Revisiting Negative Dietary Cation-Anion Difference Balancing for Prepartum Cows and its Impact on Hypocalcaemia and Performance

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Introduction

Much attention has been paid to dairy cattle nutrient requirements for maintenance and production for the major nutrient classes: protein, carbohydrates, fats, vitamins and minerals. Requirements are usually established in feeding trials using graded levels of nutrients and defining optimal performance or blood concentrations. These incorporate a factorial approach utilizing the knowledge of nutrient costs for maintenance and production, or by simulating the animal itself as a computer model. Ruminant nutritionists have been devoting much effort to subdividing the major nutrients into more specific subclasses with the goal of defining requirements more precisely to allow for better ration formulation from chemical composition of feedstuffs.

Protein, for example, is defined into rumen degradable, undegradable and soluble and insoluble fractions with further subdivisions into peptide, free amino acid, and ammonia nitrogen subclasses. Likewise, carbohydrates are divided into structural and non-structural carbohydrates with further subdivisions for each of these subclasses into their constituents.

Minerals, however, present a very different picture. This class of nutrients includes a plethora of individual components that have unique requirements and may or may not have any biological connection to each other.

Minerals have been subdivided into two subclasses, macrominerals and microminerals. This classification is not based on any biological function, but is rather based on the quantities found in animal tissues and feedstuffs with macrominerals found in percentage quantities and microminerals found in parts per million (or lower) quantities−hardly a basis for ration formulation.

Minerals are more integrally a part of all biological functions in the body than any other single class of nutrient. The functions include:

- expression and regulation of genes
- enzyme systems that regulate cellular function
- activity and functionality of vitamins

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- osmotic balance
- detoxification
- immunity
- cell membrane function
- acid-base balance and regulation
- structural (i.e. bone)

Nutrient interactions between and within nutrient classes have been researched for years (protein-to-energy, essential-to-nonessential amino acids, saturated-to-unsaturated fatty acid ratios, etc.). Comparatively, there is a paucity of literature within ruminant nutrition with regard to interaction between minerals and between minerals and other nutrient classes. This is not due to a lack of interest or importance but rather due to the extreme complexities of varying one mineral in a diet while keeping all others constant. Minerals cannot be added to a diet in their elemental forms but as salts that are combined with other minerals (NaCl, $CaCO_3$, $MnSO_4$, etc.).

This is not to say that that the subject of mineral balances, ratios and interactions is absent from the literature. Certainly subjects such as ratios of **calcium (Ca)** to **phosphorus (P)** and **nitrogen (N)** to **sulfur (S)**, interactions between **magnesium (Mg)** and **potassium (K), manganese (Mn)** and **copper (Cu)**, and vitamin E and **selenium (Se)** have been investigated and addressed. However, there are no unifying concepts on mineral balances as of yet.

The Concept of Balancing Anions and Cations in Rations

Shohl and Sato (1923) were the first to propose that mineral interrelationships were related to acid-base status. Shohl (1939) proposed that maintenance of normal acid-base equilibrium required excretion of excess dietary cations and anions. He hypothesized that consumption of either, in excess of the other, resulted in acid-base disturbances.

Once animal nutritionists began to test this hypothesis, mineral interrelationships were found to affect numerous metabolic processes. Leach (1979) and Mongin (1980, 1981) reviewed related literature and concluded that mineral interrelationships had profound influences. They theorized that for an animal to maintain its acid-base homeostasis, input and output of acidity had to be maintained. It was shown that net acid intake was related to the difference between dietary cations and anions. The monovalent macromineral ions, Na, K, and Cl were found to be the most influential elements in the expression for poultry (Mongin, 1980; 1981) and Na, K, Cl, and S (sulfur) were found to be the most influential elements in the expression for dairy cattle (Dishington, 1975). Since that time, several other minerals have been tested in various equations to verify that acid base status is the major physiological event controlled by these minerals, or more specifically, acid base status is regulated by hydrogen ion concentration in blood and affects the buffering capacity of blood or tissue.

This review will show that calculating the difference in milliequivalents of cation and anion intake will lead to a positive or negative value:

- Positive values (more cations than anion) leads to the production of more blood buffers and less hydrogen (alkalinity)
- Negative values (more anions than cations) leads to the reduction of blood buffers and allows for accumulation of hydrogen (acidity)
- The cations and anions that are most influential in this regard are Na, K, Cl, and S.

The Development of the Dietary Cation-Anion Difference Equation

Blood pH is ultimately determined by the number of cation and anion charges absorbed into the blood. If more anions than cations enter the blood from the digestive tract, blood pH will decrease. Mongin (1980) was one of the first to propose a three-way interrelationship among dietary Na, K and Cl. He proposed that the sum of Na + K - Cl [in meq per 100 g diet **dry matter (DM)**]) could be used to predict net acid intake. This sum commonly has been referred to as the dietary cation-anion balance (Tucker et al., 1988) or dietary electrolyte balance (West et al., 1991). However, Sanchez and Beede (1991) coined the term **dietary cation-anion difference (DCAD)** to represent more precisely the mathematical calculation used and avoid the erroneous connotation that mineral cations truly are balanced with mineral anions in the diet.

Numerous equations have been published for the calculation of DCAD in dairy cattle diets. The first published equation (Ender et al., 1971) was (Na + K) − (Cl + S). Since then, other DCAD equations have been proposed to account for the contributions of other macromineral ions that possibly affect acid-base status but are not completely bioavailable. These longer equations were:

- (Na + K + 0.38 Ca + 0.30 Mg) − (Cl + 0.6 S + 0.5 P) (Horst and Goff, 1997)
- (Na + K + 0.15 Ca + 0.15 Mg) − (Cl + 0.2 S + 0.3 P) (Horst and Goff, 1997)
- (Na + K + 0.15 Ca + 0.15 Mg) − (Cl + 0.6 S + 0.5 P) (National Research Council, 2001).

A fifth equation, (Na + K) − (Cl + 0.6 S), was recently proposed by Goff et al. (2004), which discounts the acidifying effects of S by 40% compared to the original 4-mineral equation [(Na + K) − (Cl + S)].

Two recent publications helped to clear up the question of all of the above proposed equations (Charbonneau, et al., 2006 and Lean et al., 2006). Both manuscripts were meta-analyses including substantial databases. They concluded the two equations, (Na + K) – (Cl + S) and (Na + K) – (Cl + 0.6 S), predicted animal responses with similar accuracy. The manuscript by Lean et al. (2006) used a larger data base in more of a multivariate approach and had a slightly higher r-squared for the former equation than the later compared with the manuscript by Charbonneau et al. (2006). Therefore, at this point, we are supporting the use of the straightforward original

4-mineral equation [(Na + K) – (Cl + S)], which is the equation used by many existing ration formulation programs.

Calculating DCAD

To actually calculate DCAD, mineral concentrations are first converted to **milliequivalents (meq)** as follows:

$$\text{meq/100 g} = \frac{(\text{milligrams})(\text{valence})}{(\text{g atomic weight})}$$

As an example, the meq (Na + K) – (Cl + S) value of a diet with 0.1% Na, 0.65% K, 0.2% Cl and 0.16% S will be calculated. In 100 g of this diet there are 100 mg Na (0.10% = .10 g/100 g or 100 mg/100 g), 650 mg K (0.65% K), 200 mg Cl (0.2% Cl), and 160 mg S (0.16 % S) per 100 g diet DM. Therefore, this diet contains:

$$\text{meq Na} = \frac{(100\text{ mg})(1\text{ valence})}{(23\text{ g atomic weight})} = 4.3\text{ meq Na}$$

$$\text{meq K} = \frac{(650\text{ mg})(1\text{ valence})}{(39\text{ g atomic weight})} = 16.7\text{ meq K}$$

$$\text{meq Cl} = \frac{(200\text{ mg})(1\text{ valence})}{(35.5\text{ g atomic weight})} = 5.6\text{ meq Cl}$$

$$\text{meq S} = \frac{(160\text{ mg})(2\text{ valence})}{(32\text{ g atomic weight})} = 10.0\text{ meq S}$$

The next step is to sum the meq from the cations and subtract the meq from the anions:

DCAD = meq (Na + K) - (Cl + S) = 4.3 + 16.7 – 5.6 – 10.0 = **+ 5.4 meq/100 g diet DM**.

Another way to calculate DCAD directly from the percentages of minerals present is to use:

DCAD = [(%Na in DM/0.023)+(%K in DM/0.039)]-[(%Cl in DM/0.0355)+(%S in DM/0.016)].

For example, using the same numbers as above:

DCAD equals (0.10% Na/0.023) + (0.65% K/0.039) - (0.2% Cl/0.0355) - (0.16% S/0.016) **= +5.4 meq/100 g diet DM.**

DCAD Impacts Blood Acid-base Status Directly and Predictably

There are numerous trials and meta-analyses published that demonstrate that altering DCAD intake of cows at any stage of the lactation cycle directly affects blood and urinary pH as well as blood HCO_3 and base excess (Sanchez et al., 1994; Spanghero, 2004; Apper-Bossard et al., 2006; Lean et al., 2006; Charbonneau et al., 2006; Hu and Murphy, 2004; Hu, et al., 2007).

The publications cited above show that within the boundaries of physiological homeostasis, there is a fairly linear and positive relationship between DCAD and blood and urinary pH as well as blood bicarbonate. On the alkaline side of blood acid-base balance, once blood pH reaches approximately 7.4 to 7.45 the response curve flattens and is non-linear. Once blood bicarbonate (HCO_3) reaches 28 to 30 meq/L the response curve also takes on a flatter, non-linear shape.

This can be illustrated in Figure 1 (Hu and Murphy, 2004) examining the relationships between DCAD, blood pH, blood HCO_3, and urinary pH in mid lactation cows in a meta-analysis involving 17 published trials and 69 feeding treatments.

Thus far we have discussed how cations and anions affect physiology, the development of the DCAD equation, how the specific minerals included in the DCAD equation can affect various physiological processes that lead to altered acid-base chemistry, and the direct relationship between DCAD and acid-base physiology. The remainder of this publication will examine the case for altering DCAD in dairy cattle to support health and production in their life cycle.

The Case for Manipulating DCAD in Dairy Cows: Negative DCAD for Prepartum Transition Cows

Many years ago, researchers discovered that a diet that reduced blood pH caused the concentration of blood calcium to increase and reduced the incidence of clinical hypocalcemia (milk fever) (Ender et al., 1962, 1971; Dishington, 1975). The concept of achieving this reduced blood alkalinity by lowering DCAD and the resulting effects on blood calcium at calving were introduced in North America by Block (1984) and further explained by the same author in a later publication (Block, 1988). This led to the practice of feeding diets with more anions relative to cations to help reduce the incidence of clinical milk fever.

DCAD and Blood Calcium at Calving

The onset of lactation causes a severe and rapid drain on blood calcium required to produce milk. If this blood calcium is not replaced as rapidly as it is reduced via bone calcium release (resorption) or intestinal absorption of calcium cows will become hypocalcemic with some developing clinical milk fever. Reducing DCAD to negative values has been shown by many authors to prevent this rapid decline in blood calcium at calving. This is best illustrated in Figure 2 and Table 1. Giesy et al. (1997) showed that when different DCADs were fed to cows that were challenged with an infusion of EDTA (ethylene-diamine-tetra acetic acid) − to remove calcium from blood−that cows maintained their blood calcium better as the DCAD was reduced (Figure 2). In a trial with periparturient cows, Leclerc and Block (1989) showed a highly significant negative correlation between DCAD and concentration of blood calcium, which was strongest from 12 hours pre- to 12 hours post-partum (Table 1). Therefore, as DCAD is reduced prepartum, blood calcium concentration is maintained at a higher level around parturition.

There are several physiological possibilities to explain how negative DCAD (acidic conditions in the blood) helps to maintain blood calcium. The three major ways to get more calcium in the blood are via intestinal absorption, bone resorption (mobilization), and kidney reabsorption. As explained by Block (1988), it is unlikely that manipulating DCAD will directly affect intestinal absorption of calcium. There is good evidence that the kidneys play a role but not by reabsorbing calcium and putting it into the blood. Rather, the effect of chronic acidosis on the kidney is to increase excretion of calcium (Goulding and Campbell, 1984; Lemann et al., 1967). Feeding negative DCAD can produce this acidosis and cause an increase in urinary calcium excretion (Takagi and Block, 1991), thereby reducing calcium retention, and causing the vitamin D-parathyroid hormone axis to increase the signals for bone mobilization of calcium.

Furthermore, metabolic acidosis directly increases bone mobilization of calcium by: (1) creating the necessary acidic environment for lysosomal and mitochondrial enzymes in the osteoclasts (bone mobilization cells) to operate; (2) allowing for the rapid production of other lysosomal and cytoplasmic acids in these cells, such as lactic and hyaluronic acids and;(3) allow for a localized reduction in pH around the bone cells to allow for bone mineral dissolution.

Another major finding was that mild alkalosis (high DCAD) reduces the ability of the periparturient cow to maintain calcium homeostasis at or near calving by reducing tissue responsiveness to parathyroid hormone (Goff et al., 1991; Phillippo et al., 1994). Low DCAD diets ($\leq$ -5 meq/100 g diet DM) through addition of anions increases target tissue responsiveness to parathyroid hormone (Horst et al., 1997).

Clinical and Subclinical Hypocalcemia (Milk Fever)

In a 2007 survey (USDA, 2009) 83.5% of all dairies in the United States reported clinical milk fever as a health problem with an incidence rate of 4.9%. This figure is only slightly below the estimated incidence in 1993 of five to seven percent (Jordan and Fourdraine, 1993). Research indicates that cows with clinical milk fever produce 14% less milk in the subsequent lactation and their productive life is reduced approximately 3.4 years when compared to non-milk fever cows (Block, 1984; Curtis et al., 1984). Furthermore, cows that recover from milk fever have an increased risk of ketosis, mastitis (especially coliform mastitis), dystocia, left displaced abomasum, retained placenta, and milk fever in the subsequent lactation (Curtis et al., 1984; Wang, 1990; 1993; Oetzel et al. 1988). Guard (1996) estimated the average cost per case of milk fever to be $334. In addition, cows with milk fever are also susceptible to secondary disorders, such as ketosis, mastitis, retained placenta, and displaced abomasums (Curtis et al., 1983).

In addition, and maybe of greater importance, is the prevalence of subclinical hypocalcemia (i.e. milk fever) which may be as high as 66% for multiparous dairy cows following calving (Beede et al., 1992). Subclinical milk fever occurs when the clinical symptoms of the disease are not seen but blood calcium still decreases substantially around parturition. Just as clinical milk fever, subclinical low blood calcium can lead to

low **dry matter intake (DMI)** postpartum, dystocia, ketosis, and retained placentas. Kimura et al. (2006) separated cows into those that had clinical milk fever and those that did not and showed that even the non-milk fever cows had a significant drop in blood calcium at parturition.

As first shown by Block (1984) and subsequently by others (Leclerc and Block, 1989; Roche et al., 2003; Penner et al., 2008) formulating diets with negative DCAD reduces the severity of the decline in blood calcium at calving, even when milk fever is not present.

The occurrence of hypocalcmia (clinical and subclinical) is a multifactorial disease that is not solely dependent on DCAD. In a comprehensive meta-anlytical model, Lean et al. (2006) described the four-mineral DCAD equation as a major contributor but also found other dietary and management factors had independent contributions (prepartum dietary calcium, magnesium, and phosphorus as well as breed and number of days fed the prepartum transition diet). Most of the prepartum diets included in this manuscript were relatively high in starch and low in fiber compared to the traditional far-off dry cow diet. However, it was recently shown that the positive effects of DCAD on blood calcium and postpartum milk production (discussed below) are prevelant as well when higher fiber, reduced starch diets are fed (Siciliano-Jones et al., 2008).

Prepartum DCAD and its Relation to Pre- and Post- partum DMI and Subsequent Milk Production

The effects of decreasing DCAD on prepartum DMI are equivocal. Horst et al. (1994) reported that the addition of >300 meq of anions/kg diet may reduce intake. Joyce et al. (1997) reported depressed DMI in multiparous cows supplemented with 471 meq anions/kg DM, whereas Moore et al. (2000) showed no decline in DMI for multiparous cows supplemented with 329 meq anions/kg DM; however, prepartum DMI was lower for heifers supplemented 329 meq anions/kg DM. In their meta-analysis, Charbonneau et al. (2006) show a negative relation between DCAD and prepartum DMI, although most individual studies did not show a significant effect. A few points stand out from the literature cited thus far:

The negative effects of DCAD on DMI appear obvious when more than 300 meq/kg (30 meq/100 g) of anionic salts are added to a diet. Heifers seem more sensitive to the anionic supplements than cows. Most of the reported research has been with anionic mineral supplements which might pose palatability problems when less than 300 meq/kg are needed.

Research reported with non-mineral based anionic supplements have not shown the DMI reduction prepartum as happens with mineral salt supplementation (DeGroot, et al., 2004; Siciliano-Jones et al., 2008).

Obviously, the prevention of milk fever will increase milk yield by about 14% in those cows that would have succumbed to the disease, will extend their productive life (Block, 1984; Curtis et al., 1984), and will reduce the incidence of other postpartum disorders (Curtis et al., 1983; Wang, 1990; 1993; Oetzel et al., 1988). In those trials that have measured postpartum milk production, multiparous cows produce more milk after being fed a prepartum diet with a negative DCAD irrespective of the occurrence of milk fever (Block, 1984; Joyce et al., 1997; Penner et al., 2008; Siciliano-Jones et al., 2008; DeGroot et al., 2010). It is not clear if this increase in production performance is directly related to improving blood calcium at calving or indirectly through reduction of other clinical and subclinical postpartum diseases. Estimated increases in milk yield when cows are fed prepartum diets with a negative DCAD range from 1,800 lbs (Degaris et al., 2004; Degaris and Lean, 2008) to 3,200 lbs (DeGroot et al., 2010).

Optimal Prepartum DCAD

Controlled experiments have not yet determined the optimal prepartum DCAD. The recommended target DCAD of -10 to -15 meq/ 100 g dietary DM may be lower than needed to achieve the desired changes in acid-base status and subsequent increases in blood Ca. However, this range of DCAD provides a margin of safety to account for varying concentrations of the minerals in feeds and K consumed from pasture or free-choice hay.

To illustrate the point of varying mineral contents of forages and to stress the need for routine forage mineral analyses by wet chemistry, Arm & Hammer Animal Nutrition has been conducting a DCAD forage testing program over the past few years. The largest data set was for the Northeast and North Central U.S. The results are shown in Tables 2a and 2b.

Tables 2a and 2b show that DCAD and individual minerals can and do have a large variation. Therefore, the chances for DCAD remaining negative may not be achievable if you balance it at near, or just below zero. A DCAD of -10 to -15 meq/ 100 g dietary DM would be more desirable to ensure that all cows always receive the low DCAD diet. It is also obvious from this table that forages have a relatively high DCAD. Grains and protein supplements tend to be slightly negative to slightly positive. Therefore, it would be unlikely to achieve a negative DCAD without the use of a specially formulated supplement containing anions.

Monitoring DCAD via Use of Urinary pH

Urinary pH and DCAD are directly and negatively related as shown in almost all cited publications above. In fact, Spanghero (2004) developed a model whereby urinary (and blood) pH can be predicted by knowing the DCAD intake of cows. Therefore, many practitioners will monitor urinary pH on a subset of prepartum transition cows to be certain that the DCAD is efficacious. Theoretically, urinary pH in individual cows less than 7.0 should be efficacious. Conversely, urinary pH less than 5.5 is too low. The

general accepted recommendation is to achieve urinary pH of 6.2 to 6.8 for Holstein cows and a bit lower for Jersey cattle.

Care must be taken in interpreting results. Because low DCAD reduces urinary pH, a clinical finding of higher pH values indicates that cows are not consuming the formulated DCAD; it does not indicate that negative DCAD does not work.

There are several possibilities to explore if a negative DCAD is being offered and cows are exhibiting high urinary pH values:

1. Cows are not consuming as much DM as expected;
2. Total ration mix was not adjusted for additional cows entering the pen;
3. Other supplements have not been accounted for (i.e., free choice minerals);
4. Forage mineral contents are changing and have not been evaluated for current DCAD values.

Methodology for Reducing Prepartum DCAD Using Products Available

Feed a negative DCAD ration in the prepartum transition period defined as the 21 days before expected calving. Most research examining the ideal time frame for transition diets have shown that 14 to 21 days prepartum maximizes cow performance and minimizes postpartum disease (Corbett, 2002; Degaris and Lean, 2008).

The steps to be taken are rather simple. The first step is to identify and isolate forages that are not high in K for use in the transition period. The second step is to remove any excess K and Na from the diet in excess of minimum requirements. Free choice salt and rumen buffers are not desirable during this time and will increase the risks associated with hypocalcemia. The final step is to supplement the diet with a product designed to deliver negative DCAD to a level consistent with a final DCAD of -10 to -15 meq/100 g DM.

Available Sources of Anions that can be Used to Reduce Prepartum DCAD

Generally, there are three classes of feedstuffs that can reduce DCAD:

1. Forages: purchased or produced on-farm.
   - Forages alone will not likely be able to reduce DCAD to acceptable negative values. However, careful selection of forages that are low in dietary potassium (K) can be used to reduce DCAD such that a minimal amount of purchased, specialty supplements would have to be used.
2. Anionic Mineral Supplements
   - The choices in this category range from purchasing specific minerals salts to reduce DCAD (eg., the chloride and sulfate salts of calcium, magnesium, and/or ammonium) to specially formulated mineral packs containing these and other salts that may or may not be mixed with flavor enhancers (distillers grains, molasses, brewers grains, etc.)

- Field experience with mineral salt products has been mixed from a palatability standpoint. They tend to be less palatable especially if more than 20 meq/100 g DM of anions has to be added to the diet.

3. Manufactured supplements not based on anionic salts designed to deliver a negative DCAD
   - These supplements tend to be value-added in that they bring additional benefits to the diet other than negative DCAD.
   - Field experience shows that these tend to be more palatable even when more than 20 meq/100 g DM of anions has to be added to the diet.
   - The two major products available with published scientific literature available to back up claims are BIO-CHLOR® (Church & Dwight Co., Inc., Princeton, NJ) and SoyChlor® (West Central, Ralston, IA).

One true assumption is that if cows are consuming the same level and amount of DCAD the physiological reaction to the negative DCAD will be to increase blood acid load and reduce urinary pH to the same degree.

The ability for BIO-CHLOR to benefit cows beyond the DCAD effect was shown by DeGroot (2004) in his Ph.D. thesis and the resulting manuscript (DeGroot et al., 2010). In this trial, prepartum cows were given one of four isonitrogenous, isoenergetic diets that differed only in DCAD. Milk production and feed intake were monitored for the first 21 days postpartum. The control diet had a DCAD of +20 meq/100 g DM. The other three diets had DCADs at -7 to -10 meq/100 g DM, each with a different supplementation regimen to deliver the negative DCAD. One used BIO-CHOR, the second used anionic mineral salts, and the third used a combination of a product called FERMENTEN plus some anionic mineral salts. This later treatment needs some explanation.

FERMENTEN is a supplement that has been shown to have the same impact on rumen fermentation as BIO-CHLOR (Lean et al., 2005) but has a higher DCAD for use in lactating cow diets. Therefore, if the theories on DCAD and BIO-CHLOR are correct, all treatments should outperform the control diet for postpartum performance, the BIO-CHLOR and FERMENTEN+anionic salts should outperform the anionic salt treatment and there should be no difference between the BIO-CHLOR and FERMENTEN+ anionic salts treatments.

Results for this trial are shown in Figures 7a, 7b, and 7c. There were no occurrences of milk fever in cows fed any of the diets and DMI was not different between treatments. Figure 4a shows the temporal pattern of postpartum milk production for the four prepartum DCAD treatments. Milk production was significantly improved in all cows fed all negative DCAD treatments over the controls. Even more striking is the start-up milk production where cows fed BIO-CHLOR or FERMENTEN+anionic salts produced upwards of 20 lbs more milk at startup than the control cows or those fed anionic salts. Figure 4b shows that the three negative DCAD treatments outperformed the controls by approximately 18 lbs. more milk per day in the first 21 days postpartum. Achieving a negative DCAD prepartum with either BIO-

CHLOR or FERMENTEN + anionic salts resulted in approximately 5 lbs. more milk per day than by using anionic salts alone (Figure 4c). Finally, as shown in Figure 4a, there were few differences in postpartum performance of cows fed prepartum BIO-CHLOR or FERMENTEN + anionic salts.

This trial shows the immediate postpartum benefit of feeding negative DCAD to prepartum transition cows even when clinical milk fever is not present. Moreover, this trial demonstrates the positive effects of BIO-CHLOR over anionic salts in achieving negative DCAD and corroborates that cows likely have better protein status at calving when fed BIO-CHLOR.

Conclusions

Milk and milk component production, disease incidence, and reproductive performance are each affected by an array of managerial, nutritional, and genetic factors. Further, these are interrelated to each other whereby each affects the outcome of the other. Certainly, there is no single nutritional or managerial factor that will guarantee or optimize the outcome within any of these functions but we can all agree that it is these three functions that ultimately affect profitability of the dairy.

The literature review and discussions above show convincing evidence that DCAD and its components play part of the integrated role affecting production performance and disease incidence for prepartum cows. While there are still unanswered questions regarding the specifics in calculating DCAD, the ideal DCAD levels to be formulated for in the diet, and some of the physiological consequences of altering DCAD, the evidence for feeding highly positive DCAD to lactating cows and negative DCAD for prepartum transition cows is extremely strong. While DCAD is not the single factor that will increase production or eliminate production related diseases, it is a major factor to consider in feeding cows.

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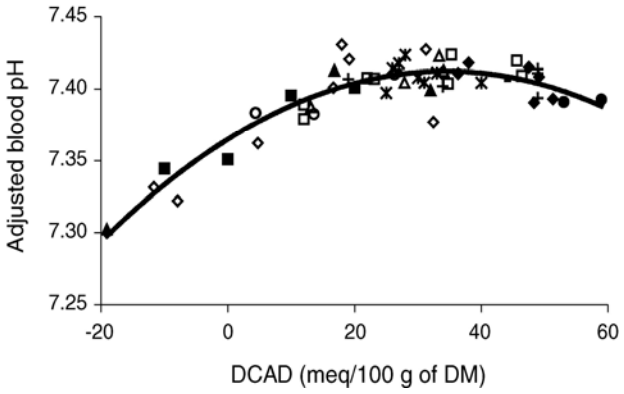
(a)



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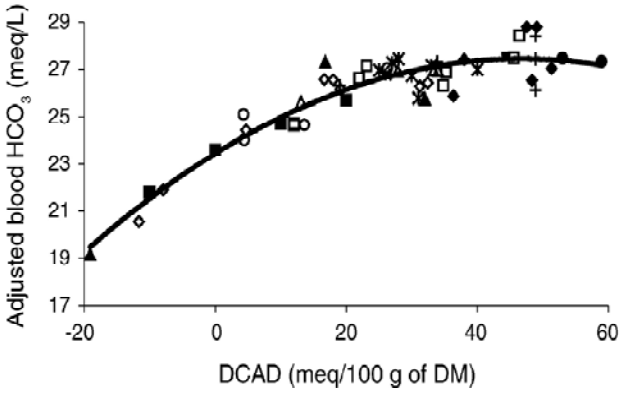

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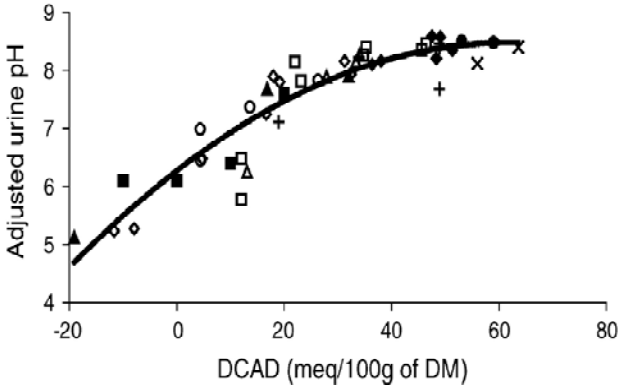


Figure 1. The relationship between blood pH (*a)*, **bicarbonate (HCO_3)** (*b)*, and urinary pH (*c)* with DCAD (Hu and Murphy, 2004).

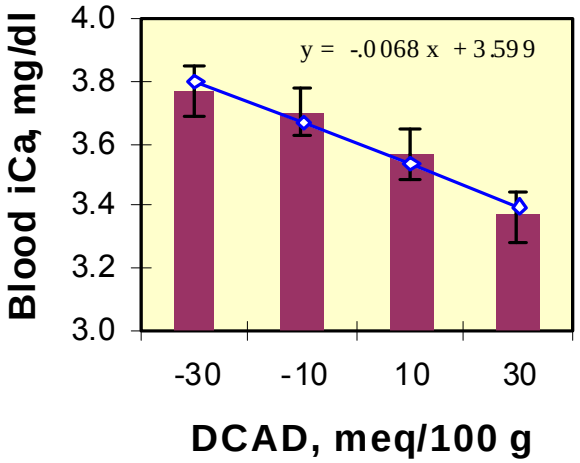


Figure 2. Blood ionized Ca response to four levels of DCAD following infusion with EDTA to mimic hypocalcemia (Giesy et al., 1997).

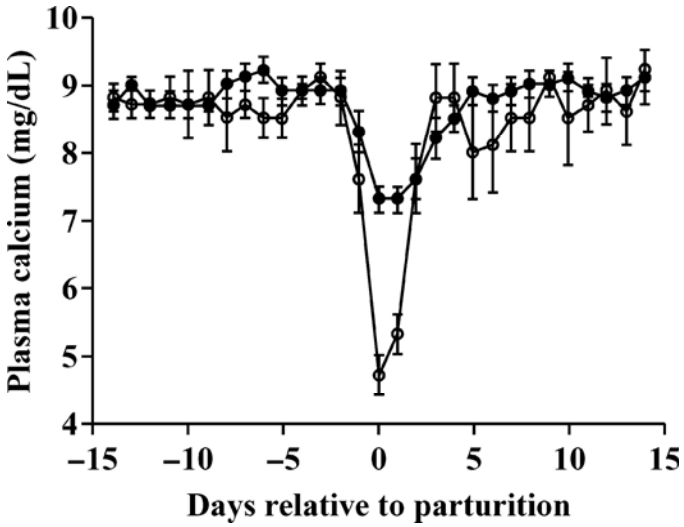
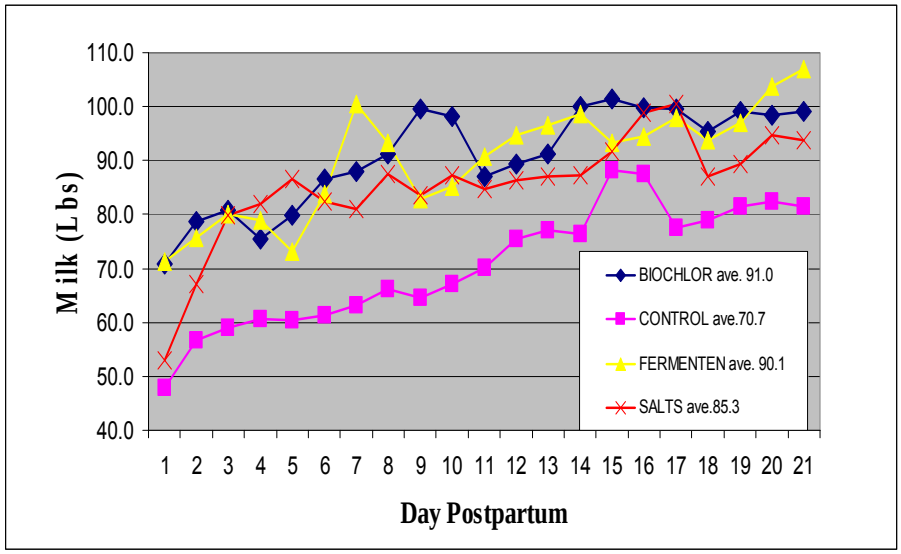
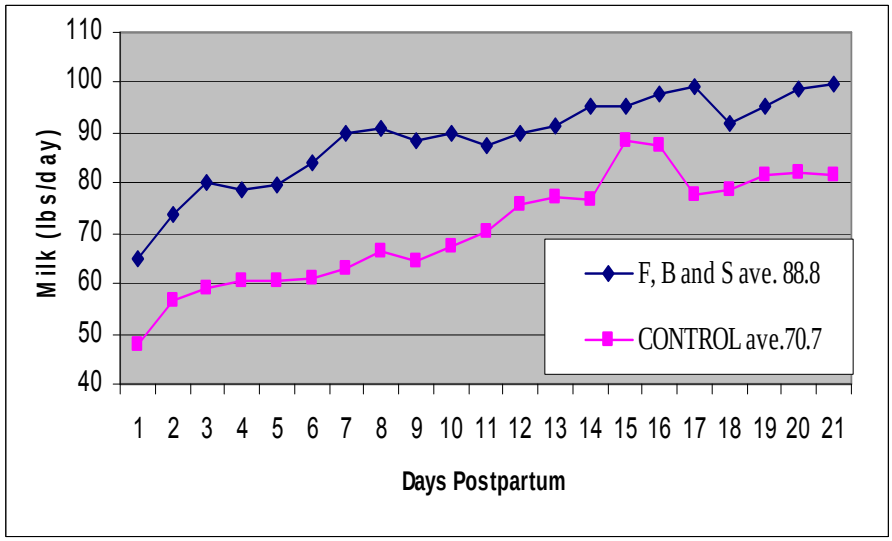


Figure 3. Plasma calcium concentrations (mean ± SEM) around the time of parturition in milk fever (n = 8) and non-milk fever (n = 19) cows; d 0 = day of parturition (from Kimura et al., 2006).

(a)


110.0
100.0
90.0
80.0
70.0
60.0
50.0
40.0
Milk (Lbs)
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
Day Postpartum
BIOCHLOR ave. 91.0
CONTROL ave.70.7
FERMENTEN ave. 90.1
SALTS ave.85.3

(b)


110
100
90
80
70
60
50
40
Milk (lbs/day)
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
Days Postpartum
F, B and S ave. 88.8
CONTROL ave.70.7

(c)

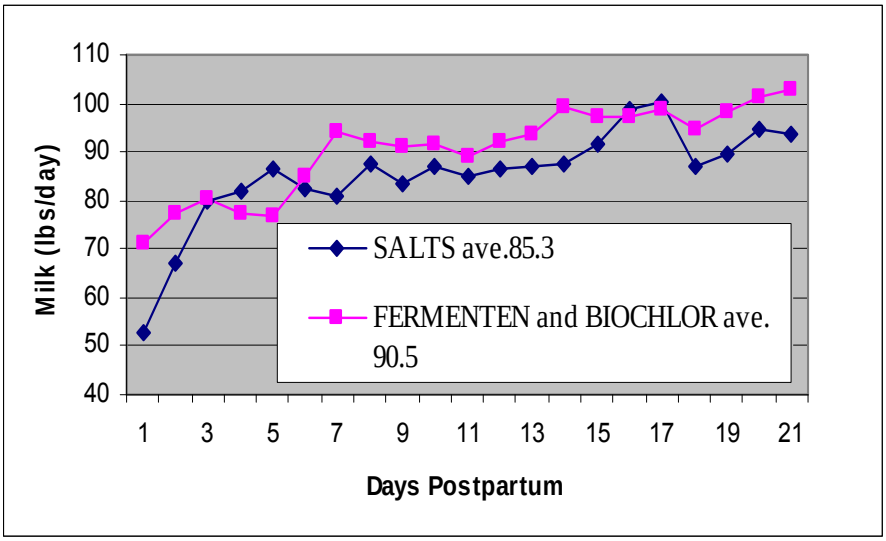


Figure 4. Postpartum milk production of cows fed prepartum transition diets with a DCAD of +20 or -10 meq/100 g DM. Negative DCAD was achieved by supplementing the control diet with anionic salts, BIO-CHLOR or FERMENTEN + anionic salts.

Table 1. Correlation between blood calcium concentration and DCAD from 48 hours prepartum to 36 hours postpartum (Leclerc and Block, 1989)

Time (hours)	Coefficient of correlation	Probability of significance (P>F)
48 prepartum	-0.47	0.048
36 prepartum	-0.38	0.121
24 prepartum	-0.33	0.162
12 prepartum	-0.46	0.046
Parturition	-0.55	0.015
12 postpartum	-0.59	0.013
24 postpartum	-0.27	0.248
36 postpartum	-0.46	0.058

Table 2a. Results from 2008 Arm & Hammer Animal Nutrition forage testing program for the Northeastern and North Central United States – DCAD variation

Northeast (ME, VT, CT, NY, PA, VA, MD, OH)					
Sample	n	Average DCAD	Minimum	Maximum	STDEV
Legume haylage	28	24.24	-5.28	63.30	17.72
Grass haylage	4	33.16	13.48	42.93	13.34
Balage	10	29.13	9.50	52.93	12.73
Corn Silage	47	24.84	-13.93	77.54	19.77
Small grain silage	8	42.45	18.44	76.27	21.36
Legume hay	18	22.82	8.66	49.13	11.13
Grass hay	18	31.11	0.61	51.96	16.76
North-central (MI, WI, IL, IN, IA, NE)					
Sample	n	Average DCAD	Minimum	Maximum	STDEV
Legume haylage	166	35.54	-9.21	82.62	18.37
Grass haylage	5	46.74	8.73	105.55	36.52
Corn silage	85	12.26	-0.21	46.94	8.53
Small grain silage	17	35.10	4.24	63.43	14.19
Legume hay	75	35.76	2.96	97.52	19.27
Grass hay	67	27.08	-9.50	119.28	20.52
Straw	7	14.03	-3.25	33.11	11.06

Table 2b. Results from 2008 Arm & Hammer Animal Nutrition forage testing program for the North Central United States – Individual mineral variation in legume haylage and corn silage

Sample Description	%Na	%K	%Cl	%S	DCAD
Legume haylage					
Average (n = 166)	0.09	2.39	0.57	0.22	35.54
MinimumN	0.00	0.82	0.02	0.07	-9.21
Maximum	0.82	3.88	1.60	0.35	82.62
STDEV	0.13	0.58	0.35	0.06	18.37
Corn silage					
Average (n = 85)	0.03	1.00	0.28	0.10	12.26
Minimum	0.00	0.32	0.10	0.05	-0.21
Maximum	0.58	2.72	1.14	0.29	46.94
STDEV	0.06	0.38	0.15	0.03	8.53

SESSION NOTES