NUTRITIONAL UNBALANCES IN CAMELIDS

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Introduction Literature citations of specific nutrient deficiency or toxicity diseases are limited, but suggest camelids have similar clinical presentation and pathologic mechanisms compared to other species. Articles addressing some aspect of nutrition represented less than 15 percent of total citations on South American camelids covering the years 1943 to 2006 in a web-based bibliography (Anonymous 2010). Most of the nutritional citations addressed digestive function, feed preference, comparative digestive efficiency and nutritional disease with only a couple specifically addressing nutrient requirements. A substantial amount of nutritional research data can be found in South America; however, much of this information has not been readily available through literature search engines in countries outside of South America. Understanding nutrient requirements for various physiologic states is fundamental to describing and diagnosing nutritional diseases. Research describing camelid nutrient requirements is sparse and typically only addresses the physiologic state of maintenance. Using these maintenance requirements, a series of factorial nutrient requirement models were developed for other physiologic states based on some extrapolation of data from sheep and goats (Van Saun 2006). These models were adopted in the recent National Research Council (NRC) publication on nutrient requirements for camelids (NRC 2007). These models can be applied to dietary formulations, but can provide a reference point in determining potential for nutritional unbalances. The objective of this presentation is to characterize various nutritional diseases of camelids and provide perspectives for future research in addressing these concerns.

Energy and protein: Camelid maintenance energy and protein requirements have been reported and both values are less than those determined for other ruminants. In contrast, camelids require more protein per unit of energy than other ruminants, possibly suggesting a greater role of protein in energy metabolism. A metabolic peculiarity of camelids is their higher blood glucose concentration compared to other ruminants, possibly a result of their reduced insulin secretion and lower tissue insulin sensitivity. These metabolic idiosyncrasies may explain the observed higher hepatic fatty infiltration susceptibility of camelids secondary to most nutritional or environmental stressors.

Camelids, like all other species, are susceptible to either a deficient or excessive intake of energy relative to requirement resulting in variable stages of protein-energy malnutrition (PEM) or obesity, respectively. In extensive management systems typical of South America, llamas and alpacas experience wide body weight deviations associated with seasonal rains and forage availability. During the dry season, animals will lose significant body weight compromising health and reproductive performance. In North America, PEM is associated with extreme cold or hot environmental conditions and feeding of poor quality forages. Obesity is lesser of an issue in South America, but a prevalent problem within the North American camelid population. Over consumption of energy results from feeding high quality forages, providing additional grain-based supplements or some combination. At the crux of recognized energy and protein dietary unbalances is dry matter intake (DMI) capacity.

Intake capacity: Comparisons of DMI capacity between camelids and other ruminants have been extensively studied, though there is not complete agreement among studies. When intake for llamas and alpacas were adjusted for metabolic body weight (MBW), there were no differences between llamas and alpacas, but intake was lower compared to sheep with the lowest intake observed with unimproved forages. Observed slower particle passage rate in camelids will constraint total feed intake as slowly digestible feed particles will be retained longer and maintain physical displacement or gastrointestinal fill. Differences in DMI results across studies may reflect wide differences in the forage quality. Maintenance DMI in the NRC (2007) system suggests 38 g/kg MBW while South American (Lopez et al 1992) data suggest higher intakes for alpacas (39-54 g/kg MBW) and llamas (40-53 g/kg MBW). With these differences in expected intake, described dietary requirements will diverge between the two systems as the absolute energy and protein requirement estimates are based on the same models.

Minerals: A number of reports primarily based on clinical observations have suggested camelids are susceptible to classical deficiency disease syndromes of calcium and magnesium. Hypophosphatemia is most often associated with underlying vitamin D deficiency. For the trace minerals, published reports have implicated copper, iodine, iron, selenium and zinc in various deficiency diseases. However, many of these publications lacked definitive diagnostic measures to adequately determine if the described mineral was truly deficient and the cause of the presenting symptoms. From the literature clearly copper toxicity and zinc deficiency are the most prevalent trace mineral disease concerns of camelids. To date there are no reported feeding trials to estimate camelid mineral requirements, though some extrapolated models have been developed (Van Saun 2006).

Published studies as well as anecdotal clinical cases suggest llamas and alpacas to be sensitive to copper toxicity, though not as keenly sensitive as sheep. As with sheep, excessive copper intake results in chronic hepatic accumulation until a saturation point is reached and copper ions are released, thus inducing hepatocellular damage. Based on these reports, camelids are prone to copper toxicity on diets with greater than 25 mg/kg copper and 16:1 copper-to-molybdenum ratio. Published reports and many clinical cases suggest some relationship between hyperkeratotic lesions affecting hairless skin regions and zinc status. Some authors suggest the skin lesions are not a consequence of zinc deficiency, but responsive to supraphysiologic zinc supplementation. One study showed low blood zinc concentration and responsiveness to zinc supplementation in camelids with these skin lesions. Calculated dietary zinc content was between 22 and 30 mg/kg, below what would be recommended for other ruminants.

Vitamins: Similar to other ruminant species, documentation for dietary supplementation of B-vitamins or associated deficiency diseases is negligible in camelids. The only noted exception is thiamin deficiency, which has been reported in a number of case study publications. In all reported cases affected animals presented with classical neurologic clinical signs associated with polyencephalomalacia. Diagnosis was presumptively made by presenting signs and rapid response to thiamin therapy or definitively
by presence of microscopic cerebral necrosis lesions on necropsy. Inciting dietary causes cases were all associated with either increased access to grain or abrupt dietary changes in which fiber intake was reduced.

Of the fat soluble vitamins, no reports describing vitamin A deficiency or toxicity have been reported. Vitamin A is considered a limiting nutrient in beef cattle production as a result of extensive ruminal degradation of dietary forms and low dietary intake when consuming stored or stockpiled forages. One published report and anecdotal clinical observations suggest camelids are susceptible to vitamin E deficiency myopathy. Vitamin D deficiency and more recently toxicity diseases have been the predominately reported vitamin-associated syndromes in camelids. In the early 1990’s Fowler reported on a hypophosphatemic rickets syndrome in growing 3 to 6 month old crias in North America, which was subsequently described in Australia. Van Saun and colleagues determined vitamin D deficiency to be the underlying cause and related vitamin D status to UV light exposure and intensity, geographic latitude, fleece color and age. Dark colored crias born in fall and winter months (September to February) were most susceptible to the disease. To the author’s knowledge this condition has not been reported in South America, which may reflect greater UV exposure at higher elevations and proximity to the equator for South American camelids. Parenteral vitamin D therapy (1000-1500 IU/kg body weight) can effectively correct the condition, but excessive vitamin D administration either orally or parenterally has resulted in a number of vitamin D toxicosis situations resulting in classical soft tissue mineralization and renal failure. Dietary vitamin D supplementation has been studied in an effort to establish feeding recommendations to prevent the disease. Camelids required greater dietary vitamin D supplementation (30-40 IU/kg body weight) to maintain blood concentrations similar to other species, which may reflect an adaptation to down regulate denovo synthesis from UV exposure potential in their natural environment.

Future directions: A better understanding of nutritional unbalances is predicated on valid documentation of camelid nutritional requirements. A priority for future research should be a focus on validating or improving upon the proposed factorial nutrient requirement models. Secondly, models to predict dry matter intake based not only on body weight measures but feed composition, namely neutral detergent fiber, should be developed. Finally, feeding trials are needed to estimate mineral and vitamin requirements.

Key words: nutritional unbalances, camelids, llamas, alpacas.

References:


