AABP and AASRP—Joint Sessions

Moderator: Paul Jones

Goat and Llama Trace Mineral Nutrition

D. G. Pugh, *DVM, MS, Dipl ACT & ACVN*[1]; **Bryan M. Waldridge,** *DVM, MS, Dipl ACVIM and ABVP*[2]
[1]*Fort Dodge Animal Health, 1154 Barnard Ave., Waverly, AL 36879*
[2]*Department of Clinical Sciences, Auburn University, AL 36849*

Introduction

The authors have observed nutritional associated diseases of goats and llamas only in cases of copper, iodine, iron, selenium and zinc deficiency.

Selenium

Selenium (Se) is required for small ruminants and llamas at about 0.1 to 0.3 ppm (mg/kg) on a dry-matter basis. Diets with Se concentrations ≥ 0.2 ppm appear to provide adequate Se in llamas, based on glutathione peroxidase activity and whole blood Se concentrations within normal ranges for other animals, and lack of clinical signs of Se-deficiency disease.

Clinical signs of Se deficiency include lameness, decreased activity, lack of endurance, dyspnea, colic, increased susceptibility to heat stress, arched back, infertility, metritis, weak or premature kids or crias, cardiac insufficiency and acute death. A diagnosis of Se deficiency can be based on both clinical signs and confirmation of deficient blood or liver Se concentration. Whole blood Se concentration is a good indicator of long term Se intake, and is less likely to be affected by dietary fluctuations than serum Se. If a dietary change has occurred within three weeks of sampling, serum Se concentration will usually be altered.

Treatment of deficiency involves Se supplementation of the ration, offering selenized trace-mineral salt sources free choice, or parenteral injection of Se-containing products. Free-choice trace mineral salt sources are usually the least expensive method to increase dietary Se intake, and the least likely way to induce Se toxicity. Individual animal consumption is variable due to sporadic intake, palatability, pasture conditions and time of the year. Salt mixes containing Se should be the only salt sources offered. Continuous feeding of a complete ration supplemented with a selenized premix is also a very effective way to prevent deficiency. A common practice among goat producers is to offer trace-mineral salt blocks. This practice may result in inadequate intake and/or broken incisor teeth of animals attempting to acquire salt. Injectable Se preparations should be administered only to increase low blood Se concentration or to treat Se-deficiency disease. Recommended dose for llamas and goats is 0.03 to 0.04 mg/lb (0.07 to 0.1 mg Se/kg) subcutaneously. Fatal Se toxicosis has been induced in a llama by excessive amounts of injectable sodium selenite. Selenium is rapidly absorbed following injection in llamas. Auburn University workers reported that whole blood Se concentration peaked within five hours following administration when llamas were injected with 0.04 mg/lb (0.1 mg Se/kg) subcutaneously. Selenium injections are unlikely to have a prolonged effect on either glutathione peroxidase activity or whole blood Se concentration in llamas consuming Se-adequate rations.

Copper

Copper (Cu) is required for bone, cartilage and tendon production, hepatic detoxification, melanin production, energy metabolism and iron and hemoglobin metabolism. Copper is absorbed predominately from the small intestine and stored in the liver. Copper absorption and availability is decreased by rising dietary intake of zinc, iron, cadmium, selenium, molybdenum (Mo), calcium and sulfur (S). Copper, Mo, and S form insoluble complexes in the anaerobic first and second compartments of the forestomachs of llamas and the rumens of goats, resulting in copper deficiency or depressed copper availability.

If the Cu content of pasture is less than 5 ppm on a drymatter basis, and/or where Mo exceeds 1 ppm and/or sulfur exceeds 2000 ppm, Cu deficiency may occur. Johnson reported a Cu:Mo ratio of < 3:1 for long-term

feeding without evidence of Cu deficiency in llamas. Roughages grown on well fertilized pastures are more likely to be deficient in Cu than those on 'unimproved' land. Signs of copper deficiency include microcytic anemia, light or faded fiber color or poor quality fiber and lameness. Goats fed 'sheep minerals' as the only source of trace-mineral supplementation, and/or pastures deficient in Cu, may experience copper deficiency disease.

Some dwarf breeds of goats may require more dietary Cu than other breeds. Occasionally young kids may present with enzootic ataxia if their dams are maintained on Cu-deficient diets. Copper accumulates in the liver in proportion to the ratio of Cu to Mo in the diet, and duration of dietary Cu exposure. Copper 'dumping' from the saturated liver secondary to stress or disease results in anemia, haemolysis, icterus, kidney failure, liver necrosis and usually death. Copper toxicity is usually preceded by episodes of stress or sudden weather changes (e.g. heat stress), particularly in the late-pregnant animal. Liver disease (e.g. toxic plant consumption, liver flukes, etc.) may decrease the amount of Cu the animal can store, and can be associated with Cu toxicosis.

Copper toxicity has been described in llamas when the Cu to Mo ratio was greater than 15:1. New Zealand workers have reported that alpacas may be more resistant to Cu deficiency and toxicity than sheep. In areas of South America where sheep are co-grazed with llamas, only the sheep appear to be affected with Cu-associated diseases. Liver tissue is the best indicator for both toxic and deficient copper conditions. Auburn University workers described a method and demonstrated its safety for liver biopsy in llamas. The procedure also is easily and safely performed in goats. The liver reflects a cumulative history of dietary copper intake. Female llamas appear to have significantly lower liver Cu than do males. Dietary copper concentrations have been suggested to be 5-15 to 15-25 ppm in the llama and 4-15 ppm in goats. Trace-mineral salt mixtures designed for most livestock will usually be sufficient if the entire diet is evaluated, and concentrations of Mo, S and other minerals are considered.

Zinc

Zn deficiency may result in dermatitis, decreased productivity, hoof maladies (or increased susceptibility to hoof disease), decreased testicular development and alterations in Vitamin A and E metabolism. Signs of Zn deficiency include decreased feed intake, impaired immunity, poor growth rate, skin disorders, depressed reproduction, and poor fiber quality. Zinc-responsive dermatosis is most commonly seen in 1-2 year old llamas or young, growing kids, and males appear to be more susceptible than females. Signs in the llama, and occasionally the goat, include appearance of papules or dry plaques of alopecia over the ventral abdomen, inguinal area, inner thighs and bridge of the nose. It appears that some llamas (males) and goats (pigmy) may have a genetic predisposition to the condition. Biopsies of affected regions will show parakeratosis.

Diagnosis can be based on clinical signs, biopsy results and plasma Zn concentrations. If zinc deficiency is suspected, serum or plasma should be properly collected into tubes specifically designed for trace-mineral analysis. Treatment includes zinc sulfate or Zn methionine daily in the diet for severe cases, or a salt mixture fed free choice with added Zn, preferably in the sulfate form. Skin lesions will usually improve after dietary modification in 2-3 months. Zn deficiency may be due to excesses in Ca intake (e.g., legume hays), or the feeding of high-phosphate-containing diets (cereal grains). A complete dietary evaluation should be performed and subsequent dietary modification instituted.

References are available upon request.