analysis. Pasture analysis should cover copper, molybdenum and sulfate and the relative concentrations of each are as important as the individual values.

### **RESPONSE TRIALS**

Seasonal hypocupremia can occur without production or economic significance. The ultimate determination of whether the presence of hypocupremia has clinical or production significance rests with a treatment response trial. In such a trial a treatment group of animals and a non-treated group (control group) are established and the response to treatment or difference between the groups is measured according to the production criteria that are being examined. The length of the trial will vary according to the production parameter that is of interest. In the case of ill-thrift or poor growth rate a significant difference may be observed within a trial lasting 1 or 2 months. In this case it would involve weighing the animals, dividing them into 2 equal groups based on weight and sex, treating one of the groups and taking subsequent weights to determine the response.

Treatment response trials should also include sampling to determine that the level and/or frequency of treatment or supplementation has been sufficient to correct the problem. This is very important, particularly with conditioned copper deficiency, because the levels of copper inclusion in the salt/mineral mix that are required to alleviate the problem can vary

according to concentrations of molybdenum in the diet. Treatment response trials and continual monitoring give this information.

### **CORRECTION OF COPPER DEFICIENCY**

#### **Injection**

Copper glycinate may be given by injection. Currently products are not available.

#### **Salt mineral mix**

Copper supplementation may also be achieved by the addition of copper, for example copper sulfate or copper lysine, to the salt/mineral mix. Most trace mineralized salts contain only 0.02% copper, which is insufficient to correct a conditioned copper deficiency, and additional copper must be added. The response of cattle to copper supplementation in the salt/mineral mix is variable within a group due to variation in salt intake. The level required for any one particular circumstance will need to be determined by local knowledge or a response trial.

Copper is transferred to the fetus in-utero and good fetal reserves of copper can be built up by treatment of the dam during pregnancy. In most situations the reserves will be sufficient to sustain the calf until the time of significant pasture intake at which time interfering substances in the pasture may rapidly induce a copper deficiency and direct supplementation to the calf will be necessary.

Copper is toxic and should not be added to the diet at levels greater than 0.02% without the specific indication of a primary or conditioned deficiency. Sheep are particularly at risk but cattle may also develop copper toxicity.

#### **Copper oxide needles (Copasure)**

These are given as a bolus. The copper needles reside in the rumen and are slowly passed to the abomasums where acid digestion releases copper. They are safe and a single administration will provide sufficient copper for a 6-month period.

## **COPPER TOXICITY**

Pre-ruminant calves and sheep of all ages are susceptible to chronic copper poisoning. Ruminant cattle are relatively resistant.

Copper poisoning occurs most commonly in sheep under conditions of moderate copper intake along with low dietary levels of molybdenum and sulfur. Pasture copper concentrations of greater than 15 parts per million have the risk to induce chronic copper poisoning in sheep if they are ingested over a period of time. Sheep may also develop chronic copper poisoning when they are fed cattle pellets or pig pellets or when they graze pastures, which have high copper contamination such as pastures fertilized with pig slurry.

Chronic copper toxicity can also be a particular problem in housed sheep. Dry feeds, particularly alfalfa, having moderate copper levels (i.e.; 5-8 ppm) but high availability are problematical. Copper contamination of drinking water from copper pipes can also add to the copper load.

Sheep with liver damage from pyrrolizidine alkaloid poisoning are particularly susceptible to chronic copper poisoning.

The disease is commonly precipitated by some stressful procedure and manifests with an acute hemolytic crisis with severe jaundice and red urine.