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MINERAL Writes

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Todd M. Owens, M.S., P.A.S.
Director of Nutrition and Technical Services
ILC Resources
3301 106th Circle
Urbandale, Iowa 50322-3740
(515) 243-8106
Fax (515) 244-3200
1-800-247-2133

www.ilcresources.com

toddo@ilcresources.com

Vitamin D in Ruminants

There are two sources of vitamin D. The fat-soluble molecule, vitamin D, is found in animal cholesterol or in fungi ergosterol. Vitamin D_2 is in fungi and can be consumed by animals from forages. This is a minor source of the nutrient. Vitamin D_3 is made in skin by the animals with exposure to the UV-B rays from the sun. In cattle, the majority of the vitamin D metabolites come from vitamin D_3 (Nelson, 2014).

The NRC requirement of vitamin D in beef cattle is approximately 300 IU/kg of dry matter intake. Outside housing makes the need for additional supplementation rare, due to sun exposure. Current protocol in the industry involves supplementing diets with vitamin D during the last week of finishing to improve beef tenderness (≥1 million IU/d). Recent research has shown conflicting results on improving tenderness. In combination with the effects of decreased short-term growth rates, feed intakes or feed efficiency from the increased supplementation, it is unlikely to see widespread adoption of this protocol in today's beef production.

Vitamin D requirements have been determined almost solely on the amount needed to maintain calcium homeostasis. New research is proving the additional benefits of vitamin D, especially significant impact on immune cell function, but much of that research has been conducted on nonruminants. Specific research on the effect of vitamin D supplementation on bovine immune systems is yet to be conducted, but linkages are being shown (Nelson et al., 2010). Other studies also found that infusing *Streptoccus uberis* infected mammary gland quarters with 25-OH vitamin D significantly decreased the severity of mastitis (Lippolis et al., 2011). Decreased plasma concentrations of 25-OH vitamin D are risk factors in humans and similarly in dairy cows (Christakos and DeLuca, 2011). In addition, plasma levels in dairy cattle receiving supplemental vitamin D at recommended NRC levels (NRC, 2001) with limited sun exposure were lower than in cows with full exposure to the sun (Hymøller et al., 2009).

Based on these recent findings and on older studies in the 70s and 80s that show feeding vitamin D at approximately two times current recommendations increases milk yields, vitamin D requirements need to be re-evaluated (NRC, 2001). The effects of vitamin D on milk yields, immune function and mastitis need to be considered in addition to the effects on calcium homeostasis.

Vitamin D does not specifically contribute to bone, mineral and immune functions. It is broken down to a metabolite and then at the cellular level the metabolites bind with proteins at specific DNA regions which regulate gene

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transcription. Cell type and condition determine what genes are controlled by the metabolites. Metabolites stimulate the proteins that bind and transport calcium in the kidneys and intestines, maintaining the flow of calcium and phosphate in circulation. In the immune system, the metabolites spur gene expression for proteins that are toxic to pathogens in the body (Nelson 2014). Concentrations of metabolites in the blood and in tissues are regulated by the body to control activating vitamin D receptors in cells. The regulation of the metabolites is determined by the concentrations of calcium and phosphate in the blood (Figure 1; Nelson, 2014).

Blood calcium levels fluctuate depending on production phases in dairy cattle. When calcium levels drop at the start of lactation, parathyroid hormone (PTH) is released which stimulates 1α-hydroxylase (1α–Ohase) activity in the kidneys. As vitamin D is metabolized to 1,25-dihydroxyvitamin D (1,25D) in the kidneys, the 1,25D concentrations increase in response to PTH and calcium levels are restored. Milk fever (low calcium levels) is not usually considered to be a result of insufficient vitamin D, but instead as a result of insufficient activation of 1α–Ohase in response to the release of the PTH. During transition, dairy nutrition can influence the response of 1α–Ohase to PTH. Producers who feed a diet with a negative dietary cation-anion difference (DCAD) can improve the response of 1α–Ohase to PTH, but excess concentrations of calcium and phosphorous prepartum can inhibit 1α–Ohase activity (Nelson, 2014).

The presence of pathogens (bacterial and viral) stimulates 1α–Ohase in immune cells. The effectiveness of the immune cells to defend against the pathogens depends on 1α–Ohase activity and the availability of the 25-hydroxyvitamin D (25D) metabolite. The availability of 25D is dependent on vitamin D intake. Reduced intake of vitamin D can impair the vitamin D-dependent functions of the immune system. The expected symptoms of vitamin D deficiency are rickets, slow growth and stiffness. When the available amount of vitamin D is insufficient, but not deficient, dairy cattle may be susceptible to infection and fall short of peak performance (Nelson, 2014).

What this means for the industry

While the majority of vitamin D comes from sun exposure, it is not a reliable source unless cattle have unlimited access to summertime sun. In modern confinement housing units, dairy herds often have limited or no exposure to sunlight. Supplementation is essential for this reason. A general rule is that cattle should receive at least 15-20 international units (IU) of vitamin D_3 daily per pound of bodyweight (Nelson, 2014). For cows this translates to a daily rate of 20,000-50,000 IU; 10,000-20,000 IU for heifers and 5,000-10,000 for calves of vitamin D_3 (Nelson, 2014).

Vitamin D is integral to controlling calcium and phosphate levels and for the natural defenses of the immune cells. More research is needed on vitamin D in order to better understand the physiology and to improve the optimization of the nutrient for increased immunity and production.

Information for this article taken from:

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Vitamin D in Ruminants

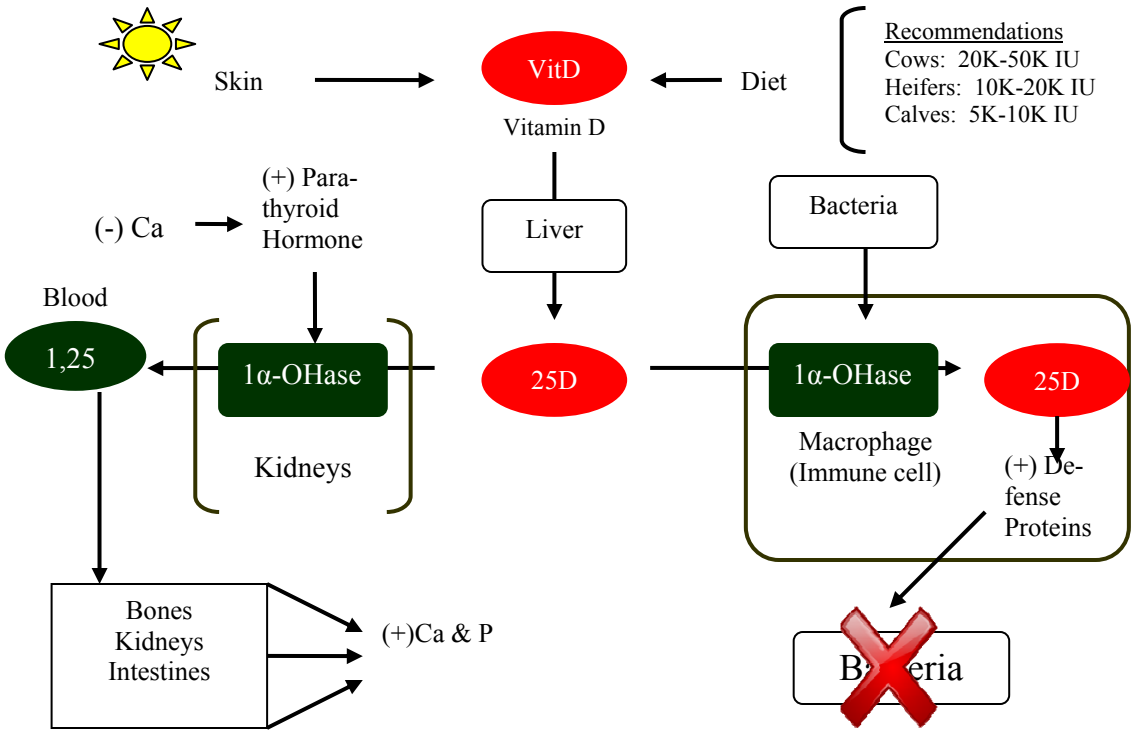

Figure 1. Functions of vitamin D in cattle.

Vitamin D metabolism and functions in cattle. Vitamin D (VitD) is quickly converted to 25-hydroxyvitamin D (25D) in the liver. The metabolite is the precursor to the active vitamin D metabolite, 1,25-dihydroxyvitamin D (1,25D). Low calcium stimulates 1α-hydroxylase (1α-OHase) in the kidneys to convert 25D to 1,25D. The 1,25D metabolite stimulates the vitamin D receptors in the bones, kidneys and instestines to increase the calcium and phosphate concentrations in the blood. Bacterial associated molecules stimulate 1α-OHase activity in immune cells. The 1,25D produced in the immune cell stimulates the production of defense proteins to kill off bacteria.

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Phytate level and phytase efficacy on early broiler performance

Phytate is a common antinutrient in animal feed ingredients. It reduces availability and increases the excrement of nutrients. Phytases are enzymes that break down phytate and increase nutrient availability. The addition of phytase is a feasible and economical substitute for the more expensive sources of phosphorus (P) supplementation. Several recent articles have looked at determining levels of phytase incorporation for maximum efficacy in performance. It is becoming apparent that many factors must be considered when formulating for phytase incorporation. Considerations must be given to the type of phytase being used, the age of the animal, stage of production, availability of P from phytate in the diet ingredients and the interactions with other nutrients in the diet. For example diets with excessive amounts of calcium reduce the solubility of phytate and the resulting absorption of P by broilers.

Experiment

The recent study by dos Santos et al. (2014) compared the efficacy of two commercially available *Escherichia coli* phytases on broiler performance and bone ash. The study was conducted using 1,024 male, one-day old Arbor Acres broilers. Birds were divided by pens into treatment groups. Each pen had ad libitum access to water and semiautomatic feeders.

Two treatment diets were formulated as basal diets containing low levels of available P (1.8 g/kg) with low (6.40 g/kg) or high (10.65 g/kg) phytate levels. Additionally, supplemental mono-dicalcium phosphate was added to ensure adequate available P or one of the two commercially available *E. coli* phytases at 500 phytase units (FTU)/kg. Phytase A was an enhanced *E. coli* phytase produced in *Trichoderoma reesei.* Phytase B was an *E. coli* phytase produced in *Pichia pastoris.* The supplementation of the diets allowed for the creation of eight experimental diets. Phytase A was found to have an analyzed activity of 4,950 FTU/g and phytase B had an analyzed activity of 6,060 FTU/g. A phytase unit is defined as the amount of enzyme required to release one µmol of inorganic P per minute from sodium phytate at 37° C and pH of 5.5.

Findings

When birds were fed diets with low available P supplemented with high phytate, the birds were significantly lighter, ate less, demonstrated higher feed conversion rates and reduced livability. Supplementation with mono-dicalcium phosphate increased feed intake, body weight gain, improved feed conversion rate, and increased livability regardless of the level of phytate in the diet. This finding indicated a significant P source x phytate interaction. Reducing the dietary phytate concentration from 10.65 to 6.40 g/kg improved performance. Studies by Liu et al.(2008) and Li et al. (2000) demonstrated this result as well. Additionally, livability results were extremely high; dead and culled animals were only observed on the low avP diets before phytase inclusion. The lower livability can be explained because the average P level of the diet was below bird requirements.

What this means for the industry

Phytate is in vegetable/plant feed ingredients which is destroyed by the activity of the enzyme phytase. Many factors influence phytase activity. Those include the catalytic and kinetic characteristics of the enzyme, the phytate concentration of the substrate, and the conditions where the reaction takes place in the digestive tract (the pH, time, and temperature). Any studies looking at phytate level effects on animal performance have to determine if the results are due to changing phytate concentrations or because of the ingredient composition of the diet(s) (Ullah, et al., 2008). One also has to determine if the results are influenced by the efficacy of the commercially available phytase. Bird age, Ca and P levels in the diet also influence how effectively the phytase hydrolyzes the phytate. Different phytase products react differently to gut conditions and the enzymatic affinity to the phytate in the ingredients. The animals themselves can influence the efficacy of phytase supplementation. Their production phase, feed intake levels, mineral requirements and immune status are all contributing factors to phytate hydrolysis. There is no clear standard defined to date and it will be further into the future before the standards are established.

Information for this article taken from:

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