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Improving Cattle Health Through Trace Mineral Supplementation

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INTRODUCTION

A number of trace minerals are required by beef cattle. Feeds consumed by cattle may supply most trace minerals in adequate amounts. However, some minerals may be severely or at least marginally deficient in beef cattle diets. Even marginal mineral deficiencies can reduce growth, reproduction and/or health of cattle showing few if any clinical signs of deficiency. Other trace minerals such as iron and molybdenum may be naturally present in feeds in levels high enough to reduce animal productivity.

Certain trace minerals affect immunity and may affect disease susceptibility in cattle. Selenium, copper, zinc, cobalt and iron have been shown to alter various components of the immune system. Reduced disease resistance has been observed in ruminants deficient in selenium, copper and cobalt. Trace mineral deficiencies may also reduce the effectiveness of vaccination programs by reducing the ability of the animal's immune system to respond following vaccination.

Calves that have been stressed due to recent weaning and shipping exhibit lowered immunity and increased disease susceptibility; therefore, an adequate supply of trace minerals is especially critical in beef cattle receiving diets for stocker cattle. Feed intake is decreased in stressed cattle and the level of certain minerals may need to be increased to compensate for the low feed intake. Furthermore, nutritional status of calves prior to weaning and shipping may greatly influence health problems shortly after shipping. Calves deficient or marginally deficient in certain trace elements are likely to be more susceptible to infectious diseases.

COPPER

In many areas of the United States, copper deficiency can be a major problem in beef cattle. In most instances, copper deficiency results from the presence of high levels of other minerals (sulfur, molybdenum or iron) in the forage that interfere with copper utilization rather than a simply deficiency of copper in the diet.

Depigmentation or bleaching of hair is usually the earliest visual sign of copper deficiency. In black cattle, the hair coat will turn a russet or pale color. The hair coat of red cattle will take on a dull, dead-like appearance when copper is deficient. Other signs of copper deficiency that may be seen in cattle include: 1) poor growth, 2) rough hair coat, 3) fragile bones, 4) diarrhea, 5) low reproduction, and 6) cardiac failure. Severe copper deficiency can result in
sudden death due to heart failure. Sudden deaths usually occur after physical exertion or excitement.

Copper deficiency may also reduce immunity and resistance to infectious diseases. When foreign materials such as infectious organisms enter the body, they are first subjected to phagocytic cells (neutrophils in blood and macrophages in tissues), which serve to bind, ingest and destroy foreign materials. Neutrophils isolated from copper-deficient cattle have a reduced ability to kill yeast organisms (Boyne and Arthur, 1981). Copper deficiency induced by feeding cattle either 5 ppm of molybdenum or 500 ppm of iron also impaired neutrophil function (Boyne and Arthur, 1986).

Low copper status has been associated with abomasal ulcers in calves (Libbey et al., 1985). Clinical signs of respiratory disease following inoculation with IBR virus and *Pasteurella hemolytica* were similar in copper-deficient and copper-adequate calves (Stabel et al., 1993). However, copper deficiency in lambs grazing improved pastures resulted in increased susceptibility to bacterial infections and greater mortality (Woolliams et al., 1986). Over a 2-year period, death losses in lambs from birth to 24 weeks of age were much higher in a low copper line compared to a high copper line (28 vs 12%). Lamb survival was enhanced in the low copper line but not in the high copper line by administration of copper at 6 weeks of age. Many of the lamb deaths were associated with bacterial infections, with *Pasteurella hemolytica* and *E. coli* being the most commonly isolated organisms.

Copper requirements can vary more than any other trace mineral. High forage concentrations of molybdenum and sulfur greatly increase copper requirements. The negative action of molybdenum on copper utilization is increased when sulfur is also high. The ratio of copper to molybdenum in the diet should be at least 3:1 to prevent copper deficiency or molybdenum toxicity. Even when molybdenum concentrations are low, sulfur levels well within the normal range (.2 to .4%) found in forages can reduce copper absorption. High dietary iron may also contribute to the development of copper deficiency. Studies with growing cattle have shown that feeding iron at levels as low as 500 ppm (typical of those often found in forages) can reduce copper status of cattle.

Some breeds of cattle also appear to have higher requirements than others. Simmental and Charolais cows and their calves had low plasma copper concentrations than Angus cattle when fed the same diets (Ward et al., 1995). Clinical signs of copper deficiency were also more apparent in Simmental than in Angus calves. Recently, we measured liver copper in weanling Simmental and Angus steers that had been managed and fed together since birth. Angus steers had liver copper concentrations over twice as high as those observed in Simmental.

ZINC

Severe zinc deficiency in cattle results in reduced growth, reduced feed intake, loss of hair, skin lesions that are most severe on the legs, neck, head and around the nostrils, excessive salivation, swollen feed with open, scaly lesions, and impaired reproduction. A deficiency of zinc in males reduces testicular development and sperm production. In females, cycling and
conception rate are decreased by zinc deficiency. Severe zinc deficiency is rare, but has been observed in ruminants grazing forages. The extent that marginal or subclinical zinc deficiency exists is unknown, but is likely more widespread. Based on zinc supplementation studies, subclinical zinc deficiency can result in impaired reproduction and decreased weight gains (Spears, 1994).

Zinc deficiency is known to severely impair immune function in laboratory animals such as the rat. A genetic disorder of zinc metabolism has been reported in Holstein and Shorthorn calves that results in severe zinc deficiency due to impaired ability to absorb zinc. Calves with this disorder show suppressed cell-mediated immune response (Perryman et al., 1989). In lambs, a marginal zinc deficiency did not affect immune responses (Droke and Spears, 1993).

**SELENIUM**

White muscle disease in young calves is a common clinical sign of selenium deficiency that results in damage in both skeletal and cardiac muscle. Affected animals may show stiffness, lameness, or even cardiac failure. Other signs of selenium deficiency that have been observed include unthriftiness (often with weight loss and diarrhea), anemia, and increased incidence of retained placenta. Selenium deficiency is a major problem in many areas despite the relatively small (.1 to .3 ppm) amount of this trace mineral required by cattle.

Studies have indicated that selenium deficiency can affect the ability of neutrophils to kill microorganisms (Boyne and Arthur, 1981) as well as antibody production following a disease challenge (Reffett et al., 1988). A 2-year study with beef cows and calves receiving pasture and corn silage marginally deficient in selenium (.03 to .05 ppm) indicated that bimonthly selenium-vitamin E injections reduced calf death losses (4.2 vs 15.3%) from birth to weaning (Spears et al., 1986). Most of the deaths in this study were attributed to diarrhea and subsequent unthriftiness. It is evident from this study that increased death losses due to selenium deficiency could easily go unnoticed under practical conditions.

Stabel et al. (1989) studied the effect of selenium status and stress on disease resistance in calves. Selenium-adequate calves were born to selenium-supplemented cows and were supplemented with selenium after birth, whereas marginally deficient calves were born to unsupplemented dams and did not receive supplemental selenium. Calves were stressed by weaning and transporting to a new location. On day 3 after shipping, half of the calves in each treatment were inoculated with *Pasteurella haemolytica*. All calves including those not inoculated showed morbidity signs including depression, increased body temperature and depressed feed intake. However, selenium deficiency did not affect the severity of the disease. Injecting stressed steers with selenium on arrival at the feedlot also did not improve health status during the stress period (Droke and Loerch, 1989).

**MOLYBDENUM**

Recent research suggests that some abnormalities normally attributed to copper deficiency may actually be due to molybdenum toxicity. It has been known for some time that
molybdenum toxicity occurs when molybdenum intake is excessively high (20 ppm or higher) and that toxicity can be overcome by providing additional copper. The addition of 5 ppm of molybdenum to a diet low in molybdenum reduced growth and feed efficiency and caused infertility in heifers (Phillippo et al., 1987). Weaning weights were reduced in calves raised from cows fed diets supplemented with 5 ppm of molybdenum (Gengelbach et al., 1994). In these same experiments, cattle fed high levels of iron had similar copper status to those fed molybdenum but did not show reduced gain or infertility. The level of molybdenum supplemented (5 ppm) in these studies is certainly within the range of molybdenum concentrations that occur in forages. Providing additional supplemental copper will generally prevent or correct adverse effects due to molybdenum.

**IRON**

Iron deficiency has decreased immunity. However, iron deficiency in grazing cattle is unlikely unless parasite infestations or diseases exist that cause chronic blood loss. In fact, cattle grazing pastures or being fed harvested silage or hay may be exposed to excessive levels of iron through forage, water or soil ingestion. The possibility of high iron contributing to copper deficiency has already been discussed. High intakes of iron may also result in cattle being more susceptible to various infectious diseases. Disease causing organisms require iron for their growth. Iron is normally bound in the body in such a manner that microorganisms have difficulty in obtaining iron for their growth. However, high dietary iron may increase the incidence of disease, because iron becomes more available for microbial growth when iron levels in the body are elevated. Long term exposure to high levels of iron can also result in tissue damage, especially in the liver and spleen.

**CHELATED TRACE MINERALS**

There has been increased interest in the use of chelated or organic trace minerals in beef cattle mineral supplements in recent years. Limited research suggest that certain organic trace minerals may enhance the immune response or improve health above that noted in animals fed inorganic trace minerals.

In a 3-year study with steers grazing pasture, zinc methionine inclusion in the mineral mix reduced incidence of footrot from 5.4 to 2.5% (Brazle, 1993). Spears et al. (1991) studied the effect of zinc level and source on immune response to vaccination in stressed steers that had recently been weaned and shipped. Steers were fed a control diet that contained 26 ppm of zinc or the control diet supplemented with 25 ppm of zinc from either zinc oxide or zinc methionine. Antibody titers were determined on serum samples collected on days 0 and 14 as a measure of the immune response to IBR and PI3 vaccination. Antibody titers against IBR on day 14 following vaccination were 47 and 31% higher in steers supplemented with zinc methionine compared to control and zinc oxide fed steers, respectively. Calves experimentally challenged with IBR virus tended to recover from the disease more rapidly when fed zinc methionine compared to zinc oxide (Chirase et al., 1991). Antibody titers to IBR were higher in stressed steers fed copper proteinate compared to those fed copper sulfate following vaccination against IBR (Nockels, 1991).
We have conducted two studies to determine the effect of feeding zinc methionine to calves prior to weaning on health and performance postweaning and shipping. Calves were weaned in North Carolina and immediately shipped to Amarillo, Texas. Zinc methionine was compared to zinc oxide and cattle received the same zinc source on arrival at the feedlot. In both studies, calves fed zinc methionine consumed more feed and tended to gain faster during the postshipping period. Morbidity rate tended to be lower for zinc methionine supplemented calves in one of the two studies.

LITERATURE CITED


