Trace Mineral Deficiencies in Cattle: A Review

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SUMMARY

This review discusses the biological role of copper, zinc and selenium in ruminant nutrition. Soil, climate, plant and animal interactions are discussed in relationship to trace mineral availability. A review of the clinical symptoms associated with copper, zinc and selenium deficiences are also presented.

RÉSUMÉ

Déficiences en oligoéléments, chez les bovins

Cet article présente le rôle biologique du cuivre, du zinc et du sélénium, dans la nutrition des ruminants. Il explique aussi les relations entre le sol, le climat, la plante et l'animal, par rapport à la disponibilité des oligoéléments. Il commente enfin les signes cliniques qui accompagnent une déficience en cuivre, en zinc ou en sélénium.

INTRODUCTION

Although trace minerals comprise less than 0.01% of the total mass of an organism, many are essential for normal function (17). A trace mineral is considered essential if its deficiency results in an impairment of function (17,62) or if its withdrawal from the body induces the same structural and physiological abnormalities regardless of species (17,62). These abnormalities are accompanied by specific biochemical changes, which can be prevented or cured, once the deficiency is corrected (62).

There are fifteen trace minerals considered essential in mammalian nutrition: arsenic, chromium, cobalt, copper, fluorine, iodine, iron, manganese, molybdenum, nickel, selenium, silicon, tin, vanadium, and zinc (59). These are classified according to their biological function. In enzymes, they act as a cofactor or as an essential part of enzyme structure. They maintain protein and nucleic acid sterochemical structure. They participate in ionic interactions affecting cell permeability and mineral matrix, and act as direct catalysts in the promotion of reactions.

These biochemical functions are frequently associated with metalloenzyme complexes or with metal ion activated enzymes (17).

SOIL AND PLANT IMPLICATIONS IN TRACE MINERAL AVAILABILITY

The trace mineral content of our natural feeds is determined primarily by the mineral availability from the soil and secondly by the actual mineral composition of soil (18,65). Factors which influence the mineral composition of the soils are: the origin of the parent rock (18), glaciation, leaching, surface erosion, evaporation, salinization, the application to the soil of pesticides, fertilizers, trace elements, manures and sludges, and the aerial fallout from industry and transportation (20,30,65). Soil factors, such as acidity, moisture or drainage conditions, temperatures and seasonal effects influence mineral uptake by crops and pastures (30).

The availability of minerals in soil depends upon their effective concentration in soil solution (28). This concentration is influenced by pH, moisture, organic matter, leaching, the presence of other elements and the microbial activity of the soil (8,65). Alkaline soils lead to an increased biological availability of some trace elements such as selenium and molybdenum (8,65). With decreasing soil pH, selenium is less available, but the uptake of some cationic metals such as copper is increased (8,65). Some trace minerals form insoluble complexes with the organic matter in the soil; copper is one of these (65). Soil leaching, erosion and long term cropping lead to a depletion of biologically active trace minerals. Other elements in the soil can form complexes with the minerals. An example of this is sulphate which will inhibit the uptake of selenate and selenite from the soil (8).

The soil zones and the origin of the soils have little influence on the availability of most trace minerals except selenium. The selenium content is high in plants grown on soils derived from recently exposed sedimentary rock and deficient in plants grown on soils from igneous rock (8).

In western Canada the highest incidence of selenium deficient grains and roughages were found in the dark brown, black and dark grey wooded zones (44,45).

Crop management and climatic conditions also influence the eventual trace mineral level in the feeds. Fertilization and/or heavy rainfall can result in lush pasture growth and the dilution of some trace minerals (8). In one study, nitrogen fertilizer increased the copper content of the forage (43). The stage of plant maturity and method of forage handling influence the availability of trace elements to the animal. In immature pastures, copper is not as available as in dried forages cut at the same stage of maturity (30). As the plant matures there is also a gradual decline in the trace mineral content, particularly copper and zinc (22).

The type of cultivar can influence the trace mineral content (22). Some

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plants are known for their selenium and molybdenum concentrating ability, while alfalfa and other legumes do not readily take up selenium (8).

In a study carried out to measure the mineral content of Saskatchewan feed grains, selenium and copper values were extremely variable. The selenium levels varied according to soil zones and the soil type. The selenium values were higher in clay soils than in loam soils. Seasonal effects influenced copper levels (46).

Results from a study in Saskatchewan indicate that 60% of the alfalfa hay, silage and brome hay analyzed were deficient in copper (less than 10 mg/kg of dry matter), and 100% were deficient in zinc (less than 40 mg/kg of dry matter). Copper was also deficient in all the clover silage, barley and oat grain samples. Deficient zinc levels were found in 88% of the barley, 40% of the wheat and 93% of the oat samples (11).

When a veterinarian is called to investigate a suspected trace mineral problem, he must realize that differences in trace mineral levels are local and related to cultivar varieties, soil conditions, climate and fertilizing practices. There can also be extreme variation in soil type within a field. The knolls may be glacial till and the lower areas alluvial deposits high in trace minerals. It is essential when taking feed and pasture samples to keep these facts in mind.

COPPER

Copper deficiency was first reported in Canada in the early 1950's (62). It has since been diagnosed in regions of British Columbia, Manitoba, Ontario, Saskatchewan and Alberta (4,14,25,41, 50,51).

Copper deficiency occurs in cattle either as a primary or secondary problem. In a primary deficiency, there is a decreased level of copper in the diet. In a secondary deficiency, there is a failure of copper absorption or utilization caused by an imbalance or excess of other elements in the ration (59).

A copper: molybdenum ratio of 2:1 in the feed is considered normal. Higher levels of molybdenum tend to bind copper, making it unavailable for absorption (36,40). Excess sulphur in the diet tends to exacerbate molybdenosis (17,35,59). Sulphates can also exert a predominant and independent effect on copper availability (21,34, 63). Copper forms a complex with molybdenum and sulphates (copper thiomolybdate) which is absorbed but unavailable and excreted through the kidney. Increments of 4 mg/kg of dietary molybdenum depressed copper availability by 50%. A similar decrease occurred with the addition of 1 g sulphur/kg of diet. Both organic and inorganic sulphur were effective in inhibiting copper repletion in hypocupremic ewes (30). It is important to determine the total dietary concentration of molybdenum, inorganic sulphates and copper, so that a better interpretation of copper availability can be made (21,64). When determining the level of dietary sulphates, one must remember that the water supply could be a significant contributing factor. The level of sulphur in the rumen also depends on the protein level in the ration and the protein solubility (42). During periods of rapid pasture growth, protein level and protein solubility are high, making the copper in the forage less available (60).

High levels of iron over extended periods of time have an influence on copper availability. Ground water or ingestion of soil, during periods of high grazing are common sources of iron (9). The level of dietary calcium, cadmium, zinc and the chemical form of copper in the diet also influence copper availability (60,63).

As there is little placental transfer of copper, the calf is dependent on the copper in the colostrum as its initial source (59). The level of copper in milk drops significantly 48 hours after calving (15,59). After weaning there is a significant decline in the absorption of copper from the small intestine (33). Generally less than 30% of the copper consumed is absorbed (7).

As the available copper diminishes, certain physiological processes fail as competition for available supplies occur. Copper deficiency produces clinical signs which are generally related to its role as a catalyst or as an essential component of various metallo enzymes or metal activated enzyme systems (17).

Anemia in cattle is a feature in some cases. This has been reported as a hypochromic macrocytic anemia (60). In our experience the calves examined at The Western College of Veterinary Medicine have consistently had a mild microcytic normochromic anemia. Copper is essential for erythrocyte production and the maintenance of their integrity in the circulation (60). In copper deficiency there is an impairment of iron release from the reticuloendothelial cells, because of a decreased activity or production of ceruloplasmin and ferroxidase II. Thus, the iron is not available for erythrocyte production (17,59). In New Zealand, copper deficiency has been implicated as a factor in postparturient hemoglobinuria through its role in maintaining the red cell integrity (52).

Skeletal lesions have been described in calves. These are either spontaneous fractures or physeal dysplasia (13,25, 39,50,51,53,60,61). The most commonly recognized lesion is physeal dysplasia (epiphysitis). The affected calves are usually stiff and lame with a marked swelling at the distal metatarsal and metacarpal physis. The biochemical lesion is a decrease in amine or lysyl oxidase; enzymes responsible for cross linkages of collagen and thus the stability and strength of bone (26,55,56, 60).

In copper deficiency, there is a breakdown in the conversion of tyrosine to melanin because of reduced amino oxidase activity resulting in achromatricia (60).

Myocardial degeneration and fibrosis associated with acute heart failure has been described. This has been associated with a reduction in cytochrome oxidase (60). The scouring associated with copper deficiency has been also related to the depletion of cytochrome oxidase in the small intestine resulting in partial villus atrophy (60).

Ninety percent of plasma copper is bound to an α_2 globulin, ceruloplasmin, some is bound to albumin, which acts as a true transporter of copper and a small portion is bound to amino acids (59). Normal plasma copper levels are considered to be 0.7-1.5 mg/L (59) There appears to be no significant change in the levels related to feed consumption or short term stress. Chronic stress or infections can lead to an increase of plasma copper (46). Plasma levels alone are not totally reliable in the diagnosis of copper deficiency. Plasma levels should be done in conjunction with liver levels wherever possible. Plasma

ceruloplasmin levels correlate well with plasma copper levels and are of value in the assessment of the copper status (60). Plasma copper levels consistently below 0.5 mg/L are strongly correlated to liver levels less than 40 ppm on a dry matter basis (60). In a survey carried out in Saskatchewan, 67% of slaughter cattle livers had less than 10 ppm of copper on a wet weight basis (normal 30-100 ppm) (6).

Copper levels in the hair are subjected to high individual variation and to environmental contamination. Hair analyses should only be used in conjunction with the entire picture and should be limited to recently grown hair (60).

To treat copper deficiencies the aim is to maintain adequate liver levels either by increasing the dietary intake or by parentral copper injections (1,54). When deciding on the method and amount of supplementation the practitioner must determine whether a primary or secondary copper deficiency exists.

ZINC

Zinc was first shown to be essential for living organisms in 1869. With the advent of purified zinc deficient diets in 1940, zinc deficiency syndrome could be consistently produced in experimental animals (17).

Zinc deficiency has been reproduced experimentally in calves and cows (37,38,49). In calves the clinical signs are a stiff gait, swelling of the hocks and knees, subcutaneous fluid accumulation and parakeratosis of the skin. Parakeratosis of the rumen epithileum, delayed wound healing, reduction in thymic size and defects in cell mediated immunity have also been described (23,37). An autosomal recessive trait has been described in the Holstein-Friesian breed of cattle. It is a single metabolic fault where there is failure of intestinal absorption of zinc. The calves are stunted, lethargic and subject to skin disorders. This condition parallels acrodermatitis enteropathica, an inherited zinc deficiency in man (23). In cows, the signs are parakeratosis around the dew claws and heel bulbs. This eventually extends up the hock and between the legs. A dermatosis also develops at the base of the teats (49). Decreased fertility and abnormal estrus behavior has been reported in cows, and retarded testicular growth in bulls (38,60).

The edematous swelling of the coronets of the rear hooves and infectious pododermatitis in dairy cattle has been related to zinc deficiency (26). Bulls with infectious pododermatitis given a zinc supplement responded favorably. Zinc has also been used as a prophylactic treatment for pasture footrot (26).

It has been suggested in several species that zinc deficiency interferes with hepatic synthesis of retinol binding protein, resulting in an impaired mobilization of retinol from the liver (19). Although this has not been studied, it is interesting to speculate what effect a marginal zinc status has on vitamin A availability in cattle.

Zinc is involved in protein synthesis, carbohydrate metabolism, and nucleic acid metabolism, through its association with enzyme systems either as a metalloenzyme or as an activator of enzymes (17,38).

Zinc is absorbed in cattle from the abomasum and small intestine (5,60). The intestinal lumen and mucosa contain a low molecular weight zinc binding factor (13,35,60). Zinc transferred to the fetal calf at the end of gestation is about 13.5% of the absorbed zinc (27).

Several factors influence zinc absorption and availability. In calves there is a greater net absorption of zinc prior to weaning (35). Some factors that influence zinc absorption in monogastrics may be important in cattle. High dietary calcium potentiates zinc deficiency. This effect may be mediated by the phytic acid and inorganic phosphorus levels in the diet (35). Different dietary sources of protein, particularly proteins of plant seed origin, require higher dietary zinc levels (35). Copper, cadmium, iron and molybdenum may impair zinc metabolism but the interactions are poorly understood (35).

In cattle parameters have been measured in an attempt to establish criteria for the diagnosis of zinc deficiency. No single parameter has proven to be satisfactory. The content of zinc in milk varies with the stage of lactation and the zinc status of the animal. Colostrum is rich in zinc (60).

Numerous factors will influence the zinc level in blood, making serum or plasma determinations an unsuitable method for diagnosing zinc deficiency. Hemolysis of a blood sample increases the zinc level; stress to the animal and physical illness decrease the plasma zinc levels (60,61).

A decline in plasma protein levels, a reduced alkaline phosphatase activity, and subnormal levels of carbonic anhydrase have been reported in zinc deficient animals but these findings are not consistent (60).

SELENIUM

Selenium was first recognized as an essential trace mineral in 1957, when found to prevent liver necrosis in rats (47,48). Subsequently, selenium deficiency, affecting a number of systems and producing a variety of lesions, has been seen in swine, poultry, horses, sheep, and cattle (8,29,31,34,47,60). In cattle, white muscle disease is the most commonly recognized problem (29,31, 60). Failure of reproductive function and a high incidence of retained placentas have been associated with selenium deficient rations. In some studies supplementation with selenium, selenium vitamin E or selenium and increased protein, significantly reduced the incidence of retained placentas (35,58). Other studies do not confirm these findings (24).

A metabolic interrelationship between selenium, vitamin E and sulphur amino acids exists at the cellular level (8,61,67). Selenium functions in the cytosol through glutathione peroxidase (GSH-Px) (8,16). Glutathione peroxidase uses glutathione, a tripeptide with a sulphur, to reduce hydrogen peroxide and organic hydroperoxides to less harmful products (67).

Glutathione peroxidase, through its role in the metabolism of hydroperoxides, may be involved in the synthesis of various prostaglandin derivatives (67). Vitamin E acts as a lipid soluble antioxidant in the cell membrane (8,16).

The most widely used assessment of selenium status is blood selenium levels (8,60). Low blood selenium is always found in selenium deficient conditions (8). A direct relationship between blood GSH-Px activity and selenium concentrations has been established (3,10,28,57,66). Selenium is incorporated into the erythrocyte GSH-Px at the time of erythropoiesis.

Thus GSH-Px levels are less effected by daily variations in the dietary level (2). Milk and tissue concentrations of selenium in general reflect the dietary intake (11,60). When the diet is adequate in naturally occurring selenium, added dietary selenium does not result in a significant increase in milk levels (12). Selenium levels in hair have also been used as an indicator of selenium status (60).

The intestinal absorption of selenium is not homeostatically controlled and a specific mechanism for selenium storage has not been recognized (8).

The animal's ability to assimilate selenium from its diet is influenced by a number of factors. The chemical form in the diet is important, selenite being more readily absorbed than the organic form (60). The selenoamino acids pass through the placenta of ewes more readily than inorganic selenium compounds (8,26,59). Heavy metals, particularly arsenic and mercury, interfere with selenium absorption and availability (8,60).

A selenium level of 0.1 mg/kg of dry matter in the ration is considered satisfactory under most conditions for growing animals (30,32,60). This can be achieved, a) by adding selenium salts to the ration, the mineral mix or salt mix, b) by parentral administration of selenium, or c) the application of selenium to the soil.

DIAGNOSIS AND TREATMENT OF TRACE MINERAL DEFICIENCIES

The diagnosis of trace mineral deficiencies is based on a complete history, an evaluation of the animals involved, feed and water analysis and the appropriate blood and tissue analyses. No single criteria used alone is adequate. A veterinarian suspecting a trace mineral deficiency should check with the local veterinary diagnostic laboratory on how to handle the samples. When interpreting the results, he should refer to the normal values obtained from that laboratory.

When estimating the minimum needs or maximum tolerances of trace minerals one must consider the extent to which other elements or compounds affect their absorption, retention and utilization. Estimates of adequacy also vary with the criteria applied, whether it be for growth, maintenance or reproductive performance (61). Recommended dietary minimums and maximums are outlined in Table I.

TABLE I
RECOMMENDED UPPER AND LOWER LIMITS OF TRACE MINERALS*
(mg/kg OF DRY MATTER IN TOTAL RATION)

Species	Beef Cattle		Dairy Cattle	
Mineral	Lower limit	Upper limit	Lower limit	Upper limit
Iron	30	400	100	400
Manganese	25	850	25	850
Cobalt	0.1	10	0.1	10
Iodine	0.6	20	0.6	20
Sulphur	1000	3000	2000	6000
Copper	10	100	10	100
Zinc	50	500	50	500
Selenium	0.1	5	0.1	5

*Nutrition Council of the Canadian Feed Industry Association (CFIA)

CONCLUSION

One of the questions often asked by practitioners is "Why is there an apparent increase in trace mineral deficiencies in Canada?" The answers are: a) improved diagnostic and analytical methods available, b) improved genetic selection of livestock for increased growth rates and higher production potentials, c) changes in the traditional cropping practices that have been altered by poor soil management, improved fertilization methods or improved plant breeding and d) modification of traditional feeding programs to improve the feed to gain ratio by newer animal production techniques, feed additives and synthetic ingredients.

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