



Where'd the Sun Go? Vitamin D and Bone Development

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Most people are familiar with vitamin D being known as the “sunshine” vitamin. This is a result of vitamin D being formed in the skin when exposed to certain wavelengths of ultraviolet (UV) light. Most people now also recognize the role that vitamin D plays in bone development. Observations linking sunshine to bone structure go back to Greek history. During the industrial revolution in the mid 1800’s, it was recognized that children living in the city had more bone growth problems than children raised in rural locations. It was not until the early 1920’s that the compound vitamin D was discovered and characterized. Soon thereafter it was determined that UV exposure of dairy products could increase vitamin D content and reduce risk of rickets in children. This approach has been used to the present to help fortify vitamin D intake in the human population. Unfortunately, with current consumption trends where milk intake is decreasing and carbonated beverage intake increasing, we are seeing an increasing prevalence of rickets in children.

Rickets is an abnormal bone growth disease of children and young growing animals of all domestic species and related to nutritional deficiencies of calcium, phosphorus, vitamin D, or some combination. A rickets syndrome in juvenile llamas and alpacas characterized by a shifting leg lameness and enlargement of the joints, most noticeably the carpus (knee), has been described in the early 1990’s by Dr. Murray Fowler (1,2). Affected crias have variably shown a slowed growth rate, reluctance to move, and humped-back

stance. Radiographic evidence of boney growth plate changes (Figure 1) and low serum phosphorus concentrations were consistent with a diagnosis of rickets. Affected crias typically were still nursing and between 3 and 6 months of age. The disease occurrence was most prevalent during the winter months (December to March). The seasonal occurrence of this syndrome, coupled with the age and nursing status, helped focus our attention on vitamin D as an underlying cause, rather than the obvious issue of low phosphorus. The Camelid Research Group at Oregon State University had investigated the role of vitamin D in hypophosphatemic rickets over a period from 1993 to 1999, completing a series of five studies addressing aspects of disease etiology, treatment, and prevention.

Finding the Cause. . .

In a survey of affected and normal crias on various individual farms located in the northwest, we were able to identify significantly lower blood phosphorus and vitamin D concentrations in affected compared to normal crias (3). One might ask which comes first, phosphorus deficiency or vitamin D deficiency? There is no precedent for phosphorus deficiency on a milk-based diet. In comparing blood phosphorus and vitamin D concentrations among normal and affected animals, we found phosphorus concentration to be highly associated with vitamin D in affected animals, but not in normal animals. Additionally, we showed that vitamin D supplementation alone,

without phosphorus, was sufficient in correcting both low blood phosphorus and vitamin D concentrations. The role of vitamin D in calcium regulation is well documented; however, it also has been shown to control intestinal absorption of phosphorus.

In a second study, we attempted to answer the question regarding seasonal incidence of this syndrome. Blood calcium, phosphorus and vitamin D concentrations were measured monthly over a period of one year in a number of adult and growing llamas and alpacas. Again we found vitamin D concentrations in affected crias to be highly associated with blood phosphorus concentrations (4). Additionally, month of birth was also associated with blood phosphorus concentrations in young growing crias. This determination is consistent with owner's observations that fall-born crias are more susceptible to this problem compared to spring-born crias. Our results showed crias born between September and February had the lowest blood vitamin D concentrations compared to crias born between March and August (Figure 2). These data provide some rationale for the seasonal prevalence of this disease, but why are vitamin D concentrations so low in fall-born crias?

The Root of the Problem. . .

In this same study, we also documented wide seasonal swings in blood vitamin D and phosphorus concentrations in adult and yearling llamas and alpacas. We also showed that animals with dark colored fleece (black) had significantly lower vitamin D concentrations compared to white or cream colored animals. Additionally, shearing during the summer resulted in increased vitamin D concentrations within a week's time. Clearly, exposure to the summer sun's rays is crucial to maintaining

blood vitamin D concentrations. During the winter months, less UV radiation reaches the northern latitudes as a result of changes in the earth's axis rotation. Without adequate UV exposure during the winter months, blood vitamin D concentration drops dramatically. This affects all crias equally, why the problem in fall-born crias?

To understand this aspect, we need to address issues of vitamin D reserves. Blood is the storage organ for vitamin D, unlike liver which is the storage organ for vitamins A and E. Vitamin D does not appreciably cross the placenta in the pregnant female, thereby the newborn cria is born with very low vitamin D status. The pregnant female will concentrate vitamin D in the colostrum (first milk) to a level consistent with her own vitamin D status. Vitamin D status of the newborn cria will then be determined by the vitamin D content and amount of colostrum consumed. This further underscores the importance of ensuring good colostrum consumption by the newborn cria. Still, how does this explain the problem in fall crias? Fall-born crias may not receive sufficient vitamin D in colostrum due to their dam no longer being exposed to appropriate UV light. This also results in an inability for the cria to make vitamin D. In contrast, spring-born crias receive colostrum from dams that are being exposed to UV radiation and they have the summer months available to make their own vitamin D and store reserves. Ultimately this combination of events results in dramatic differences in cria vitamin D status during the critical growth period of three to six months of age (see figure 2).

A Solution. . .

When this health problem was being confronted by owners, a simple solution to the problem was not to have fall-born crias. This drastic action is not really necessary. In

subsequent studies (yet to be published) we have shown vitamin D injections capable of successfully treating affected crias and potentially preventing problems. Our preferred method would be to supplement adequate vitamin D in the diet to prevent the disease entirely. Similar to reported results from an Australian study (5), injections of vitamin D can increase and maintain adequate vitamin D concentrations in adults or crias. Using the commercial vitamins A and D preparations available in the U.S., we found supplementation of vitamin D between 1,500 and 2,000 IU/kg (700 and 900 IU/lb) body weight resulted in adequate blood vitamin D concentrations for three months. Most vitamin D preparations contain 75,000 IU/ml, which translates into approximately 1 ml per 100 lbs body weight. Label directions suggest intramuscular injection of vitamin preparations, but they can be very irritating. During our studies all injections were given by this route; however, we have found a similar response to subcutaneous injections and less problems with injection site reactions. It is highly recommended that you work with your veterinarian in determining appropriate level of therapy as products may differ in concentration. This dosage can be used in adults as well as growing crias.

One can supplement the late pregnant female to improve vitamin D concentrations in her colostrum. This approach can be problematic in handling a late pregnant animal and possibly inducing enough stress to initiate premature delivery. Based on sheep work, vitamin D injections would need to be given during the last 14 days of pregnancy to effectively increase colostrum concentrations. This response has not been confirmed in camelids, but injections more than one month prior to birthing may not improve colostrum vitamin D status. A preferred approach would be to

supplement the cria at or soon after birth and repeat in three months if necessary.

A better approach still would be to provide sufficient vitamin D in the diet to maintain adequate vitamin D status throughout the winter months. Our preliminary research has shown some interesting findings with dietary vitamin D supplementation in camelids. Llamas and alpacas seem to poorly absorb dietary vitamin D and have a higher requirement compared to other ruminant species. Based on some feeding trials, we have made a recommendation of feeding supplemental vitamin D at a rate of 30 to 40 IU/kg (13 to 18 IU/lb) body weight. This translates into 2,250 IU vitamin D/day for an animal weighing 150 lbs. If you are feeding a supplement at 0.3 or 1 lb/100 lbs body weight, then the vitamin D concentration in the supplement should be 5,000 or 1,500 IU/lb, respectively.

We have determined that vitamin D is an essential nutrient in llama and alpaca diets for ensuring good growth and bone development. Farms geographically situated in the northern US and all parts of Canada need to evaluate their animal's vitamin D status as winter rapidly approaches. Vitamin D injections can be used to stave off any problems or treat any clinically affected animals. Continuous oral supplementation would be the preferred method, but few products are available with adequate amounts of vitamin D. Work with your veterinarian in monitoring your risk and enacting a preventive program.

References:

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Figure 1. Radiograph of a cria with hypophosphatemic rickets. Arrows highlight abnormally widened and irregular growth plates of long bones in rear limb.

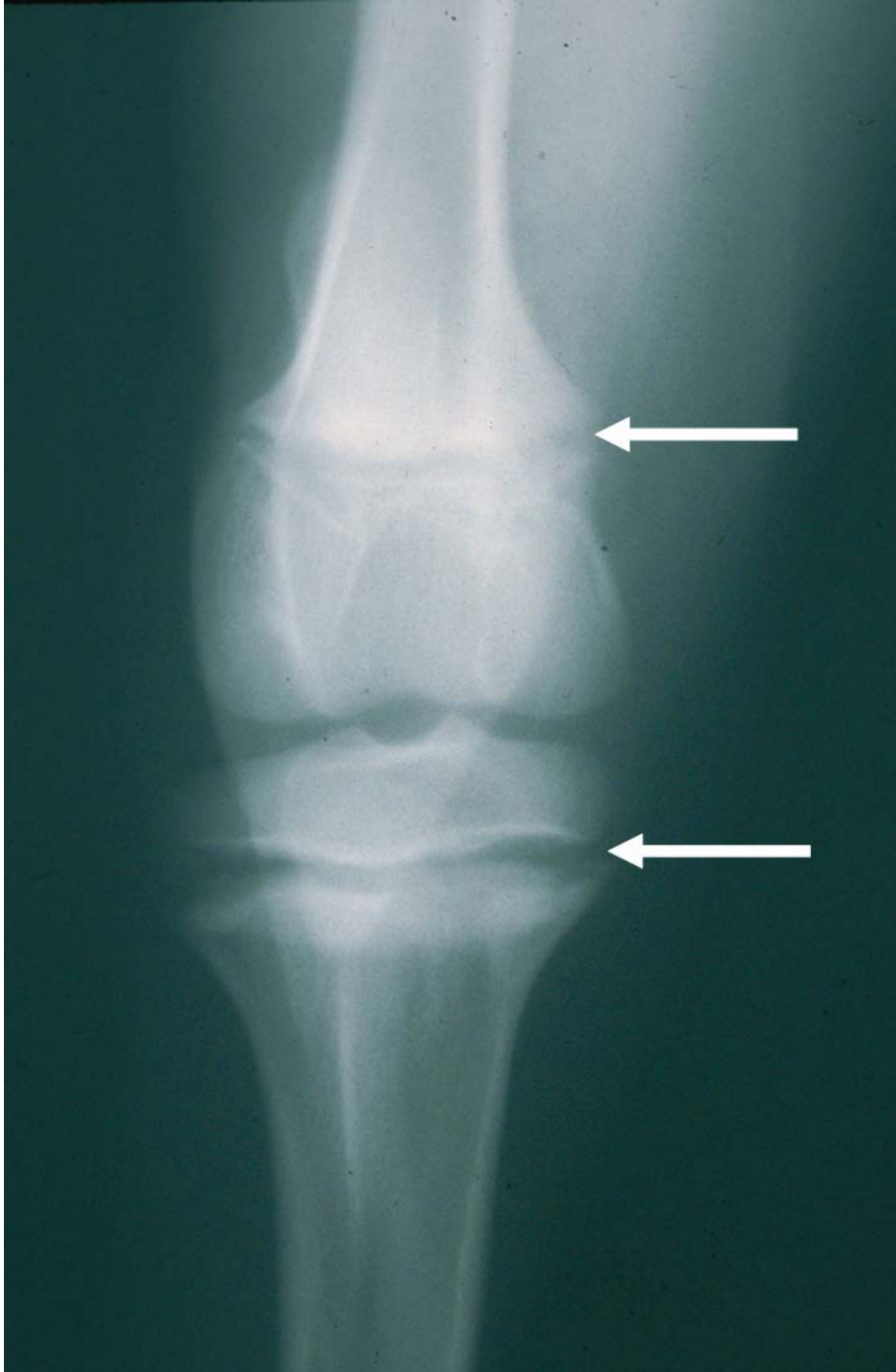


Figure 2. Comparison of serum vitamin D concentrations in crias over the first year of life categorized by month of birth (From Smith et al., 2001).

