THE CATION-ANION DIFFERENCE CONCEPT: WHERE ARE WE NOW?

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INTRODUCTION

Before answering the question posed by the title, we should first review what the dietary cation-anion difference (**DCAD**) concept is and what past investigations have revealed. The DCAD is an expression used to quantify the relative charge, positive or negative, of a feedstuff or a total mixed ration. To calculate DCAD, use the following formula:

$$[\%Na/0.023 + \%K/0.039] - [\%S/0.016 + \%Cl/0.0355] = meq/100g\ DM$$

This equation will yield the milliequivalents of charge per 100 g of diet DM; positive if the diet has a relatively greater concentration of cations (Na, K) and negative if the anionic side (S, Cl) of the equation is greater.

In general, prepartum diets with DCAD values less than zero, "negative" or "anionic" diets, offer effective control of periparturient metabolic disorders associated with the reduced concentrations of blood calcium observed at the onset of lactation (milk fever, retained placenta, downer cow syndrome).

Basal diets fed to dry cows generally have DCAD values between +20 and +30. Therefore, to implement the negative DCAD solution to the problem of hypocalcemia, anionic salts (i.e. calcium sulfate, calcium chloride, ammonium sulfate, ammonium chloride, magnesium sulfate, calcium sulfate, calcium chloride) are supplemented to lower the ration DCAD to some negative value.

PAST EFFORTS

Until recently much of the DCAD work has focused on establishing how effective the concept was with respect to milk fever prevention. The modern era of DCAD research began in Canada in 1984. Elliot Block fed two prepartum diets having DCAD values of +33 or -13 meq/100g. The anionic salt supplemented diet (-13) reduced the incidence of clinical milk fever to 0% from 47%.

Others have also observed significant reductions in the incidence rate of clinical hypocalcemia. Oetzel et al. (1988) reduced milk fever incidence from 17% to 4% by feeding a -8 DCAD prepartum diet.

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Comparing diets with DCAD values of +98 and -23, Goff et al. (1991) reported that the anionic salt supplemented cows had a milk fever incidence of 4%, reduced from 26% in cows fed the cationic diet.

The largest study to date on this subject, conducted by researchers at the University of Florida, was reported in 1992. Beede et al. found that by lowering the prepartum diet DCAD from +5 to -25, the incidence of milk fever was cut by over 50%.

The incidence rate of all metabolic disorders was reduced, by anionic salt supplementation, in a study by Joyce et al. (1994a) which compared prepartum diet DCAD values of +35, +30, and -7.

FUTURE WORK

Where then does the next thrust of work with the DCAD concept take us? As evidence supporting the efficacy of anionic salts has mounted over the past decade, nutritionists are, more and more, recommending that their clientele include anionic salts in the pre-fresh ration. Yet, for all the success of negative DCAD diets in reducing periparturient metabolic problems, a basic question remains; "How do they do what they do?" Presented next is the biology of milk fever followed by the parts of the puzzle uncovered so far in answer to this question.

Milk Fever is caused by a general failure in the systems that access and utilize the reservoir of calcium in bone tissue and calcium supplied via the diet. Several mechanisms are interrelated and responsible for the maintenance of a stable concentration of circulating calcium. The responsible tissues are the kidney, controller of urinary excretion; the intestine, where calcium is absorbed into the system; and the bone, principle organ of calcium storage. The major calcitropic endocrine controls of calcium metabolism are parathyroid hormone, vitamin D and its metabolites, and calcitonin.

At parturition, the commencement of lactation causes a drain of the calcium available in the circulating pool for the formation of colostrum. To illustrate the extent of this loss, consider that colostrum may contain 2.3 g of calcium per liter. Therefore, production of 10 liters of colostrum would require 23 g of calcium for one milking. Twenty-three grams of calcium is approximately 9 times the amount of calcium present in the entire plasma pool (Goff et al., 1987).

As blood calcium concentration drops, the parathyroid gland begins to secrete parathyroid hormone (**PTH**). The target organs of PTH are the kidney and bone. At the kidney, PTH causes the increased production of activated vitamin D and reduces the excretion of calcium in the urine. Vitamin D and PTH act synergistically on the bone to increase bone resorption of calcium. At the intestine, vitamin D stimulates the production of calcium biding protein(s). These protein(s) carry dietary and endogenous calcium from the lumen of the gut across the brush border and transport it into the bloodstream. Together these systems act to restore the concentration of blood calcium to the normal level of 8-10 mg/dl.

Therefore, physiologically speaking, the options when it comes to improving periparturient calcium status are, 1) calcium excretion via the urine must be reduced, 2) calcium mobilization from the bone must be increased and/or, 3) calcium intake from the gut must be enhanced.

In the attempt to understand the mechanism behind the efficacy of a negative DCAD prepartum diet, we can eliminate the first option. All studies in which anionic salts were fed and urinary calcium excretion quantified report significant increases in urine calcium concentrations (Fredeen et al., 1988, Gaynor et al., 1989, Wang and Beede, 1992). Leaving, then, two possibilities to explain the impact of anionic salts. Unfortunately, even with only two choices left, research investigating the "how?" questions of anionic salt supplementation has produced conflicting results.

THE CASE FOR BONE RESORPTION

When Block (1984) compared the effects on periparturient health for cows fed diets with a +33 or -13 DCAD, he concluded that the improved calcium status of cows fed the anionic salts
was due to increased bone resorption. Cows resorbed bone at an increased rate to access the buffering systems needed to counteract the mild acidosis resulting from consumption of the salts. To make this determination, Block quantified the concentrations of hydroxyproline in the blood. Hydroxyproline, an amino acid unique to collagen and bone tissue, is used as a measure of how actively bone tissue is being formed and resorbed.

In 1991, Goff et al. found that by lowering the DCAD of the prepartum diet from 98 to -23 meq/100 g diet DM, calcium status periparturiently was improved for cows fed the -DCAD diet. Further, the hydroxyproline concentration in the blood was significantly higher in cows fed the anionic salts. Again, this seems to indicate that dissolving bone tissue was the agent used to increase blood calcium.

THE CASE FOR INTESTINAL ABSORPTION

Belgian research in 1978 (Lomba et al.) pooled and analyzed data from across several dietary treatments and found that a significant correlation existed between calcium absorption and DCAD when cows were in positive calcium balance. Cows fed -DCAD diets possessed improved ability to absorb dietary calcium from the intestine compared with cows fed cationic diets.

Work done in Idaho by Joyce et al. (1994b) found that cows fed anionic salts prepartum (-7 DCAD vs. +30 DCAD and +35 DCAD) had improved calcium status at parturition. However, as no differences existed in hydroxyproline concentrations between treatments, it was deemed that the improved calcium status observed was at least partially attributable to enhanced intestinal absorption.

OTHER FACTORS

Clearly, nothing so complex as the interaction of biological factors to influence blood calcium concentration can be reduced to a simple black or white. What we gain by simplifying and categorizing the choices into these two general theories is a starting point for the consideration of the subject. However, this discussion would be incomplete without a mention of how other factors may also be playing a role when we see one cow fed anionic salts succumb to milk fever while most others remain healthy.

The final piece of our puzzle may lie somewhere in the transmission of signals from lowered blood calcium to the parathyroid gland to the kidney to the bone and to the gut. Using regression analysis, Goff et al. (1991) demonstrated that the PTH response to decreased blood calcium was similar for cows fed both the cationic and anionic diets. Theoretically, similar levels of PTH should have led to a similar kidney response in the production of activated vitamin D. Differing slopes for the lines regressing vitamin D on PTH suggest that the kidneys of cows fed the cationic diet were refractory to PTH stimulation. In the work from Idaho, Joyce et al. (1994b) found an improved vitamin D response to decreasing blood calcium for cows fed anionic salts compared with cows fed the cationic diet. Lastly, in 1992, Goff et al. reported that cows fed anionic salts prepartum had an increased concentration of vitamin D receptors in the colon mucosa postpartum.

Taken together, these bits of evidence suggest that neither enhanced intestinal absorption nor increased bone resorption should be solely congratulated for aiding cows fed anionic salts in the maintenance of blood calcium concentrations. But instead, we are left to conclude that these mechanisms may both be involved. And the reasons that one system improves more than the other in a particular herd remain open for debate.

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