DCAD Nutrition For Dairy Cattle

Research Summary

A complex concept with simple, yet profound, results

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Contents

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 defined 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 divided into rumen degradable, undegradable, soluble and insoluble fractions with further subdivisions into peptide, free amino acid and ammonia nitrogens. Likewise, carbohydrates are divided into structural and nonstructural carbohydrates with further subdivisions for each of these subclasses into their constituents.

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

Minerals have been divided 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
- 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 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 can be added as salts that are combined with other minerals ($NaCl$, $CaCO_3$, $MnSO_4$, etc.).

This is not to say 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, as yet there are no unifying concepts on mineral balances.

The objective of this *DCAD Balance Research Summary* is to elucidate and review the importance of the balance of dietary anions and cations (as macrominerals) in rations and in dairy cattle metabolism. To accomplish this, published and unpublished research data will be reviewed, as well as some widely accepted physiological chemistry.

THE CONCEPT OF BALANCING ANIONS AND CATIONS IN RATIONS

Background Research

Shohl and Sato (1922) 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, Sodium (Na), K and Chlorine (Cl) were found to be the most influential elements in the expression for poultry (Mongin, 1980; 1981) and Na, K, Cl and S 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 (meq) of cation and anion intake will lead to a positive or negative value:

- ◆ Positive values (more cations than anions) 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.

Development of the DCAD 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* (i.e., 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 four-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 database in more of a multivariate approach and had a slightly higher r-squared for the former equation than the latter compared with the manuscript by Charbonneau, et al. (2006). Therefore, at this point, we are supporting the use of the straightforward original four-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 meq as follows:

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

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% = 0.10 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:

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

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

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

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

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   =   (0.10% Na/0.023) + (0.65% K/0.039) – (0.20% Cl/0.0355) – (0.16% S/0.016)
             = +5.4 meq/100 g diet DM

The above calculations are based on actual molecular weights. The equation can be simplified further by using factors:

DCAD   =   (%Na x 43.48) + (%K x 25.64) - (%Cl x 28.17) - (%S x 62.5)

Using the preceding numbers:

| DCAD	=	(0.10%Na x 43.48) + (0.65%K x 25.64) - (0.20%Cl x 28.17) - (0.16%S x 62.5) = +5.4 meq/100g diet DM

Note: Some publications refer to DCAD per 100g dietary DM and others refer to DCAD per kg of dietary DM. The difference would represent the addition or deletion of a zero (i.e., 5.4/100gDM= 54/kg).

How DCAD Can Alter Acid-Base Physiology

One of the physiological processes that can affect acid-base status is intestinal absorption of these particular ions in the DCAD equation (Figure 1). In the posterior segment of the small intestine, when Na is in excess of Cl, the Cl is absorbed in exchange for a blood bicarbonate ion (HCO_3^-) to maintain electrical neutrality. If insufficient Na is present to allow for the electrically neutral absorption of NaCl, there can be an excess drain of blood HCO_3^-, leading to an acidotic condition in the blood. Alternatively, there is potential for intestinal exchange of ingested Na with circulating blood H^+ when Na is in excess of Cl. This event would lead to a metabolic alkalosis.

FIGURE 1. The effect of excess anions or cations on blood acid-base chemistry.
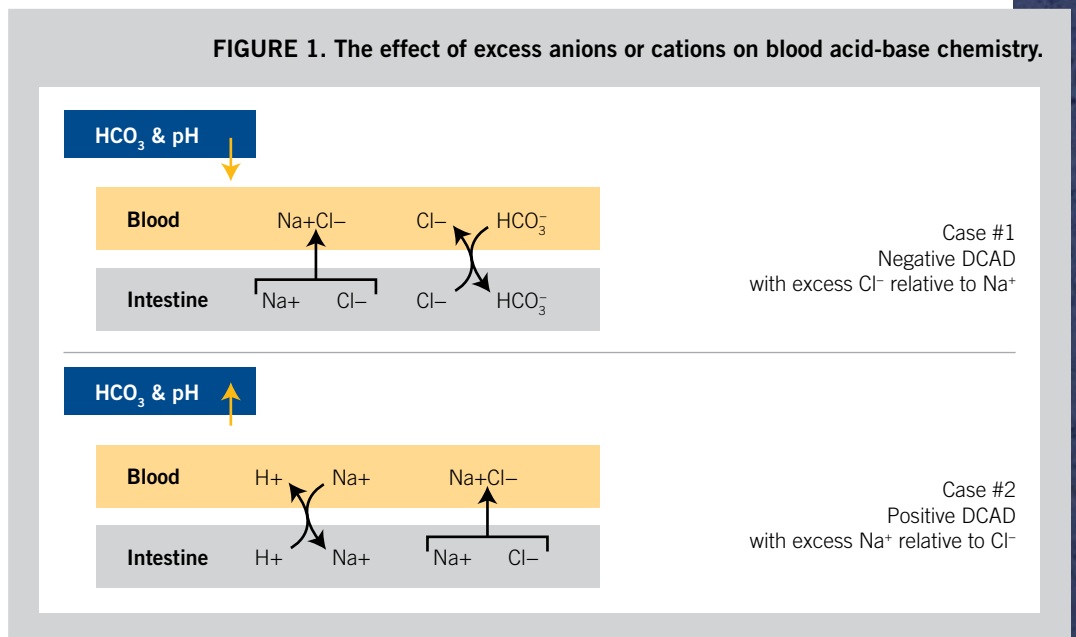
Another process involves the mobilization of hydrogen (H) in the proximal tubules of the kidney and secretion of H, as well as ammonia production in the distal tubules of the kidney (Figure 2). These processes depend on reabsorption of Na to neutralize (electrically) the absorption of HCO_3^- from the tubular cell to the blood. If excess Cl is present in the glomerular filtrate, Cl in the filtrate and HCO_3^- in the cell may exchange, resulting in NaCl reabsorption and a reduction of HCO_3^- absorption. Furthermore, when the animal is under the stress of mild acidosis in extracellular fluids, the kidneys conserve HCO_3^- ions by reabsorption while the reverse is true for alkalosis (Ganong, 1985). To maintain electrical neutrality the Cl ion is exchanged for HCO_3^- from tubular fluid because of a preponderance of Cl in the extracellular fluids. In this manner, optimal levels of Cl in relation to other ions are needed to maintain acid-base balance.

FIGURE 2. Role of Na in mobilization of H in proximal tubules (a), secretion of H in distal tubules (b), and ammonia production in distal tubules (c) (c.a. = carbonic anhydrase).
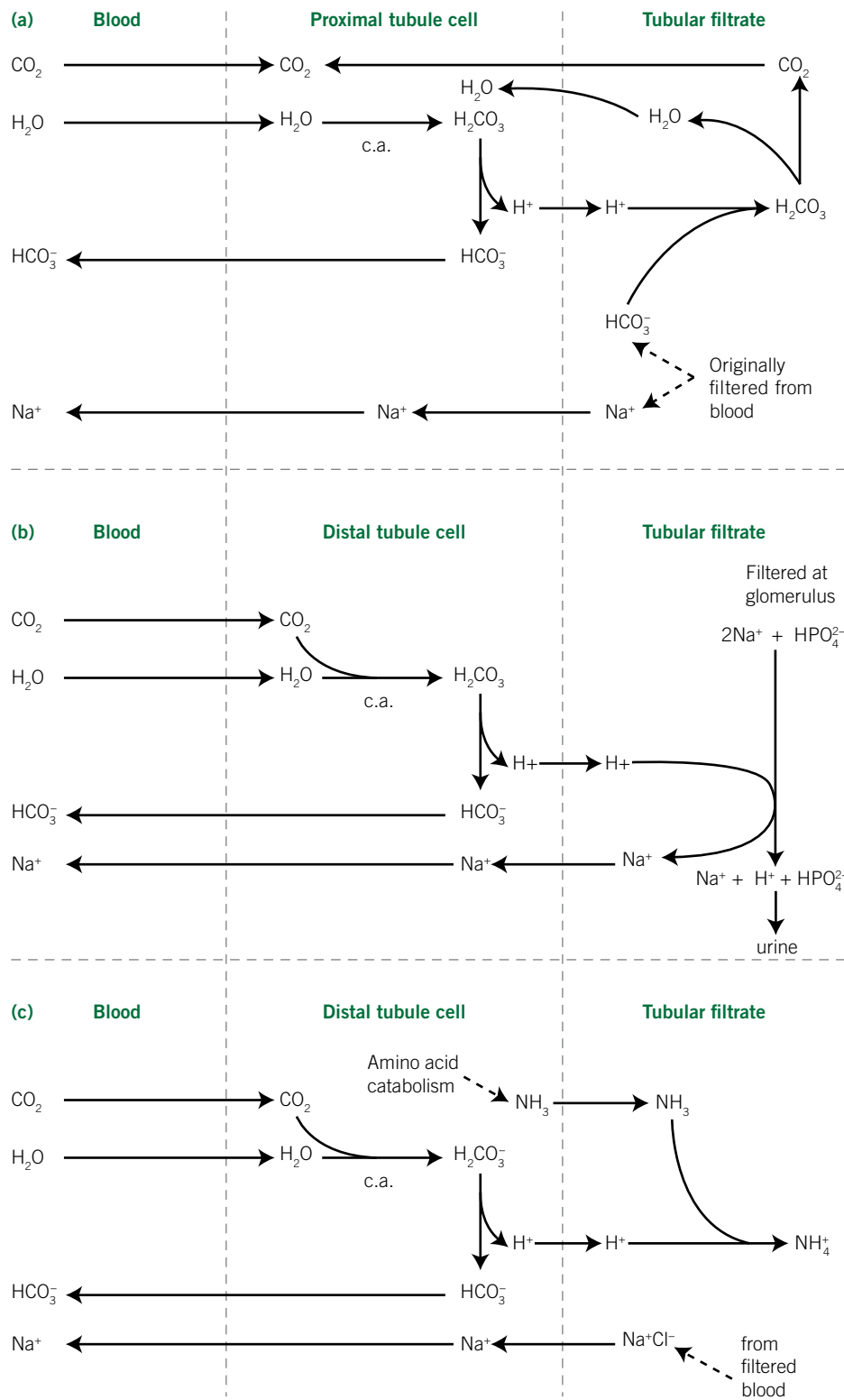
Source: Block, 1994.

The final mechanism to be discussed is the phenomenon called "the chloride shift." This is illustrated in Figure 3 where an integrated approach of erythrocytes (red blood cells) in tissue, plasma and lung is present while electrical neutrality is maintained. Figure 3 shows the principal protein buffer in blood, which is the potassium salt of oxyhemoglobin ($KHbO_2$). Carbon dioxide (CO_2) produced from tissue metabolism and respiration reacts with water (H_2O) to form carbonic acid (H_2CO_3) inside the erythrocyte. Some of the H_2CO_3 enters plasma while the rest reacts with $KHbO_2$ to form HCO_3^-, liberating oxygen (O_2) for respiration and K. The HCO_3^- enters plasma in exchange for Cl. Sodium bicarbonate ($NaHCO_3$) is formed in plasma and the Cl that entered the erythrocyte is neutralized by K released in the exchange of HCO_3^- with Cl. This reaction is reversible in the lungs where Cl that transfers back to the plasma neutralizes the Na released when HCO_3^- re-enters the erythrocyte for removal of CO_2 in respiration. Again, if these ions are out of balance with each other, even if present in nontoxic or nondeficient amounts, the production of alkalosis or acidosis is possible via insufficient exchange of HCO_3^- and hydrogen (H) (Georgievskii, 1981; Ganong, 1985).

FIGURE 3. Reaction of erythrocyte in tissue, lung, and plasma will change during respiration in relation to Na, K and Cl and the chloride shift.

Source: Block, 1994.
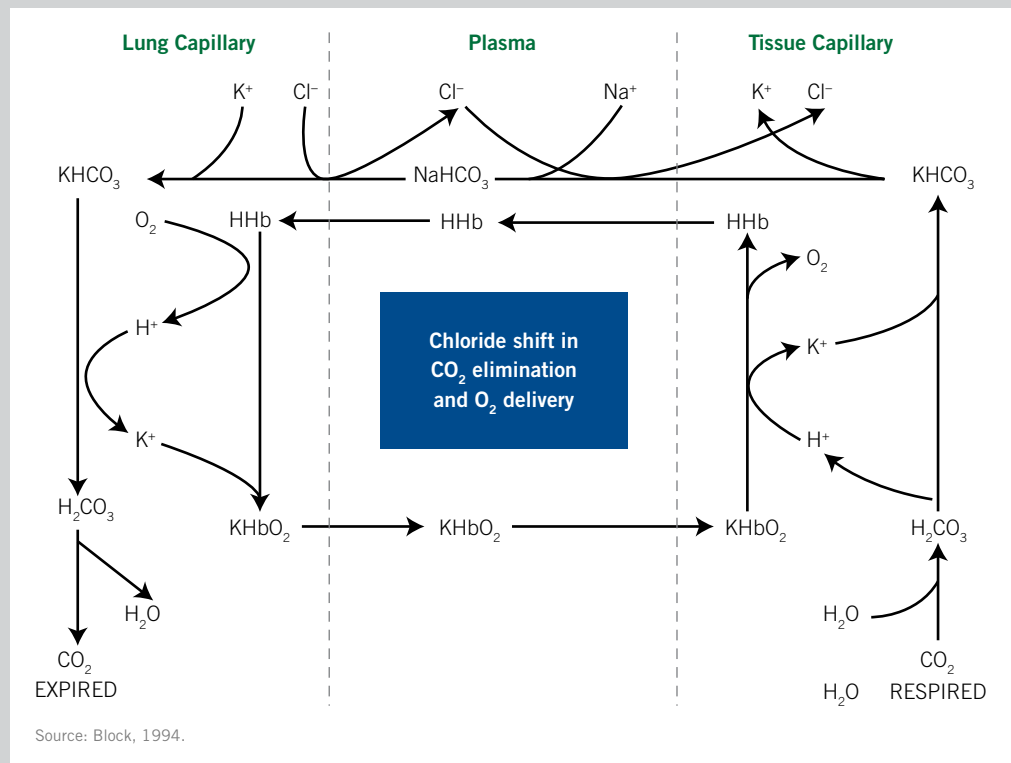
DCAD Impacts Urine pH/Acid-Base Relationship

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, 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 nonlinear. Once blood bicarbonate (HCO_3) reaches 28 to 30 meq/L the response curve also takes on a flatter, nonlinear shape.

Figure 4 examines the relationships between DCAD, blood pH, blood HCO_3 and urinary pH in midlactation cows in a meta-analysis involving 17 published trials and 69 feeding treatments (Hu and Murphy, 2004).

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 lifecycle.

FIGURE 4a, b, c. The relationship between blood pH (a), bicarbonate (HCO_3^-) (b), and urinary pH (c) with DCAD.
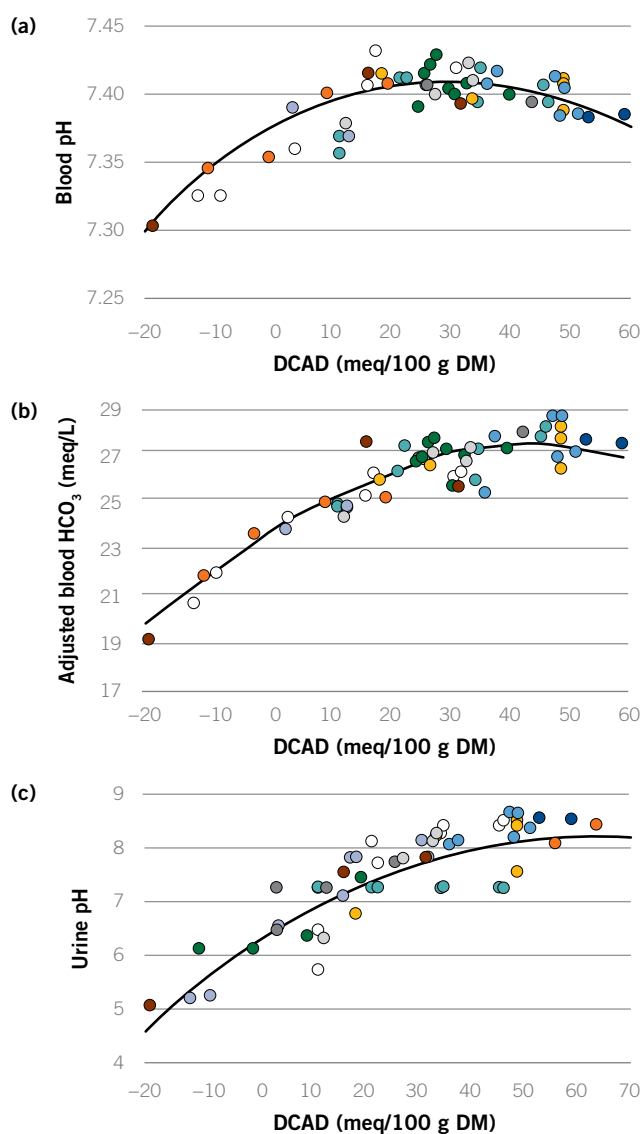
Source: Hu and Murphy, 2004.

THE CASE FOR MANIPULATING NEGATIVE DCAD IN PREPARTUM TRANSITION DAIRY 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 values 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 5 and Table 1. Giesy, et al. (1997) showed that when different DCAD levels were fed to cows that were challenged with an infusion of EDTA (ethylene diamine tetra-acetic acid) to remove calcium from blood, cows maintained their blood calcium better as the DCAD was reduced (Figure 5). In a trial with periparturient cows, Leclerc and Block (1989) showed a highly significant negative correlation between lower DCAD and concentration of blood calcium, which was strongest from 12 hours pre- to 12 hours postpartum (Table 1). Therefore, as DCAD is reduced prepartum, blood calcium concentration is maintained at a higher level around parturition.

FIGURE 5. Blood-ionized Ca response to four levels of DCAD following infusion with EDTA to mimic hypocalcemia.

$y = -.0068 x + 3.599$

Source: Giesy, et al., 1997.
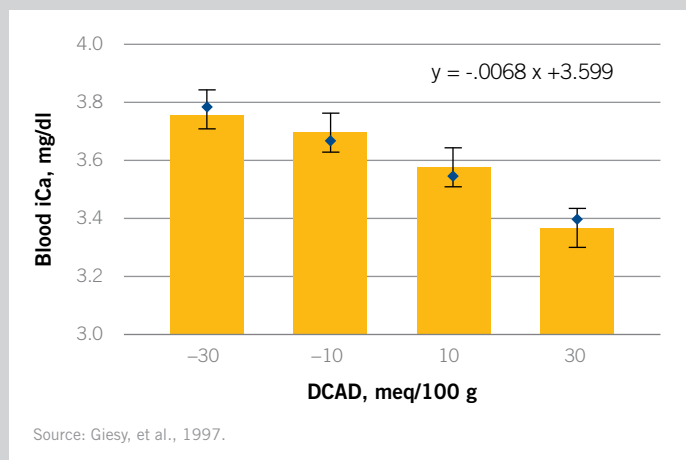
TABLE 1. Correlation between blood calcium concentration and DCAD from 48 hours prepartum to 36 hours postpartum.

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

Source: Leclerc and Block, 1989.

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., 1976). Feeding a ration with a 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. allowing 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 and Horst, 1991; Phillippo, et al., 1994). Low DCAD diets ($\leq$ -5 meq/100 g diet DM), through addition of anions, increase target tissue responsiveness to parathyroid hormone (Horst, et al., 1997).

The Effects of 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 5 to 7% (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; Oetzel, et al., 1988). Guard (1994) estimated the average cost per case of milk fever to be $334.

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 cows that had no clinical milk fever had a significant drop in blood calcium at parturition.

FIGURE 6. Plasma calcium concentrations (mean ± SEM) around the time of parturition in milk fever (n = 8) and nonmilk fever (n = 19) cows; d 0 = day of parturition.
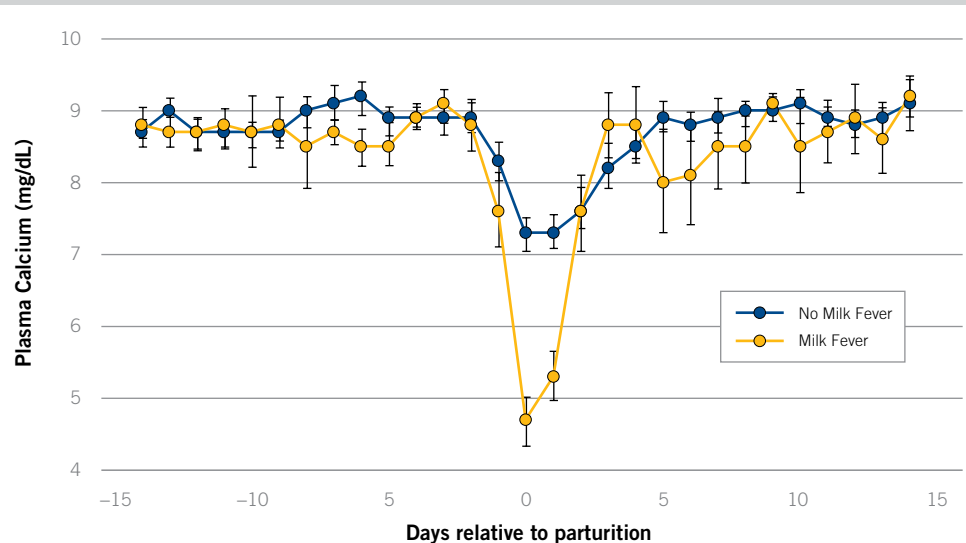
Source: Kimura, et al., 2006.

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 clinical and subclinical milk fever 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 they also found other dietary and management factors had independent contributions, including prepartum dietary Ca, Mg and P, 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 feeding a negative DCAD on blood calcium and postpartum milk production are also prevelant when higher-fiber, reduced-starch diets are fed (Siciliano-Jones, et al., 2008).

DCAD EFFECTS ON DMI AND MILK PRODUCTION

Decreasing DCAD in Prepartum Diets

The effects of decreasing DCAD to negative levels on prepartum dry matter intake (DMI) are equivocal. Horst, et al. (1994) reported that the addition of >300 meq of anions/kg diet may reduce DMI. 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 relationship 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 effects of negative DCAD on reducing 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 nonmineral-based anionic supplements has not shown the DMI reduction prepartum as happens with mineral salt supplementation (DeGroot, 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; Jordan 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; DeGroot, 2004; Penner, et al., 2008; Siciliano-Jones, et al., 2008). 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, 2004).

Finding Optimal Prepartum DCAD

Controlled experiments have not yet determined the optimal prepartum DCAD. The recommended target DCAD of −10 to −15 meq/100 g 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 conducted a DCAD forage testing program.

Both tables illustrate the large variation in ration DCAD and individual mineral levels. 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 DM would be more desirable to ensure that all cows always receive a negative or 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.

TABLE 2a. 2007-2009 Arm & Hammer Forage Testing Program[SM] results for the United States and Canada show wide DCAD variation.

Northeast

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Legume Haylage (n = 88)	38.60	-5.28	75.17	18.64
Grass Haylage (n = 10)	37.48	13.48	95.35	22.59
Balage (n = 18)	42.09	9.50	98.45	24.18
Corn Silage (n = 85)	20.94	-13.93	77.54	16.94
Small Grain Silage (n = 16)	39.18	14.40	76.27	17.99
Legume Hay (n = 40)	32.49	8.66	79.63	14.84
Grass Hay (n = 50)	29.53	0.61	56.28	14.20

Southeast

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Corn Silage (n = 7)	20.92	6.78	54.80	15.69
Small Grain Silage (n = 7)	44.99	25.73	87.51	24.18
Grass Hay (n = 2)	36.09	n/a	n/a	n/a

North Central

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Legume Haylage (n = 747)	49.54	-9.21	427.04	46.97
Grass Haylage (n = 10)	42.61	8.73	105.55	25.81
Balage (n = 29)	37.17	-2.06	78.31	19.86
Corn Silage (n = 376)	17.59	-2.62	100.00	14.01
Small Grain Silage (n = 74)	60.74	4.24	78.31	21.66
Legume Hay (n = 252)	49.42	-1.29	394.47	53.06
Grass Hay (n = 232)	35.48	-19.95	189.08	26.44
Straw (n = 41)	22.99	-3.25	70.57	14.43

TABLE 2a. continued.

South Central

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Corn Silage (n = 11)	46.10	-14.05	275.37	79.66
Small Grain Silage (n = 7)	27.14	17.56	37.88	7.89
Legume Hay (n = 3)	24.40	n/a	n/a	n/a

Northwest

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Corn Silage (n = 3)	18.56	n/a	n/a	n/a
Legume Hay (n = 16)	41.46	-0.49	77.65	24.07

Southwest

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Legume Haylage (n = 10)	36.88	13.37	63.65	16.57
Corn Silage (n = 21)	21.78	-2.27	53.73	17.70
Small Grain Silage (n = 13)	27.69	2.68	63.04	17.28
Legume Hay (n = 20)	33.98	8.54	75.71	17.37

Canada

Sample Type	Average DCAD	Minimum	Maximum	Standard Deviation
Legume Haylage (n = 15)	35.57	8.95	59.78	13.36
Corn Silage (n = 11)	36.94	12.35	44.29	10.47
Small Grain Silage (n = 32)	23.98	7.69	63.52	12.23
Legume Hay (n = 38)	30.25	-1.34	72.53	14.94
Grass Hay (n = 20)	29.30	-11.92	57.00	18.76

TABLE 2b. 2008 Arm & Hammer Forage Testing Program℠ results for North Central United States show varying mineral levels in each forage.

Sample Description	%Na	%K	%Cl	%S	DCAD
Legume Haylage≠					
Average (n = 166)	0.09	2.39	0.57	0.22	35.54
Minimum	0.00	0.82	0.02	0.07	-9.21
Maximum	0.82	3.88	1.60	0.35	82.62
Standard Deviation	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
Standard Deviation	0.06	0.38	0.15	0.03	8.53

≠ Individual Mineral Variation In Legume Haylages And Corn Silage.
2008. Data on file.

Monitoring DCAD

Urinary pH and DCAD are directly 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 effective. Theoretically, urinary pH less than 7.0 in individual cows 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 (6.0 to 6.4).

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

IMPLEMENTING BIO-CHLOR® IN PREPARTUM TRANSITION DIETS

BIO-CHLOR versus SoyChlor

Based on manufacturing processes, these products bring different advantages to a diet other than a negative DCAD.

The only published direct comparison (as of September 2009) was reported by Froetschel, et al., (2004). The study was designed to test if there were differences in efficacy between the two products for the acidification ability and their ability to maintain blood calcium when the cow is stressed with dramatic removal of blood calcium. The results are shown in Table 3 (for yearling steers simulating calcium depletion in cows). At the same DCAD, BIO-CHLOR® Rumen Fermentation Enhancer (Church & Dwight Co., Inc., Princeton, NJ) was numerically superior in maintaining DMI and blood calcium, but there were no statistical differences between the treatment means. The ability of both products to reduce urinary pH was almost identical in this trial.

The difference between these two products lies in their ability to deliver dietary protein, the amount of protein delivered and the form in which it is delivered.

TABLE 3. A comparison of the efficacy of equal DCAD from BIO-CHLOR and SoyChlor to maintain DMI and blood calcium and to reduce urinary pH in cows.

Item	SoyChlor	BIO-CHLOR
DMI (kg/d)	23.90	25.20
DMI % BW	2.09	2.25
Urine pH	6.62	6.61
Blood-ionized Ca during EDTA infusion, mg/dl (ability to maintain blood Ca during stress of Ca depletion)	5.94	6.89
Blood-ionized Ca as a percent of pre-EDTA, mg/dl (ability to maintain blood Ca during stress of Ca depletion as a percentage of the prestress levels)	62.60	66.10

Source: Froetschel, et al., 2004. Data on file.

SoyChlor® (West Central, Ralston, IA) is manufactured by treating various protein supplements and by-products with hydrochloric acid (HCl). The treatment of protein sources with HCl has a benefit of reducing protein degradability in the rumen, but it can also make protein unavailable. The current description for SoyChlor is 20% crude protein and a DCAD of −298 meq/100 g on a dry matter basis.

BIO-CHLOR is manufactured by taking corn fermentation liquors from the production of certain amino acids, applying these to a carrier and drying the mix in a patented drying process. The corn fermentation liquors contain bacterial residues and their nitrogen content, although mainly nonprotein nitrogen (NPN), is in the form of peptides (short chains of amino acids), free amino acids and nucleotides (DNA and RNA). The drying process makes these nitrogen sources more slowly degradable such that they can be used as precursors for bacterial growth as opposed to having the rumen microbes synthesize these nutrients. BIO-CHLOR has (dry basis) 51% crude protein and a DCAD of −309 meq/100 g DM basis. Therefore, both BIO-CHLOR and SoyChlor have equivalent DCAD contents, but they are different in their sources, types and levels of protein delivered.

BIO-CHLOR® is a registered trademark of Church & Dwight Co., Inc.

Stimulating Rumen Bacterial Protein Synthesis

By supplying the rumen microbes with bacterial fragments, peptides, nucleotides and free amino acids in a preformed state, the efficiency of bacterial protein production in the rumen increases an average of 15%. This increase in efficiency has been shown to be nutritionally important to the cow and consistent across many different diet formulations (Lean, et al., 2005). The advantage of stimulating bacterial protein synthesis in the rumen versus bypassing the rumen with dietary protein is that rumen microbes generally contain a higher-quality protein in terms of amino acid complement and have greater intestinal digestibility than bypass protein sources.

The ability of BIO-CHLOR® to stimulate rumen bacterial protein synthesis and increase the efficiency of its synthesis versus soybean meal (SBM) or SoyChlor was examined in a study (unpublished) conducted at the Rumen Fermentation Profiling Laboratory at the University of West Virginia under the direction or Dr. W. Hoover.

BIO-CHLOR Research and Applications

A trial was conducted using 12 continuous culture rumen fermenters. Three prepartum transition diets were formulated to be isonitrogenous based on SBM, SoyChlor or BIO-CHLOR as the protein supplement. Four replicated fermenters were created for each diet. The effects of these supplements on the total diet are highlighted in Tables 4a and 4b.

Table 4a shows that organic matter and neutral detergent fiber (NDF) digestibility in the rumen for the total diet was equivalent when BIO-CHLOR replaced SBM. However, the SoyChlor diet had a reduced ($P < 0.05$) digestibility for both organic matter and NDF. While there were no differences between diets for the digestion of the nonstructural carbohydrates, the differences in NDF digestibility were sufficient for the SoyChlor diet to show a significant ($P < 0.05$) reduction in total carbohydrates digested.

TABLE 4a. Nutrient digestibilities in continuous culture rumen fermenters for isonitrogenous diets.

Item	SBM	BIO-CHLOR	SoyChlor	P =
Organic Matter, %	55.7[a,b]	57.7[a]	50.7[b]	0.035
Neutral Detergent Fiber, %	41.5[a]	41.3[a]	33.6[b]	0.040
Nonstructural Carbohydrate, %	85.8	86.3	84.5	0.638
Total Carbohydrate Digested, g/d	26.0[a]	26.2[a]	21.8[b]	0.0001

[a, b] Means in the same row with different superscripts differ ($P < .05$).
Source: Rumen Fermentation Profiling Laboratory, Morgantown, WV. Data on file.

Table 4b shows the results of rumen protein partitioning. The SBM diet had a numerically, but not significantly, higher digestibility for crude protein than SoyChlor, and both of these were much lower ($P < 0.05$) than the crude protein digestibility in the diet when BIO-CHLOR® was present. The efficiency of bacterial protein production was measured as a percentage of the total carbohydrates digested. Although there were no statistically significant differences in the measurement between BIO-CHLOR and SoyChlor, the BIO-CHLOR diet produced more ($P < 0.05$) bacterial protein per day than the SoyChlor diet. Nutritionally, bacterial protein is of higher quality and intestinal digestibility than rumen undegradable protein. Therefore, the BIO-CHLOR diet would deliver more metabolizable protein with a better amino acid balance than either SoyChlor or SBM.

TABLE 4b. Nutrient digestibility and bacterial protein production.

Item	SBM	BIO-CHLOR	SoyChlor	*P =*
Crude Protein Digested, %	71.7[b]	87.9[a]	67.8[b]	0.0001
Microbial N, g/day	1.11[b]	1.25[a]	1.10[b]	0.005
Total Carbohydrate Digested, g/d	26.0[a]	26.2[a]	21.8[b]	0.0001
Efficiency of Bacterial Protein Production (% of total carbohydrates digested)	42.7[b]	47.6[a]	50.5[a]	0.006

a, b Means in the same row with different superscripts differ ($P < .05$) Results measured as microbial nitrogen produced per unit of carbohydrate digested in continuous culture rumen fermenters for isonitrogenous diets where the protein was SBM, BIO-CHLOR, or SoyChlor.
Source: Rumen Fermentation Profiling Laboratory, Morgantown, WV. Data on file.

BIO-CHLOR, therefore, has the advantage over other anionic supplements of delivering the desired anions in a palatable form and stimulating microbial protein production to a point that no other anionic supplement is capable of achieving.

The ability of BIO-CHLOR to benefit cows beyond the DCAD effect was demonstrated by DeGroot (2004) in his Ph.D. thesis. In this trial, prepartum cows were given one of four isonitrogenous, isoenergetic diets that differed only in DCAD (n = 25 per treatment). 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 DCAD levels 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® Rumen Fermentation Enhancer (Church & Dwight Co., Inc., Princeton, NJ) and anionic mineral salts. This later treatment needs further explanation, provided in the following overview.

Rumen Fermentation Enhancers (RFE)

FERMENTEN is a supplement that has been shown to have the same impact on rumen fermentation as BIO-CHLOR (DeGroot, 2004), 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 plus anionic salts should outperform the anionic salt treatment and there should be no difference between the BIO-CHLOR and FERMENTEN plus anionic salts treatments.

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RFE Research and Applications

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 7a shows the temporal pattern of postpartum milk production for the four prepartum DCAD treatments. Milk production was significantly improved in all cows fed negative DCAD treatments over the controls. Even more striking is the start-up milk production; cows fed BIO-CHLOR® or FERMENTEN® plus anionic salts produced upwards of 20 lbs. more milk at start-up than the control cows or those fed anionic salts. Figure 7b 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 plus anionic salts resulted in approximately five lbs. more milk per day as compared to feeding anionic salts alone (see Figure 7c). Finally, as shown in Figure 7a, there were few differences in postpartum performance of cows fed prepartum BIO-CHLOR, or FERMENTEN plus anionic salts.

FIGURE 7a. Postpartum milk production by treatment group.

- BIO-CHLOR ave. 91.0 (DCAD −10)
- Control ave. 70.7 (DCAD 20)
- FERMENTEN ave. 90.1 (DCAD −10)
- Salts ave. 85.3 (DCAD −10)
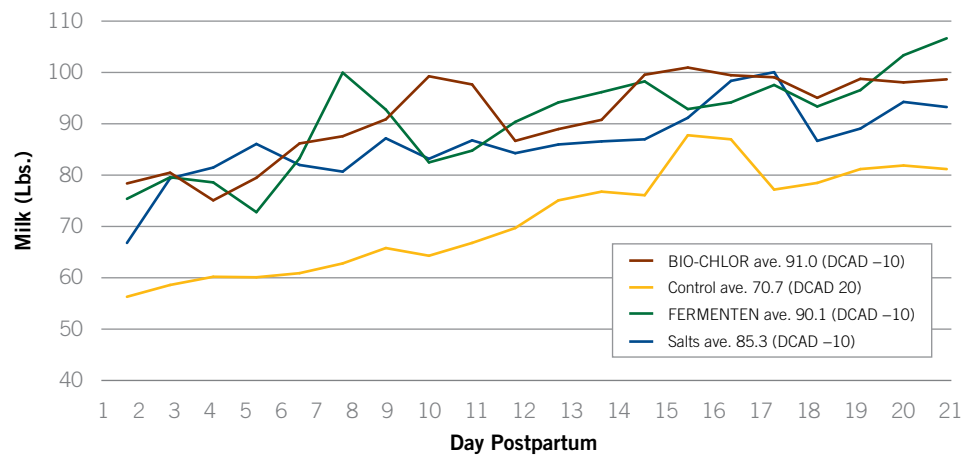
Source: DeGroot, 2004.

FIGURE 7b. Postpartum milk production: negative vs. positive ration DCAD.

- FERMENTEN, BIO-CHLOR and Salts ave. 88.8 (DCAD −10)
- Control ave. 70.7 (DCAD 20)
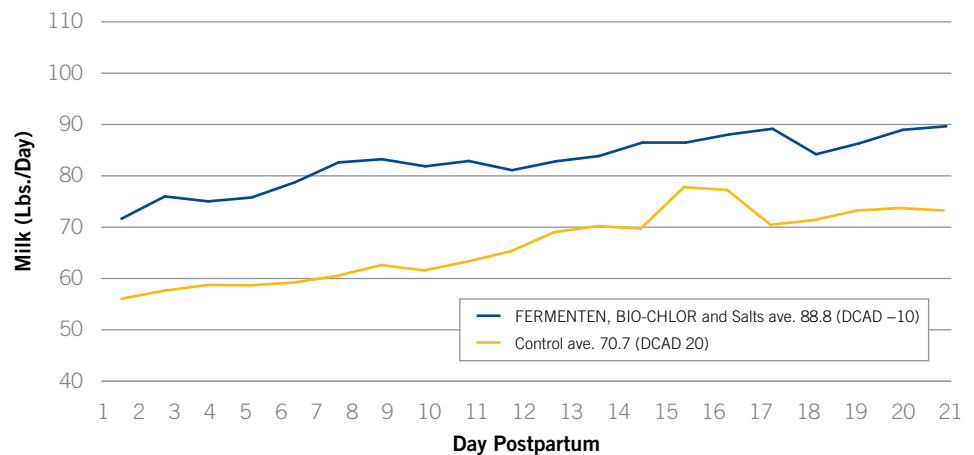
Source: DeGroot, 2004.

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FIGURE 7c . Postpartum milk production: rumen fermentation enhancer advantage.
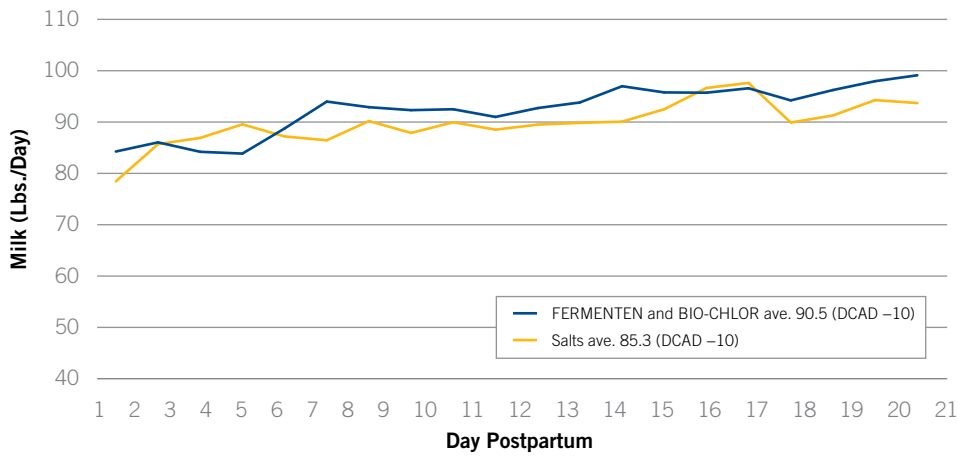
Source: DeGroot, 2004.

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 are likely to have better protein status at calving when fed BIO-CHLOR.

METHODS FOR ALTERING DCAD IN PREPARTUM DIETS

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 has 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 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 to Help Reduce Prepartum DCAD

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

1. Forages (purchased or produced on-farm)
   - ◆ Forages alone will likely not be able to reduce DCAD to acceptable negative values.
   - ◆ Careful selection of forages that are low in dietary K can be used to reduce DCAD so 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 (i.e., the chloride and sulfate salts of Ca, Mg, 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 200 meq/100 g DM of anions have 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 200 meq/100 g DM of anions have to be added to the diet.
   - ◆ The two major products available with published scientific literature available are BIO-CHLOR® and SoyChlor.

How the DCAD Concept Can Be Applied to Lactating Cows

Any work done by an animal requires additional respiration by cells. Figure 8 shows a diagram depicting the end results of respiration. This includes the burning of fuels, the consumption of oxygen, the release of carbon dioxide (CO_2) and the production of acid (hydrogen – (H^+)). This acid accumulation occurs in part from the CO_2 production and its combining with water in blood to form carbonic acid (Figure 3), and also from the production of acids as an end product of respiration and the burning of metabolic fuels (i.e., lactic acid).

FIGURE 8. Diagrammatic representation of how respiration (work) of cells contributes acid load (H+) and carbon dioxide (CO_2) to blood.
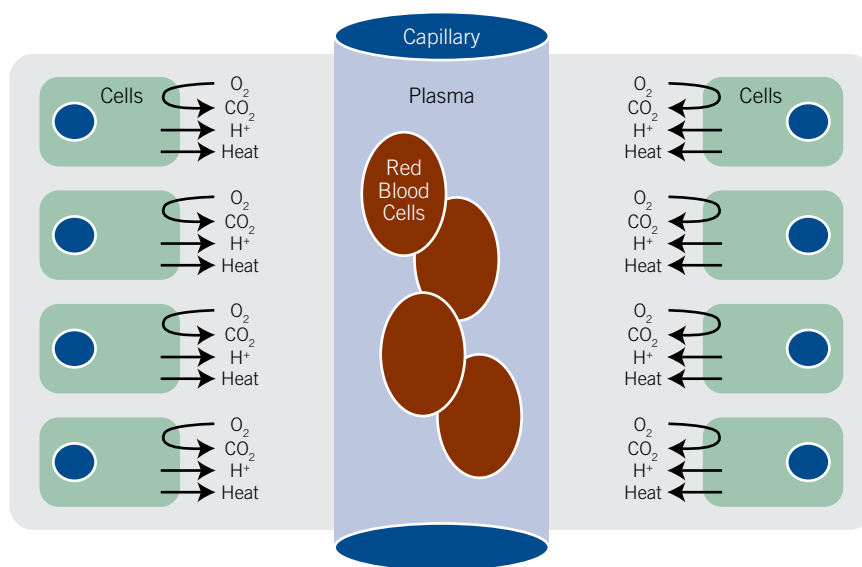
The more "work" done by the animal, the more CO_2 and acid is pumped out into the circulation for disposal. This work can be in the form of physical exercise, and also in the form of physiological and metabolic work such as tissue growth and milk production. In fact, many textbooks in lactation biology equate the modern dairy cow to the marathon runner in terms of metabolic work performed.

If these acids and CO_2 are allowed to accumulate, the muscle or mammary cells performing the work slow down their processes or stop functioning. The best example of this that most people can relate to is when prolonged physical exercise leads to muscle fatigue requiring a "rest period" before that activity can be resumed. This rest period is needed to remove the acid accumulation and restore tissue oxygen levels.

The lactating cow produces a tremendous quantity of metabolic acids. Obviously mammary cells produce these acids through the synthesis of milk, milk fat and milk protein. However, there are other acids that enter the system. Fatty acids that are mobilized from fat stores, volatile fatty acids that are absorbed from the rumen, and ketones all contribute to the acid load. In fact, when animals stop eating and milking due to ketosis, it is really the acidosis from the ketone bodies that is causing the symptoms (technically termed "ketoacidosis").

The Effects of Increasing DCAD During Lactation

As production increases, so does the acid load in the cow. With insufficient blood-buffering capacity the cow will not be allowed to fully express her production potential and will reduce her DMI and production accordingly. It makes sense, therefore, that cows require more blood-buffering capacity as DMI and productivity increase.

Several meta-analyses have been published over the years showing a direct and positive relationship between positive DCAD, blood pH and blood bicarbonate (Sanchez, et al., 1994; Hu and Murphy, 2004; Hu, et al., 2007). All of these studies included published trials with cows in mid-to-late lactation. The optimal DCAD in these meta-analyses for maximizing DMI and blood bicarbonate was in the range of 37 to 42 meq/100 g DM using the three-mineral equation (Na + K – Cl). The three-mineral equation was used because few of the trials reported S content of the diets. This would indicate that if S were included, the optimal four-mineral DCAD equation would be more in the range of 30 to 35 meq/100 g DM. The two meta-analyses that reported milk production (Sanchez, et al., 1994; Hu and Murphy, 2004) also found that positive DCAD was positively correlated with fat-corrected milk production (FCM) within the same optimal DCAD range as DMI and blood bicarbonate (Figures 4 and 9).

FIGURE 9. Relationship between DCAD and 4% FCM in a meta-analysis.
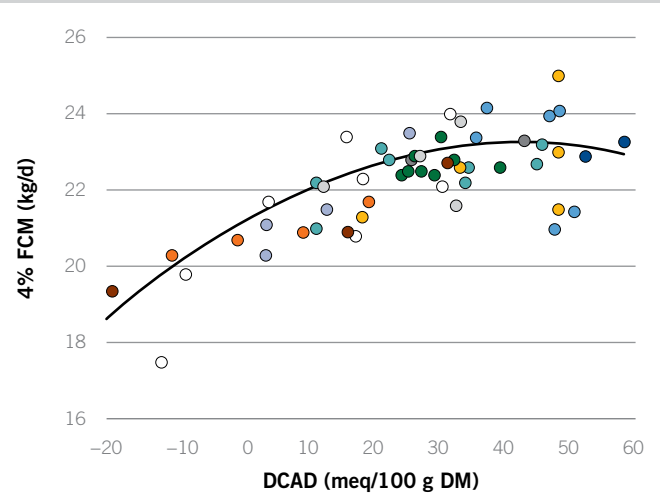
Source: Hu and Murphy, 2004.

The example of the meta-analytical results in Figure 9 match those of Sanchez, et al. (1994) and show that for cows at moderate milk production levels DCAD can be optimized at 37 to 42 meq/100 g DM using the three-mineral equation (30 to 35 meq/100g DM with the four-mineral equation). Obviously missing from both the data sets were cows producing high quantities of milk and components.

DCAD theory, as cited above, would indicate that if cows are capable of producing high volumes of milk and components, the optimal DCAD should be much higher than these meta-analyses would indicate. The need for more blood-buffering capacity would be greater in these cows.

TABLE 5. Milk production responses from four field trials where DCAD was increased.

Early Lactation Field Trials		Trial Description				
Trial		1 - Winter	2 - Winter	3 - Winter	4 - Spring	
		Florida	Florida	California	Idaho	
Item	Units	Cl Removed	K Added	K Added	K Added	Average
DCAD for Controls	meq/100 g	18.9	18	38	33	28.78
DCAD for Treatments	meq/100 g	25.1	26	43	39	35.02
% K for Controls	K, % DM	1.38%	1.20%	1.52%	1.50%	1.45%
% K Treatments	K, % DM	1.33%	1.50%	1.80%	1.70%	1.63%
Milk Response	lbs./cow/day	3.0	5.4	2.4	1.5	2.1
Fat Response	lbs./cow/day	NA	0.14	0.04	0.01	0.15
3.5% FCM Response	lbs./cow/day	3.0	3.3	2.0	1.9	3.2

Source: Sanchez, et al., 2002.

One of the first field trial results examining the effects on lactating cows of increasing DCAD was reported by Sanchez, et al. (2002). Those trials were conducted in nonheat stress periods of the year and DCAD was manipulated upwards by either reducing dietary Cl or by increasing dietary K. Irrespective of the methodology employed to increase DCAD or the initial DCAD of the control groups, all trial sites showed improvements in milk yield and FCM (Table 5).

Since the publication of the meta-analyses by Hu and Murphy (2004), two additional research trials were published utilizing higher-producing cows earlier in lactation.

Positive DCAD Research and Applications

A trial reported by Hu, et al. (2007) investigated the effects of increasing DCAD (four-mineral equation) at two different dietary crude protein (CP) concentrations. One of the theories behind this design was to further investigate the relationship between DCAD and protein utilization. There are other publications that have shown that increasing DCAD can improve milk protein production (Wildman, et al., 2007) and that metabolic acidosis can negatively affect protein metabolism (Cai and Zimmerman, 1995; Welbourne, et al., 1986; May, et al., 1986).

The results of this study by Hu, et al. (2007) are shown in Table 6. The effects of DCAD as a main factor in this trial were positive, resulting in higher DMI and all measures of milk production performance ($P < 0.05$). The effects of DCAD in this trial were also found to be linear—as DCAD increased from –3 to 22 and to 47 meq/100 g DM, all parameters of performance increased in a linear manner. Dietary protein in this trial had no effect as a main factor on any performance parameters except for the effect of increasing milk urea nitrogen (MUN) with higher dietary crude protein.

Although there were no interactions between DCAD and protein, there is an indication that DCAD may play a role in nitrogen metabolism in that the MUN across diets was reduced with higher levels of DCAD ($P < 0.05$). Also interesting in this trial was the fairly high response of milk fat (percentage and pounds per day) to DCAD. This subject will be addressed later but is consistent with other trials such as the ones shown in Table 5. Milk protein percentage significantly improved by increasing DCAD ($P < 0.05$), but amount of milk protein produced per day only tended ($P = 0.07$) to be increased by DCAD.

TABLE 6. Comparison of three diets with varying DCAD and CP levels.

Variable	DCAD = –3 16% CP	DCAD = –3 19% CP	DCAD = 22 16% CP	DCAD = 22 19% CP	DCAD = 47 16% CP	DCAD = 47 19% CP	SE	A	B	C	D	E
DMI, kg/d	24.9	23.9	26.1	25.7	27.0	28.2	1.9	NS[2]	<0.01	NS	NS	NS
BW, kg	678	675	680	677	682	678	12	NS	NS	NS	NS	NS
Milk												
Yield, kg/d	35.3	36.7	36.4	35.8	36.1	36.6	2.3	NS	NS	NS	NS	NS
4% FCM, kg/d	31.1	30.3	32.4	33.1	33.9	34.5	2.7	NS	0.01	NS	NS	NS
Fat, %	3.12	2.85	3.27	3.46	3.57	3.62	0.50	NS	0.02	NS	NS	NS
Fat, kg/d	1.12	1.04	1.19	1.25	1.29	1.32	0.17	NS	0.01	NS	NS	NS
Protein, %	3.12	3.09	3.20	3.16	3.24	3.24	0.09	NS	<0.01	NS	NS	NS
Protein, kg/d	1.10	1.11	1.15	1.11	1.15	1.18	0.07	NS	0.07	NS	NS	NS
Lactose, %	4.79	4.82	4.78	4.79	4.90	4.90	0.09	NS	0.03	0.05	NS	NS
Lactose, kg/d	1.69	1.76	1.74	1.72	1.78	1.80	0.12	NS	NS	NS	NS	NS
SNF, %	8.58	9.01	9.11	8.98	9.09	8.82	0.13	NS	<0.01	NS	NS	NS
SNF, kg/d	3.18	3.21	3.22	3.17	3.27	3.31	0.21	NS	NS	NS	NS	NS
Urea, mg of N/dl	17.0	21.0	13.7	21.5	13.7	19.5	2.24	<0.01	0.02	NS	NS	0.08
SCC, x 1000/ml	46	34	37	36	38	31	19	NS	NS	NS	NS	NS

The top header spans: DCAD = –3 (16% CP, 19% CP); DCAD = 22 (16% CP, 19% CP); DCAD = 47 (16% CP, 19% CP); Orthogonal Contrast[1] (SE, A, B, C, D, E).

[1]Contrast A: 16% CP vs. 19% CP; Contrast B: linear effect of DCAD; Contrast C: quadratic effect of DCAD; Contrast D: interaction between CP concentration and linear effect of DCAD; Contrast E: interaction between CP concentration and quadratic effect of DCAD.
[2]NS: nonsignificant ($P > 0.10$).
Source: Hu, et al., 2007.

IMPLEMENTING DCAD PLUS® IN LACTATING DIETS

Increasing Dietary Potassium

In a more recent trial, White, et al. (2008) utilized cows in their first 12 weeks of lactation to investigate the effect of increasing DCAD. Table 7 shows the nutrient composition of the experimental diets used in this trial. The control diet had the addition of DCAD Plus® Feed Grade Potassium Carbonate (Church & Dwight Co., Inc., Princeton, NJ), to increase dietary K from 1.2% to 2.0% of the total DM and to increase DCAD from 22 to 44 meq/100 g DM. All other nutrients were similar between the two diets.

TABLE 7. Nutrient content of experimental diet fed to cows for their first 12 weeks of lactation.

	CONTROL	DCAD Plus
Nutrient	**Composition (dry basis)**	
Dry Matter (%)	60.1	59.4
Crude Protein (%)	16.1	16.1
Soluble Protein (% of CP)	34.1	31.1
ADF (%)	19.8	19.3
NDF (%)	35.0	34.7
Calcium (%)	0.76	0.76
Phosphorus (%)	0.4	0.39
Magnesium (%)	0.4	0.4

	CONTROL	DCAD Plus
Nutrient	**Composition (dry basis)**	
Potassium (%)	1.21	2.05
Sulfur (%)	0.26	0.25
Sodium (%)	0.51	0.51
Chloride (%)	0.54	0.54
DCAD (meq/100 g)	21.52	43.68

Source: White, et al., 2008.

DCAD and Milk Fat Relationship

We have seen in almost all trials (extensive field trials and intensive controlled trials) that milk fat percentage and production consistently increase when higher DCAD is fed to cows. While the phenomenon is real it is difficult to explain from a physiological or biochemical standpoint. It does not appear that higher DCAD corrects an existing fat depression or low fat test, but it does appear that normal fat tests become elevated. There is no relationship found in the literature that directly connects milk fat production to either DCAD or K. Therefore, the following discussion is highly speculative.

Some possibilities for the increase in milk fat with increasing DCAD can be:

1. Increasing the cation load (Na and K) will increase blood concentration of these cations, thereby increasing recycling of these to the rumen via the bicarbonate forms of these ($NaHCO_3$ and $KHCO_3$). This can provide more rumen buffering and decrease the amount of trans-fatty acid production via biohydrogenation (Bauman, et al., 2003).
    a. The problem with this theory is that DCAD can increase fat production by reducing anions and increasing cations in the diet. Reducing anions will increase DCAD, but will not increase the Na and K load in the blood.
    b. Another problem with this theory is that, if true, sodium buffers (sodium bi- and sesquicarbonates) would cause increased fat tests alone other than increasing an already low fat test. This has not been shown to happen in the literature.

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2. Another theory could be that the creation of a highly buffered intra- and extra-cellular environment in the mammocyte (milk secreting cells of the mammary gland) causes either fatty acid uptake from the blood and/or fatty acid synthesis in the mammary to occur to a greater extent or more efficiently.

   a. Although a bit more farfetched, this theory has some merit as we know the mammary gland regulates the amount of acid (H) secreted in milk. Reducing the H load in the mammary may allow for greater synthesis and secretion.

   b. The milk fat in the trials was not analyzed for fatty acid profile. In future trials this should be done to determine if trans-fatty acid levels are altered and if the increase in fat is from preformed long-chain fatty acids or from the synthesized short chain fatty acids. These analyses would give us an indication of rumen involvement and if we are affecting fatty acid uptake by the mammary gland, fatty acid synthesis by the mammary gland, or both.

DCAD Plus® Research and Applications

Table 8 shows the performance parameters and statistical analyses for this trial. With the exception of milk protein, all performance parameters were significantly and positively affected by increasing DCAD using DCAD Plus®. Body weight was not affected by treatment, and although DMI was numerically increased by 1.32 lbs/day (600 g/day), this was not significantly different from cows offered the control diet.

TABLE 8. Body weight (BW), DMI, and milk and milk component production when low and high DCAD diets were fed to cows through the first 12 weeks of lactation.

Item	Treatment		P < Treatment	P < Week	P < Treatment x Week	Linear	Quadratic
	Control	DCAD Plus					
BW, kg	669.0 ± 20.0	674.0 ± 20	0.49	0.002	0.93	0.18	0.28
DMI, kg/d	26.2 ± 0.9	26.8 ± 0.9	0.20	<0.0001	0.09	0.26	0.004
Milk, kg/d	39.3 ± 1.4	40.8 ± 1.4	0.01	0.01	0.46	0.48	0.01
ECM, kg/d	41.3 ± 1.4	44.3 ± 1.5	0.00	0.24	0.72	0.55	0.45
3.5% FCM, kg/d	42.2 ± 1.6	46.1 ± 1.6	<0.0001	0.09	0.70	0.61	0.51
Fat, kg/d	1.55 ± 0.06	1.75 ± 0.06	<0.0001	0.03	0.37	0.50	0.75
True protein, kg/d	1.16 ± 0.03	1.14 ± 0.03	0.38	0.12	0.78	0.06	0.06
Milk Composition							
Fat, %	3.96 ± 0.09	4.31 ± 0.09	<0.0001	<0.0001	0.27	0.47	0.42
True Protein, %	2.97 ± 0.04	2.79 ± 0.04	<0.0001	<0.0001	0.91	0.36	0.09
Casein, %	2.42 ± 0.04	2.30 ± 0.04	<0.001	<0.001	0.57	0.49	0.01
Lactose, %	4.85 ± 0.05	4.92 ± 0.05	0.04	<0.0001	0.71	0.06	0.35
Solids, %	12.60 ± 0.14	12.79 ± 0.14	0.02	<0.0001	0.38	0.32	0.69
SNF, %	8.32 ± 0.08	8.18 ± 0.08	<0.0001	0.0001	0.40	0.03	0.04
MUN, mg/d	10.38 ± 0.25	10.62 ± 0.25	0.21	<0.0001	0.30	0.15	0.62
SCC, 1,000/ml	230.1 ± 113.7	265.4 ± 113.4	0.83	0.40	0.63	0.12	0.44
Feed Efficiency							
Kg milk/kg DMI	1.53	1.57	0.09	<0.0001	0.63	0.45	0.12
Kg FCM/kg DMI	1.61	1.72	<.0001	<0.0001	0.72	0.62	0.26
Kg ECM/kg DMI	1.58	1.65	<.001	<0.0001	0.68	0.52	0.31

Source: White, et al., 2008.

Milk protein production was not significantly affected by treatment, but due to the significant increase in milk yield the percentage of true protein was lower in the DCAD Plus® group. This result is different from the trial reported in Table 6 (Hu, et al., 2007). The explanation for the difference may lie in the way diets were formulated for metabolizable protein. In the study by Hu, et al. (2007) diets were formulated on the CPM Dairy Model for cows producing 40 kg/day of 3.5% FCM. Because cows actually produced less than 39 kg/day of 4% FCM all cows had more than sufficient metabolizable protein to allow expression for an increase in milk protein. In the trial reported in Table 8 the same CPM model was used and diets were formulated for an average of 45 kg/day of 3.5% FCM. Cows fed DCAD Plus produced an average of 46 kg/day of 3.5% FCM during the 12 weeks. Therefore, cows were producing significantly more milk and milk protein than supported by the formulation. Cows fed DCAD Plus could only produce the same amount of total milk protein as the control group because they had no excess protein in the diet to express a higher production. This led to a similar amount of milk protein production, but the increased milk volume reduced the percentage of protein in the milk.

Other interesting observations in Table 8 are the milk production efficiency values. While gross efficiency (kg milk/kg DMI) only tended ($P = 0.09$) to be improved with higher DCAD, the efficiencies based on FCM and energy-corrected milk (ECM) were significantly ($P < 0.001$) improved with higher DCAD. There was a 7% increase in FCM efficiency and 4.5% increase in ECM efficiency shown in this trial by simply increasing DCAD. This indicates that the utilization of nutrients improved in an environment that was well-buffered and/or not stressed by a high acid load.

The temporal patterns for FCM production and DMI are shown in Figures 10a and 10b. Cows began their lactation at very similar FCM production levels, but quickly separated according to treatment with the high DCAD group outperforming control cows consistently throughout the trial. Although DMI was not significantly affected by DCAD, Figure 10b does show a strong tendency for the cows fed the higher DCAD to have higher DMI during most of the trial.

FIGURE 10a, b. Temporal pattern of 3.5% FCM production (a) and DMI (b) when cows were fed a control diet with a DCAD of 25 meq/100 g DM and a treatment diet with a DCAD of 42 meq/100 g DM (DCAD Plus).
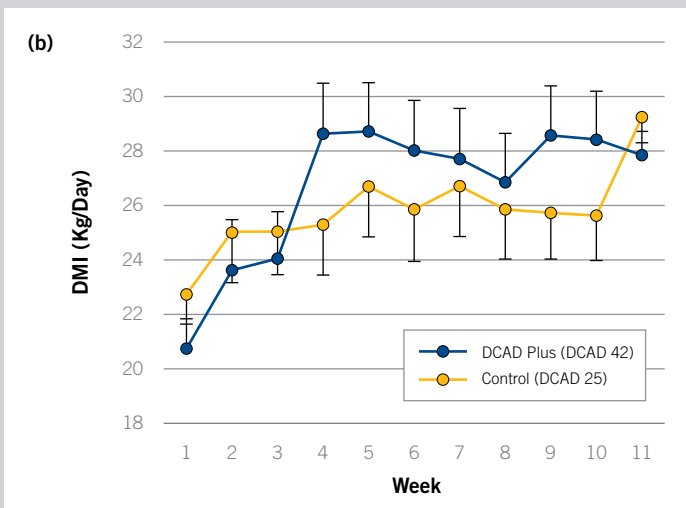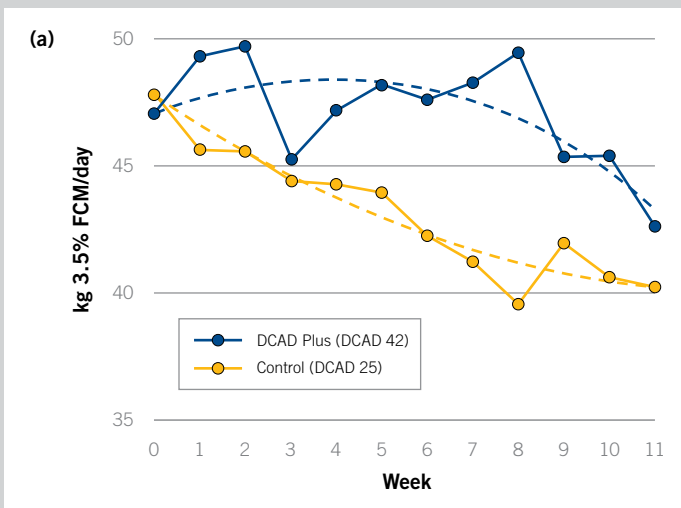
Source: White, et al., 2008.

THE ROLE OF POTASSIUM AS A NUTRIENT

The DCAD theory assumes that none of the minerals involved are either deficient or toxic in concentration in the diet. Since each mineral has its individual role in metabolism outside of the DCAD concept we still need to be certain that rations are formulated with sufficient amounts of the individual minerals and that they are not in extreme excess of requirement.

Mineral Balance Trials

We have already seen trials reported above where increasing DCAD by increasing K, increasing K + Na and by reducing Cl will improve production performance. However, there are indications in the literature that the current recommendation for K required by dairy cows may be insufficient. The NRC (2001) nutrient requirements for K will vary with milk production as milk is an important excretory route for K. However, few rations are balanced by NRC with more than 1.2% dietary K.

Silanikove, et al. (1997) conducted a mineral balance trial with cows during the two-week prepartum and postpartum periods and again after seven weeks of lactation. Diets were formulated for slightly higher than NRC requirements for minerals, and milk production was 39 to 41 kg/day in the postpartum measurement periods (two weeks and seven weeks postpartum, respectively).

TABLE 9. The balance of Na, K, and Cl (milliequivalents per day) in six cows 2 weeks before calving (Period 1), 2 weeks postpartum (Period 2), and 7 weeks postpartum (Period 3).

Period	Intake			Output			
	Feed	Water	Total	Milk	Urine	Feces	Apparent Retention
Na+							
1	1866[a]	109[a]	1975[a]	* * *	1834[a]	377[a]	-236
2	1786[a]	262[b]	2048[a]	696	1015[b]	458[b]	-122
3	2579[b]	320[b]	2899[b]	750	1357[ab]	730[b]	61
SEM	111	17	120	27	159	81	104
K+							
1	2693[a]	0.0	2693[a]	* * *	2583[a]	397[a]	-288
2	3219[a]	0.0	3219[a]	1746	1796[b]	611[a]	-925*
3	4763[b]	0.0	4763[b]	1847	2176[ab]	1201[b]	-461
SEM	176	0.0	176	114	170	85	179
Cl−							
1	2880[ab]	201[a]	3081[a]	* * *	2319[a]	470[a]	292[a]*
2	2620[b]	484[b]	3104[a]	1252	1056[b]	625[a]	171[a]
3	3673[a]	591[b]	4264[b]	1396	1112[ab]	920[b]	836[b]*
SEM	278	33	299	98	234	76	111

[a,b]Within columns and ions, means with different superscripts differ ($P < 0.05$).
*Significantly different from 0 by Student's t test analysis.
Source: Silanikove, et al., 1997.

The results shown in Table 9 indicate the following:

1. Cows tend to be in a negative Na balance in the transition and postfresh period, but re-establish positive Na balance by the seventh week of lactation. The negative balance is only a trend because the standard error associated with the balance is almost as large as the negative balance itself.
2. Cows are always in positive chloride (Cl) balance and although a goal is to minimize its dietary concentration to increase DCAD, it is unlikely that cows will be deficient or in negative balance for Cl.
3. Cows were in negative K balance for the duration of the trial in spite of meeting the NRC requirements. This negative K balance was similar in magnitude to the Na balance for the prepartum period but was eight times greater through the period immediately after calving. Even by seven weeks postpartum cows were still in negative K balance.

In the trial described earlier by White, et al. (2008), increasing DCAD from 22 to 44 by increasing only dietary K from 1.2% to 2.0% of the DM had a positive effect on production performance. Those researchers also conducted a K-balance study (unpublished results at the time of writing this publication). Figure 11 shows the K-balance data for individual cows at 2, 3, 4, 5, 6 and 7 weeks of lactation. The results show that there are cows that are near zero or are in negative K balance even when the dietary levels are well within NRC guidelines (at 1.2% DM in this case).

FIGURE 11. K Retention of cows fed diets with a low DCAD (control) or a high DCAD (DCAD Plus®) at weeks 2, 3, 4, 5, 6 and 7 of lactation.
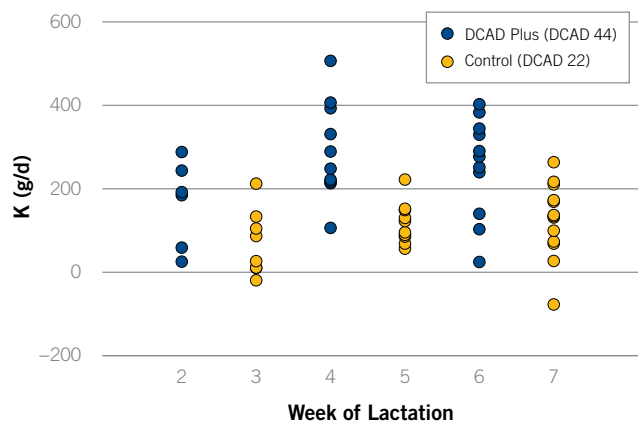
Source: White, et al., 2008.

Both the trial by White, et al. (2008) and Silanikove, et al. (1997) were conducted in nonheat-stress conditions. The ramifications of these results are that our description of K required by the cow is an underestimation. Even without considering DCAD, these trials indicate that a K concentration of 1.4% of the DM may be required to truly cover the K needs in an entire herd of high-producing cows.

The Role of DCAD During Heat Stress

This subject will be reviewed without great detail as there is an excellent review authored by West (1999).

Heat stress imposes some unique metabolic conditions that will require elevated DCAD and dietary K specifically. One of these conditions is the physical and physiological reaction of the cow to heat stress. In order to cool herself she increases respiration by panting. In so doing she is "blowing off" CO_2 from the blood, creating a CO_2 deficit inducing a metabolic acidosis and respiratory alkalosis. In order to replace the needed CO_2 an increase in DCAD will increase blood bicarbonate capable of forming additional CO_2.

The second condition is that the loss of K through excess saliva, increased urination and increased sweat all put more demands on the cow and her diet to replace the lost K.

As a result of the above it is recommended to increase K concentration in the diet to as much as 1.8 to 2% of the DM and DCAD by an extra 5 to 10 meq/100 g DM.

Reaching an Ideal Sodium:Potassium Ratio

There is little evidence in the literature that would support the concept of an ideal Na-to-K ratio for dairy cattle. Thus far the published work would suggest that DCAD is more important than the ratio of any minerals in the equation (Hu and Kung, 2009; Tucker, et al., 1988; West, et al., 1992; Wildman, et al., 2007).

We must keep in mind, however, that there are minimum and maximum allowable levels for these minerals (NRC, 2001; 1980) that must be respected. Within those tolerances, there appears to be little support for an ideal ratio. This will be discussed again below in deciding how to balance a ration for DCAD.

The Role of Magnesium

There is a well-known nutritional interaction between K and Mg in that the ratio of dietary K to Mg should be in the range of 4:1 (Georgievskii, 1981). The interaction is in the absorption of these nutrients in that excess K can impair the absorption of Mg. When the ratio is about 4:1 this competitive absorption is minimized.

Since dietary K should exceed 1.4% of the DM in lactating cows and since dietary Mg plays a critical role prepartum as well, the general recommendation is to fix dietary Mg at about 0.4% of the total ration DM irrespective of stage of lactation. Higher levels than this might be desirable but impractical as the typical sources of Mg (MgO) can cause diarrhea when raised to 0.45% ration DM. The use of $MgSO_4$ is contraindicated in lactating rations because of the DCAD-lowering effect of the sulfur.

METHODS FOR ALTERING DCAD IN LACTATING DIETS

It appears from the discussions above that the optimal range of DCAD in lactating cow diets is dependent upon level of production, with higher production requiring higher DCAD. Since there are few trials that have tried to identify an ideal DCAD for animals we will use the existing data to make some general recommendations:

- ◆ Holstein cows immediately postpartum and those cows producing in excess of 80 lbs. of 3.5% FCM should have a DCAD of 35 to 45 meq/100 g DM.
- ◆ Holstein cows producing between 60 and 79 lbs. of 3.5% FCM should target a DCAD between 30 and 35 meq/100 g DM.
- ◆ Holstein cows producing less than 59 lbs. of 3.5% FCM should target a DCAD between 25 and 30 meq/100 g DM.

Now that the DCAD targets are set, there is a five-step procedure to achieving these levels:

1. Use a source of Mg that does not contain chlorides or sulfates to achieve a dietary Mg level of 0.4%.
2. Remove as many chloride and sulfate salts as possible from the diet. This step alone will increase DCAD.
3. Increase dietary Na to 0.8% of the dietary DM using a sodium buffer (sodium bicarbonate or sodium sesquicarbonate). The maximum allowable (recommended) dietary sodium level has been set at approximately 0.8% of the total DM (NRC 1980).
4. Verify ration DCAD through forage testing using wet chemistry macromineral analysis.
5. Feed DCAD Plus® to increase dietary K to at least 1.6% of the ration DM to achieve the recommended DCAD levels. The choice of K sources is limited to potassium carbonate as other sources deliver CI or S to the diet, reducing DCAD and increasing ration costs. Feeding DCAD Plus allows you to successfully boost ration DCAD without the counterproductive effects of anions (i.e., KCl), which could lower DCAD and defeat the goal of achieving a positive DCAD.

Other potassium carbonate ingredients can have severe reactions when they come in contact with moisture; however, when DCAD Plus comes in contact with moisture it does not heat, making it safe for cows to consume. A unique, patented manufacturing process makes DCAD Plus stable during mixing and feeding—making it the ideal supplement for balancing DCAD in dairy cattle.

SUMMARY

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 whereby each affects the outcome of the other. Certainly, there is no single nutritional or managerial factor that will guarantee or optimize the outcome of 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 an integral role affecting production performance and disease incidence for both pre- and postpartum cows. While there are still unanswered questions regarding the specifics in calculating DCAD and some of the physiological consequences of altering DCAD, the evidence for feeding highly positive DCAD diets to lactating cows and negative DCAD diets for prepartum transition cows is extremely strong. While DCAD is not the single factor that will increase production or eliminate metabolic- and production-related diseases, it is a major factor to consider when formulating rations.

It is our hope that the information provided in this Research Summary provided an in-depth view into the value of the DCAD concept and benefits of balancing DCAD in dairy cattle diets. DCAD balancing can be complex concept, but provides simple, yet profound results to any dairy operation. More information on the DCAD concept and the Arm & Hammer Animal Nutrition product portfolio can be found by visiting AHDairy.com.

Arm & Hammer Animal Nutrition, a business of Church & Dwight Co., Inc., is proud to offer products and support that help dairy producers improve herd productivity, health and performance. We are confident in saying that the products and resources cited in this Research Summary will do just that.

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