Small ruminant vitamins and minerals

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Abstract

Nutrition is paramount to small ruminant health and production. All ruminants, though similar, vary in nutrient requirements and predisposition to nutritional toxicities and deficiencies. Therefore, it is important to understand that both ovine and caprine are not to be considered small bovine. The objective of this paper is to serve as a reminder regarding the necessity of adequate nutrition in small ruminant species. It is also intended to provide a brief overview regarding the importance of several minerals and vitamins and their associated toxicities and deficiencies.

Key words: caprine, minerals, ovine, vitamins

Introduction

Nutrition is one of the foundation blocks of small ruminant health.[6] Focus on infectious agents often overshadows nutrition when complications arise in livestock. Adequate supplementation of minerals in small ruminant species is paramount for proper physiological processes. Inadequate or excessive supplementation can lead to detrimental effects reflected by impaired immune system function, reproduction, and growth.[28] Understanding the importance of the function of minerals and vitamins, deficient and toxic associated complications, interactions with other nutrients, and diagnostic sampling is critical in small ruminant practice.

Copper

Copper (Cu) is an essential trace mineral that is critical for the proper functioning of numerous enzymes and biochemical processes in small ruminants. Several enzymes in which Cu is associated with include hephaestin, ceruloplasmin, tyrosinase, cytochrome C oxidase, and super oxide dismutases. Hephaestin is responsible for the oxidation of ferrous iron (Fe2+) within enterocytes to ferric iron (Fe3+) allowing for mobilization and transportation by ceruloplasmin. Melanin, responsible for providing coat pigmentation, is produced through the hydroxylation of tyrosine by the enzyme tyrosinase. Cytochrome c oxidase is vital for cellular respiration as it is part of the electron transport chain. Superoxide dismutases are integral in both immune system function and wool quality.[28] Common sources of Cu include forage and mineral supplements. Water from footbaths containing Cu sulfate are a potential source associated with excessive Cu accumulation and intoxication.[22]

Although essential, excessive Cu supplementation can be detrimental in sheep. Sheep are more sensitive to Cu in comparison to goats and cattle. Excessive Cu supplementation is commonly unintended. One of the most common sources of excess is the use of feed or feedstuffs intended for other species. Feeds intended for other species tend to have higher Cu concentrations in order to meet that species' nutrient requirements.[4,23] Sheep consuming loose or block mineral supplements intended for cattle are potentially at risk of accumulating toxic amounts of Cu. Many feed products that are labeled for cattle or other species often have signage specifically stating that the product should not be used for sheep. These warnings should be heeded. Copper poisoning in sheep has been associated with sheep grazing pasture where porcine manure had been applied.[17] Clinical signs of a Cu associated hemolytic crisis include anorexia, weakness, icterus and hemoglobinuria (red to brown urine). Acute death within 24-48 hours may also be observed. On post mortem evaluation, tissues may appear icteric and the liver friable. Sheep that succumb to Cu intoxication exhibit dark "gun-metal" colored kidneys.[11,23,33]

Inadequate dietary supplementation or decreased bioavailability are the main causes of deficiency.[20] The presence of antagonists in the diet and environment play a large role in the availability of Cu to the animal. Iron (Fe), sulfur (S), and molybdenum (Mo) all are antagonistic to Cu.[24,28] Clinical signs of deficiency are associated with loss of enzyme function and include anemia, poor wool quality, depigmentation and immune system dysfunction.[27,28] Adequate supplementation of Cu to does and ewes is believed to be critical for proper neurologic development. Lack of adequate supplementation has been shown to result in sway back.[3] Three forms have been reported; neonatal paralysis, delayed, and atypical. Sheep are the most sensitive while in goat kids, the delayed form predominates.[27,28]

Copper is essential for proper physiological processes, and should not be completely withheld in small ruminants solely based on their sensitivity. Copper should be supplemented at adequate levels.[6] Prevention of excess supplementation is critical in avoiding Cu intoxication in sheep. Feed and feedstuffs formulated and labeled for other species should not be provided to sheep. Feed intended for sheep should not have a Cu to Mo ratio >10:1. Ideally the ratio should be 6:1. Feeds possessing ratios of >20:1 provide significant risk of excessive Cu accumulation.[24] Reduction of Cu in the feed and administration of sodium molybdate can help in reducing further Cu accumulation in affected animals.[28]

Selenium

Selenium (Se) is widely known for its role in immune system function and antioxidant properties. It is an essential component of glutathione, glutathione peroxidases and selenoprotiens.[26] Aside from preventing and limiting free radical formation, Se also plays an integral role in thyroid hormone synthesis (T4 to T3) in various tissues through several iodothyronine 5'-deiodinase enzymes.[28] Dietary sources of Se include grains, legumes, and other forages.[23] The concentration of Se within vegetation is dependent on the region and composition of the soil as the Se content of soil varies greatly by region.[35]

Selenium toxicosis commonly develops through ration misformulations, drenches, consumption of high Se forage, or through incorrect/excessive parenteral supplementation.[5,18] Regional soil content and vegetation may result in disproportionate concentrations of Se. Numerous plant species accumulate Se. Obligate accumulators include species of the Astragalus, Oonopsis, Stanleya and Xylorrhiza genera. It is not uncommon for plants in these genera to accumulate upward of 1,000 ppm of Se.[23,32] Lambs that ingest between 2-4 mg/kg sodium selenite

or 4-8 mg/kg selenomethionine developed respiratory distress. Lambs exhibited myocardial necrosis and pulmonary hemorrhage and edema.[32] White muscle disease in ruminants is a common syndrome associated with Se deficiency. The deficiency associated syndrome is usually predominant in young lambs or kids but can also be observed in older juvenile animals. Generalized ill thrift with weakness is the typical presentation of this syndrome. Gross examination of cardiac and skeletal musculature reveals pallor.[8,9]

Zinc

Zinc is a known component of over 3,000 enzymes. As with other trace minerals, zinc plays an integral part in immune system function through maintenance of the epidermal barrier and super oxide dismutase enzymes.[28] Gene expression is largely dependent on zinc.[10] Retinol binding protein, responsible for transport of vitamin A from the liver to other tissues is also dependent on zinc. Supplementation of zinc is predominately through dietary means.[28]

Toxicity of zinc in small ruminants is not often reported but has been reported in suckling lambs provided excess.[7] Concentrations of Zn high enough to cause toxic effects would likely originate from ration misformulations.[1] Goats or sheep located near facilities that galvanize steel may be exposed to zinc dust or exhaust that covers the forage.[30] Deficiency is due in part to inadequate supplementation or decreased bioavailability. Calcium and phosphorous as well as Cu impair zinc absorption within the GI tract.[24] Signs of deficiency include reduced feed intake and immune system function. Reproduction may be negatively impacted by Zn deficiency.[28] Severely deficient individuals may develop hyperkeratotic parakeratosis, a crusty scaling of the skin. Animals may also present with anorexia, abnormal hoof growth and foot soreness.[21]

Manganese

Manganese plays a significant role in immune system function and in structural development. As with Cu and Zn, Mn is a component of super oxide dismutase enzymes. It is also integral in glycosyl transferase enzymes that allow for proper cartilage and bone development.[28] Forage and grasses tend to possess greater concentrations of Mn than silage.[14] Manganese is further supplemented by either dietary or parenteral means. Manganese is tightly regulated within the body, and toxicities are rare.[28] Inadequate supplementation of Mn in the dam during gestation can lead to skeletal anomalies in offspring. Extremities exhibit enlarged joints with shortened long bones. This has been observed in calves.[13,31] Microscopic examination reveals chondrodysplasia within the metaphyseal region of the bone. Deficiency can also be brought on by excessive Fe consumption.[28]

Vitamin E

Alpha tocopherol is the active form of vitamin E within the body. Although it holds many functions, one of the primary roles of vitamin E is acting as an antioxidant to prevent tissue damage from free radicals while also supplementing the action of glutathione peroxidase enzymes.[6] Vitamin E is provided through both dietary and parenteral means.[24] Adequate supplementation of vitamin E along with Se is necessary to prevent nutritional myopathy or white muscle disease.[6] Sodium selenite has the potential to reduce liver vitamin E.[32]

Antagonists

Sulfur, molybdenum and iron are three of the most important antagonistic minerals in ruminant nutrition. The mechanism of antagonism includes binding to the mineral making it unavailable for absorption, competition for transporters or storage space, or through down regulation of shared transporters.[12,28] Primary sources of S associated antagonism include high dietary sulfur or elevated sufate within water.[34] Vegetation growing in Mo rich soil can possess high levels of Mo. All three minerals impair absorption of Cu. Both Mo and S alone are able to inhibit Cu absorption by 0.5% and 3.1% respectively. However, when combined in the rumen, thiomolybdate formation can result in an approximately X% inhibition of Cu absorption.[29] Sulfur and Se share similar transporters, so excessive S can result in impaired Se absorption. Both Fe, Cu, and Mn share the divalent metal transporter 1 (DMT1) within the GI tract. Excessive Fe from feedstuffs not only compete for the transport use but also decreased DMT1 activity following Fe saturation within enterocytes.[12] Zinc and Cu both compete with each other for metallothionine and ultimately storage within the liver.[28]

Diagnostics

When evaluating mineral and vitamin status in small ruminants, proper sample selection and submission is imperative for optimal analysis and interpretation. Fresh liver should be submitted for trace mineral analysis as it is the main storage organ of trace minerals. Antemortem liver of approximately 0.005-0.006 g DM are sufficient for diagnostic interpretation.[2,25] Although simple to collect, serum is not the optimal sample for Cu analysis. Serum Cu concentrations do not necessarily correlate with hepatic concentrations. Submission of suspect feed and water samples is encouraged to evaluate for excess or deficiencies. Due to accumulation of minerals over time, the submitted sample may not be representative of what was consumed by the individual that lead to the accumulation.[23] Since milk and colostrum serve as major sources of vitamin E, evaluation of vitamin E in stillborn lambs or kids is of little diagnostic help.[24] Both liver and serum can be used to evaluate for vitamin E concentrations.[15,16] Collection of serum for vitamin E should be performed to in a manner similar to plasma to best hemolysis that may degrade the sample. Serum should be placed in tubes occluded from light, centrifuged, and chilled or frozen.[19]

Conclusion

Although ruminants, sheep and goats should not be considered small bovine. There is variation between species in regards to necessities and tolerances to different nutrients. The nutrients presented are all essential for life and must be provided adequately. Presentation of clinical signs and lesions associated with deficiencies and toxicities also vary between species. The manner in which small ruminants are supplemented not only depends on their age and use but also by geographic region. Methods of supplementation must also be taken into consideration.

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