



The Importance of Dietary Cation-Anion Difference (DCAD) in Prepartum and Postpartum Periods of Dairy Cattle Breeding

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Abstract

The most important period of a dairy cow breeding is the first few days of postpartum. In this period the metabolism of the cow is under severe stress before transitions to lactation period. In this period, there is a huge demand on nutrients to meet requirements for milk production. This period is most susceptible period for some diseases and metabolic disorders. Heifers commonly experience milk fever, retained placenta, displaced abomasum, and mastitis. A glossy passage from the dry pen to the fresh pen is critical to complete high peaks and high production throughout the lactation.

Hypocalcemia (low blood calcium) resulting from inadequate calcium metabolism is common among fresh cows, and can lead to milk fever and other health disorders. Dairy nutritionists are beginning to fine-tune anionic salt feeding and macromineral nutrition recommendations according to the cation-anion difference concentration in the diet.

Many papers on feeding anionic salts and dietary cation-anion difference (DCAD) concepts have appeared recently in the scientific literature and popular press. The objective of this paper and presentation will be to deliver the newest information on current strategies to feed and supplement the close-up dry cow to prevent hypocalcemia and associated disorders.

Key Words: Dietary Cation-Anion Difference, Dairy Cattle, Prepartum and Postpartum Periods.

Introduction

Efficient milk production continues to require the dairy cow to experience gestation and parturition each year. The transition from pregnant, non-lactating to non-pregnant, lactating is too often a disastrous experience. Most of the metabolic diseases of dairy cows: milk fever, ketosis, retained placenta, and displacement of the abomasum occur within the first 2 weeks of lactation. The etiology of many of those diseases that are not clinically apparent during the first 2 weeks of lactation, such as laminitis, can be traced back to insults that occurred in early lactation. In addition to metabolic disease, the overwhelming majority of infectious diseases, especially mastitis, but also diseases such as Johne's disease and Salmonellosis, become clinically apparent during the first 2 weeks of lactation. The wellbeing of the cow and her profitability could be greatly enhanced by understanding those factors that account for the high disease incidence in periparturient cows. Three basic physiologic functions must be maintained during the periparturient period if disease is to be avoided. These are: I. adaptation of the rumen to high energy density lactation diets to reduce the degree of negative energy balance experienced by the cow; II. maintenance of normocalcemia; and III. reducing the degree of immunosuppression that occurs around

parturition. Both metabolic and infectious disease incidence are greatly increased whenever one or more of these physiological functions is impaired (Goff et al., 1997).

The transition period, as defined by Grummer (1995), is the 3 weeks before and after parturition. The transition period is considered the most difficult time of the annual cycle of the dairy cow (Keady et al., 2001), determining the cow's health, production, and reproduction in the subsequent lactation. At or near calving, most cows experience some degree of hypocalcemia (Horst et al., 1997). Clinical hypocalcemia or milk fever affects up to 9% of dairy cows (Goff et al., 1987). In a 2007 survey (USDA, 2007), 83.5% of all dairies in the United States reported clinical milk fever as a health problem with an incidence rate of 4.9%. Guard (1996) estimated the average cost per case of milk fever to be \$334. In addition, cows with milk fever are susceptible to secondary disorders, such as ketosis, mastitis, retained placenta, and displaced abomasums (Curtis et al., 1983). Several nutritional strategies have been used in the prevention of hypocalcemia, including Ca restriction during the prepartum period (Goings et al., 1974) and decreasing the DCAD {mEq [(Na + K) – (Cl + S)]/100 g of DM} during the last 3 to 4 wk of gestation (Block, 1984; Goff and Horst, 1997b). Because of the difficulty in formulating very low calcium diets, recent interest has focused on DCAD (Horst et al., 1997). Although decreasing DCAD through removal of cations is effective in preventing hypocalcemia (Goff and Horst, 1997a), this generally precludes the use of farm-grown forages high in potassium. The alternative to lowering the DCAD of prepartum diets is to feed anionic salts [MgSO₄, MgCl₂, NH₄Cl, (NH₄)₂SO₄, CaCl₂, and CaSO₄] or products specifically formulated to deliver a low or negative DCAD. Several trials and meta-analyses have been

published demonstrating that DCAD is negatively correlated to blood and urinary pH (Sanchez et al., 1994; Hu and Murphy, 2004; Spanghero, 2004; Apper-Bossard et al., 2006; Charbonneau et al., 2006; Lean et al., 2006; Hu et al., 2007). Diets high in Na and K cause blood pH to increase, resulting in a state of mild metabolic acidosis (Goff et al., 2004). This in turn reduces the ability of the periparturient cow to maintain Ca homeostasis at or near calving by reducing tissue responsiveness to parathyroid hormone (Phillippo et al., 1994; Goff and Horst, 1997b). Low DCAD diets (≤ -5 mEq/100 g of diet DM), achieved by addition of anions, increase target tissue responsiveness to parathyroid hormone by preventing mild metabolic alkalosis (Horst et al., 1997). Several studies have shown improved Ca metabolism by decreasing DCAD through feeding anionic salts (Joyce et al., 1997; Moore et al., 2000). In these studies, the decline in blood Ca in the periparturient period of ± 2 d (Day) of calving was investigated. However, only one study (Block, 1984) has shown a positive milk yield response (7%) for cows fed anionic salts prepartum. In addition, anionic salts can have a negative effect on prepartum DMI (Joyce et al., 1997; Moore et al., 2000). The reduction in prepartum DMI caused by the addition of anions is often attributed to decreased palatability, but may represent a response to metabolic acidosis induced by anionic salts (Vagnoni and Oetzel, 1998). Regardless of the exact mechanism by which anionic diets elicit their negative effect on prepartum DMI, decreasing DMI may have an adverse effect on metabolism. The present study was conducted to determine if prepartum DCAD (+20 or -10 mEq/100 g of DM) and source of anions (feed-grade anionic salts or commercially available feedstuffs formulated to deliver negative DCAD) affect peripartum DMI, Ca homeostasis, and milk production in the subsequent lactation.

Dairy Cattles in Parturition Period

The close-up dry cow period, defined as the time from three weeks prepartum to parturition, is an extremely critical time for the dairy cow; possibly defining her entire lactational performance (Wang, 1990). Many years ago, researchers discovered that a diet that was acidic caused the concentration of blood calcium to increase. This led to the practice of feeding a diet with more anions relative to cations to help reduce milk fever problems. The increased blood calcium in these cows not only prevented them from going down with milk fever, it also reduced problems like retained placentas and displaced abomasums. These problems were tied to a calcium deficiency that prevented the muscles from contracting. Therefore, nutritionists began feeding prepartum cows diets with less cations than anions to help increase blood calcium around the time when it was very deficient at calving. These diets are described as anionic diets or diets with a low or negative cation-anion difference. Milk fever may affect 5-7% of all high producing adult dairy cows in the United States (Jordan and Fourdraine, 1993). In addition, the prevalence of subclinical hypocalcemia may be as high as 66% for multiparous dairy cows following calving (Beede et al., 1992). 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, 1988). Guard (1996) estimated that the cost associated with a single case of milk fever is approximately \$334, when considering lost production

and income, veterinary costs, and treatment costs. According to (Curtis et al., 1984) research, it was determined that there was a significant correlation between milk fever, dystocia, placental residence and Ketosis, and in these cases, ketosis, As a result of this disease can lead to; diarrhea, silent heats and ovarian cysts,

Hypocalcemia and Milk Fever

Milk fever, also known as parturient paresis, is a well-known metabolic disorder that occurs at or near calving, particularly in high producing cows. A fairly common problem, it's estimated to occur at the rate of 5-10% nationwide (Horst, 1986). Recently, the economic loss associated with milk fever was estimated at \$334 per occurrence (Guard, 1996), including cost of treatment and loss in milk production. Incidence tends to increase with age and is higher in Jerseys compared to Holsteins. Cows that recover from milk fever are less productive and more susceptible to other health disorders. Disorders such as ketosis, mastitis, retained placenta, displaced abomasum, and uterine prolapse. Milk fever results from severe hypocalcemia. Most cows experience subclinical hypocalcemia after parturition, but some experience severe hypocalcemia that leads to milk fever. Normal blood calcium is 8-12 mg/ dl. When blood calcium is reduced to 7.5 and 5 mg/dl, abomasal motility is reduced 30 and 70%, respectively (Daniel, 1983). This is due to the vital role of calcium in muscle contraction. Reduced abomasal motility from low blood calcium can lead to displaced abomasum. During the dry period, calcium needs are minimal, but after parturition large amounts of calcium are exported into milk. This sudden calcium drain must be countered by increased calcium absorption from the gut or calcium resorption (mobilization) from bone. Bone is the primary source of calcium when absorption from the gut is less than required, because bone contains

nearly all of the calcium stores in the body. Absorption of calcium in the gut occurs primarily in the small intestine and is facilitated by 1,25 dihydroxyvitamin D3. Calcium resorption from bone is influenced by 1,25 dihydroxyvitamin D3, parathyroid hormone, and blood calcium. As blood calcium drops, parathyroid hormone is released to enhance calcium resorption from bone. Hypocalcemia results when mechanisms of calcium absorption and resorption are insufficient to meet calcium demands. If untreated, most cows with milk fever die within a day. Methods for treating milk fever involve elevating blood calcium. For down cows, treatment normally consists of 8-10 g intravenous calcium. Due to the role of the vitamin D metabolite 1,25 dihydroxyvitamin D3 in calcium absorption and resorption, some have recommended supplementing large quantities of vitamin D orally before calving to alleviate hypocalcemia and milk fever. Recent work (Goff et al., 1986, 1989) has suggested intravenous or intramuscular injection of parathyroid hormone may reduce incidence of milk fever. Calcium gels have been used successfully to administer supplemental calcium in hopes of elevating blood calcium. The gels are typically CaCl₂ or calcium propionate. CaCl₂ can cause ulceration of the mouth and digestive tract, induce severe metabolic acidosis, and reduce dry matter intake. Calcium propionate has the advantage of containing propionate, a readily available energy source for the cow.

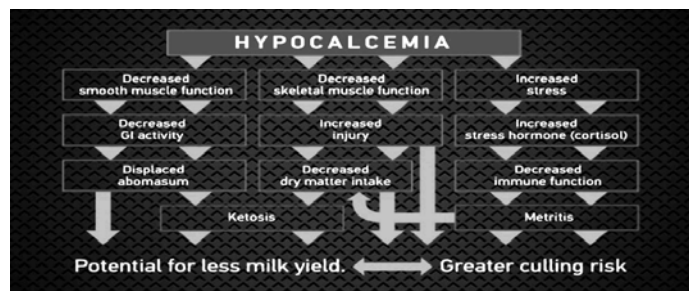


Figure 1. Hypocalcemia and related disease (Curtis et al., 1984).

Dietary Cation-Anion Difference (DCAD)

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 plus K minus Cl (in meq per 100 g diet 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 *cation-anion difference* to represent, more precisely, the mathematical calculation used and to avoid the erroneous connotation that mineral cations truly are balanced with mineral anions in the diet. Expressed in its fullest form, DCAD would be written as:

Equation 1: $\text{meq} [(Na + K + Ca + Mg) - (Cl + S + P)] / 100\text{g of dietary DM}$

A problem with including the multivalent macrominerals (Ca, Mg, P, and S) in the DCAD expression for ruminants, relates to the variable and incomplete bioavailability of these ions compared to Na, K and Cl. The expression that has been used most often in non-ruminant nutrition is the monovalent cation-anion difference expressed as:

Equation 2: $\text{meq} (Na + K - Cl) / 100\text{ g dietary DM}$

This expression was considered superior for nonruminant nutritionists because it comes closest to representing feed ions that are completely dissociated and solubilized from their respective salts, and absorbed into the body. Because of the additional use of sulfate salts in prepartum rations, the expression that has gained the most acceptance in ruminant nutrition, and is the most common expression used in ration software, is:

Equation 3: $\text{meq} [(Na + K) - (Cl + S)] / 100\text{ g dietary DM}$

Calculation of Dietary Cation-Anion Difference in Dairy Cattle Rations

To actually calculate DCAD using Equation 3 mineral concentrations are first converted to meq as follows (Goff et al., 1986, 1989):

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

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 (minimum recommendation for dry cows; NRC, 1989) will be calculated. 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:

$$\text{meq (Na + K) - (Cl + S)} = 4.3 + 16.7 - 5.6 - 10.0 = +5.4 \text{ meq}/100 \text{ g diet DM.}$$

Another simpler way to calculate DCAD is to use:

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

For example, using the same numbers as above, the calculated DCAD equals $(0.10\% \text{ Na}/0.023) + (0.65\% \text{ K}/0.039) - (0.2\% \text{ Cl}/0.0355) - (0.16\% \text{ S}/0.016) = +5.4 \text{ meq}/100\text{g diet DM.}$

Note that values calculated on a per 100 g basis are 10 times less than on a per kg basis (100g =

kg/10). Note also that the DCAD equation with only Na, K, and Cl in it yields a value approximately 10 DCAD units higher than with the equation with Na, K, Cl, and S in it (assuming S is equal to 0.16%)

Important Points in Balancing DCAD for Dairy Cow Rations

- ✓ The cations (Na and K) and anions (S and Cl) should be analyzed in feeds with wet chemistry. Do not use NIR for mineral determinations.
- ✓ Meet the requirements for Na, K, S, and Cl.
- ✓ It is possible, in some cases, to increase DCAD to the recommended +25 to +30 range for lactating cows by reducing S. After this, DCAD can be increased by adding sodium bicarbonate, sodium sesquicarbonate, or potassium carbonate.
- ✓ Greater cation amounts are needed for lactating cows during hot weather due to increased needs for Na (.45%), K (1.5%), and magnesium (.35%).
- ✓ Dry cows should have low DCAD diets approaching 0 and in some cases be negative if metabolic regulation is attempted with the use of anionic salts or hydrochloric acid during dry period transition. High-K forages will have to be limited or eliminated from rations to do this. Urinary pH between 6.2 and 6.8 indicates the effectiveness of anion sources and should be used as the criterion for effective inclusion amounts (Goff et al., 1986, 1989).

Dietary Cation-Anion Difference and blood Acid-Base Status

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, excess mineral cations relative to anions or excess

anions relative to cations, 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) 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 (Mongin, 1980).

Ration Dietary Cation-Anion Difference Balancing in Prepartum Period for Prevention of Hypocalcemia

The traditional method of preventing milk fever has been to restrict calcium intake during the dry period. In theory, this helps condition the cow to calcium deficiency and makes her better able to respond to the acute, intense calcium demands which occur when lactation commences. Calcium intake during the dry period is usually restricted by replacing some or all of the alfalfa hay in a dry cow diet with a grass hay and using additional corn silage and concentrates. This feeding practice does help reduce the incidence of milk fever, but it has several drawbacks. Feeding concentrates and/or corn silage to dry cows may be expensive and may predispose cows to over-conditioning and subsequent fatty liver syndrome, ketosis, and/or abomasal displacements because of their high energy density. Diets consisting of grass hay and corn silage alone have been advocated; however, such diets usually contain sub-optimal levels of protein. Avoidance of alfalfa in the prepartum diet because it is too high in calcium is unfortunate because alfalfa is usually the least expensive and most readily available source of forage protein. Avoidance of alfalfa in the dry period also forces

a detrimental forage switch at the time of calving, since lactating rations are usually based on alfalfa forage. DCAD is more important in controlling milk fever than calcium intake (Goff and Horst, 1997; Oetzel et al., 1988; Oetzel, 1991). Dietary calcium does somewhat influence the incidence of milk fever; however, it does so in a non-linear fashion. Both high and low dietary calcium were associated with slightly lower incidence rates of milk fever (Oetzel, 1991). High concentrations of dietary potassium (a strong cation and dietary alkalinizer) caused milk fever in a recent study (Goff and Horst, 1997), but differing levels of dietary calcium had no effect on milk fever incidence (Table 1).

Table 1. Effect of dietary potassium and calcium on milk fever and urinary pH in Jersey cows (Goff and Horst, 1997).

Diet	DCAD ^a (meq/kg)	Milk fever cows/Total cows	Milk fever treatments/Diet	Urinary pH (Mean ± SEM)
Low Ca ^b - Low K ^c	-150	0/9	0	5.8 ± .22
Low Ca - Medium K ^c	+150	4/11	9	8.0 ± .08
Low Ca - High K ^c	+450	7/9	15	8.1 ± .11
High Ca ^b - Low K	-150	2/10	2	5.7 ± .09
High Ca - Medium K	+150	6/9	9	7.9 ± .11
High Ca - High K	+460	3/12	4	8.2 ± .06

Dairy cattle diets with a high DCAD (alkaline diets) tend to cause milk fever, while a low or negative DCAD (acidic diets) tends to prevent milk fever (Block, 1984). Acidic diets promote bone mobilization (osteocytic resorption) since bone (along with the kidney) acts as a buffer against excessive systemic acidity. Acidic diets have minimal effect on intestinal absorption of calcium (Fredeen., 1988). Additionally, low DCAD diets have been shown to increase the amount of 1,25 dihydroxyvitamin D produced per unit increase in parathyroid hormone (Damir et al., 1994; Goff et al., 1991; Phillippo et al., 1994). This increases osteoclastic

bone resorption, which is probably the most important calciotropic effect of acidic diets (Fredeen., 1988).

Most typical diets fed to dry cows will have an DCAD [using the formula (Na + K) - (Cl + S)] of about +100 to +250 meq/kg dry matter. Addition of a cationic salt (such as sodium bicarbonate) to the dry cow diet increases DCAD and increases the incidence of milk fever. Adding anionic salt(s) (minerals high in Cl and S relative to Na and K) or mineral acids to the diet lowers DCAD

and reduces the incidence of milk fever. Adding three equivalents of anions to 12 kg of diet dry matter lowers DCAD by 250 meq/kg Properties of common anionic salts are presented in Table 2.

Table 2. Approximate retail costs and properties of anionic salts used in prevention of milk fever (Oetzel et al., 1988).

Anionic salt	Molecular Weight	Equivalent Weight	Cost:	
	(g)	(g)	(\$/cwt)	(¢/eq)
MgCl ₂ · 6H ₂ O	203.3	101.7	92.50	20.7
MgSO ₄ · 7H ₂ O	246.5	123.2	30.00	8.1
CaCl ₂ · 2H ₂ O	147.0	73.5	21.00	3.4
CaSO ₄ · 2H ₂ O	172.2	86.1	19.00	3.6
NH ₄ Cl	53.5	53.5	40.00	4.7
(NH ₄) ₂ SO ₄	132.1	66.1	21.50	3.1

Ration Dietary Cation-Anion Difference Balancing In Postpartum Period For Lactating Cows

In order to calculate DCAD it is necessary to know the concentrations of Na, K, S, and Cl and multiply by a constant that is derived from the atomic weight.

The equation for calculation of DCAD most often used is: milliequivalents (Na + K) - (S + Cl)/100 grams dry matter.

To get the milliequivalents of each mineral: mineral, percent of dry matter/constant for mineral based on atomic weight = milliequivalents/100 grams of dry matter.

Using the 2001 NRC requirements for Na, K, S, and Cl as percent of dry matter at 100 pounds of milk per day gives:

	Required %	Constant		Milli-equivalents
Na	0.22%	0.023	=	+10
K	1.06%	0.039	=	+27
S	0.20%	0.016	=	-13
Cl	0.28%	0.0355	=	-8

Total cation milliequivalents/100 grams = 10 + 27 = +37

Total anion milliequivalents/100 grams = 13 + 8 = -21

Cation-anion difference = 37 - 21 = +16

Therefore, the minimal DCAD for lactating dairy cows should be around +16, but as indicated above the optimal is greater than this.

Using Dietary Cation-Anion Difference (DCAD) to Formulate Rations for Close-Up Dry Cows

- ✓ The first step in calculating DCAD is to have a macromineral analysis for all feeds in the diet. Wet chemistry techniques are recommended for accurate mineral analysis.
- ✓ Select forages that are low in potassium. This will result in a lower DCAD, thus reducing the amount of anionic salts necessary to achieve desired effects.
- ✓ Calculate DCAD using the formula presented in this paper.
- ✓ If anionic salts are to be supplemented, start with magnesium sulfate, because it's the most palatable. Add until total magnesium is 0.4% of dry matter. Next, add calcium sulfate and/or ammonium sulfate until sulfur is 0.4 to 0.5% of dry matter. Last, add calcium chloride and/or ammonium chloride until DCAD is -5 to -15 milliequivalents per 100 g dry matter.

- ✓ Elevate the calcium to 1.5 to 1.8% of dry matter. Negative DCAD diets increase urinary calcium excretion, so more dietary calcium is necessary to meet requirements.
- ✓ After a week of feeding anionic salts, monitor the urine pH of close-up dry cows. If pH is above 7.0, more anionic salts can be added. If pH is 6.5 to 5.5 and dry matter intake is acceptable, then continue with the current diet. However, if pH is less than 5.5 or dry matter intake has significantly declined, remove some of the anionic salts.

When selecting anionic salts, consider the source. Some sources may have a higher bioavailability, thus increasing the likelihood of sufficient mineral absorption. Do not use sodium chloride and potassium chloride because they contribute both anions (sodium or potassium) and cations (chloride), so their effect is neutralized. When using ammonium salts, check non-protein nitrogen levels to avoid ammonia toxicity. Minimize ammonium salt in diets with more than 70-75% of its protein in the degradable form (Davidson et al., 1995).

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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