

Macromineral and DCAD Guidelines for Early Lactation Cows that Peak Over 130 Pounds of Milk

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INTRODUCTION

Most cows on commercial dairies have the genetic potential to peak over 130 pounds of milk. Feeding diets with the optimum concentration of macrominerals and DCAD is becoming one of the tools producers have to maximize this potential.

Dairy nutritionists have begun to fine-tune buffer feeding and macromineral nutrition according to the cation-anion difference concentration in the diet. Numerous papers on feeding buffers, macromineral recommendations, 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 relevant information related to this subject for the early lactation dairy cow. For a general review and broader examination of these and other related topics see Sanchez (1999a,b) and reviews by Beede et al. (1995) and Horst et al. (1997).

UNIQUE FEATURES OF THE EARLY LACTATION COW

This conference is known for excellent papers on the transition cow. Only a partial review of this information will be presented here.

It is not unusual for a high producing cow in the first 100 days in milk (**DIM**) to consume 50 lbs of DM/day with more than half of this amount is in the form of concentrates without posing any particular problems to the cow if the diet is well balanced. However, this same diet consumed by a fresh cow can cause a severe ruminal acidosis. The major difference in rumen function between these two stages of lactation can explain the different responses to the same diet. During the dry period, cows generally consume a diet that is principally composed of forages and, by consequence, is more fibrous than the type of diet offered in lactation. This nuance affects rumen function in two ways. First, the rumen flora is adapted to a diet that is low in non-fiber carbohydrates (**NFC**) during the dry period allowing for a large population of cellulolytic bacteria and a low population of amyolytic bacteria. As the amyolytic bacteria also generate lactic acid, their decrease is accompanied by a decrease in the bacteria that utilize lactic acid (Goff, 1999). If the ration is changed abruptly at calving the capacity of the rumen flora to metabolize lactate, the principal acid responsible for acute rumen acidosis, is at a minimum at the initiation of lactation. The lactate producing bacteria increase in numbers rapidly as the amount of NFC in the diet increases but the lactate-utilizing bacteria adapt more slowly (3 to 4 wk). Therefore, the risk of lactate accumulation in the rumen is high with abrupt changes from high to low fiber diets.

The second factor has to do with the length and number of rumen papillae. These papillae are the absorptive surface in the rumen and they primarily absorb the volatile fatty acids (VFA) generated during normal fermentative processes. These papillae can decrease in size by half during the dry period by reason of the low dietary NFC (Dirksen et al., 1985). If dietary NFC increases abruptly at calving, with high levels of fermentable carbohydrates, the amount of VFA produced far exceeds the capacity of the rumen to absorb them leading to elevated concentrations of VFA in the rumen. This situation leads to the phenomenon known as subacute rumen acidosis (SARA) and contributes to reduced DMI and feed digestibility as well as laminitis in the early postpartum period.

HEALTH PROBLEMS OF THE EARLY LACTATION COW

One of the major reasons cows do not have high peaks and high milk production in early lactation is that they are prone to health problems during the transition period. The principal metabolic problems gravitate around three principal axes: 1) disorders related to energy metabolism (fatty liver, ketosis, subacute and acute ruminal acidosis); 2) disorders related to mineral metabolism (milk fever, sub-clinical hypocalcemia, udder edema,); 3) problems related to the immune system (retained placenta, metritis, mastitis). We will see in the next sections some of the etiology and preventive measures for these problems and the nutritional considerations related to these three axes. It is necessary to mention here that these disorders are interrelated and that the conditions conducive to the development of one problem in one category can lead to a disorder in another category. For example, milk fever or subclinical hypocalcemia can lead to loss of muscular tonicity resulting in an increased risk of retained placenta and/or displaced abomasum in spite of retained placenta being classified as a problem related to the immune system. This paper primarily focuses on acidosis related problems but it can't be overemphasized that these disorders are not independent of one another.

Curtis et al. (1985) conducted a retrospective analysis of the risk factors associated with metabolic problems. Their analysis revealed that older cows were more at risk for retained placenta, milk fever and mastitis. Cows having retained placentas were more at risk for developing mastitis and ketosis. Cows with ketosis were 12 times more likely to develop displaced abomasums. Generally, this analysis suggests that that prevention of one problem can decrease the incidence of others from developing.

NUTRITIONAL CONSIDERATIONS DURING THE TRANSITION PERIOD

The transition period is marked by major hormonal changes. While these hormones are causing a reduction in DMI there is an increase in nutrient requirements by the cow to support fetal growth, mammogenesis and lactogenesis (Bell, 1995; Grummer, 1995). This increase in nutrient demand is partially met by the DMI and partially by the mobilization of body tissues. Although the hormonal milieu will drive a certain amount of this body mobilization, excessive body catabolism is undesirable for health, reproduction and milk

production. It is, therefore, essential to pay particularly close attention to the formulation of rations in this transition period, both pre- and post- partum.

Regulation of energy metabolism and glucose homeostasis. Glucose is a substance that plays a fundamental role in all living beings. In the last weeks of fetal development, the fetus uses an estimated 46% of maternal glucose taken up by the uterus (Bell, 1995). Additionally, a cow producing 30 kg of milk per day uses at least 2 kg of blood glucose to synthesize lactose for milk (Bell, 1996). The end of pregnancy and the beginning of lactation, therefore, represent a time when there is a massive increase in need for glucose. This poses an enormous challenge for the liver that has to synthesize all of this glucose from propionate and amino acids as well as a challenge for other tissues and organs that have to adapt to a reduction of glucose availability. Glucose is an equally important energy source for the ovary and the reduced glucose availability in the beginning of lactation can negatively impact the reestablishment of ovarian activity after calving (Rabiee et al., 1999).

The concentration of plasma insulin decreases dramatically as calving approaches and remains low in the first weeks postpartum. The concentration of plasma somatotropin increases dramatically and rapidly postpartum. This decrease in insulin combined with a decrease in sensitivity of adipose tissue to insulin and the increase in somatotropin results in a decrease in the synthesis of triglycerides in adipose and favours their mobilisation (Bell, 1995; Lanna and Bauman, 1999). Therefore, there is a resulting increase in plasma NEFA at calving. The somatotropin, in concert with elevated cortisol, stimulates the liver to produce more glucose.

These coordinated changes in metabolism permit the cow to respond to the accrued nutrient demands for milk synthesis. However, due to many factors, including those previously mentioned, the metabolic processes do not adjust sufficiently and a disequilibrated metabolism leads to many of the typical problems associated with transition cows.

Metabolic problems associated with energy nutrition. The mobilization of lipids in the beginning of lactation is a normal and required process to help the cow meet her energy demands for lactation. However, when the quantity and/or the speed of mobilization are exaggerated the incidence of metabolic problems increase significantly. It is not uncommon to find a ketotic cow also having problems with fatty liver and displaced abomasum. Ruminal acidosis is also a frequent problem for cows at the beginning of lactation because of highly fermentable rations and insufficient rumen adaptation to these rations.

Rumen acidosis. When acid production in the rumen resulting from fermentation of organic matter exceeds the capacity for the animal to absorb these acids or be neutralized, ruminal pH falls. This acidosis, even at the subclinical level, will cause a reduction in cellulolytic bacteria in the rumen and contribute to an overall reduction in feed digestibility. Furthermore, the acidosis reduces rumen motility and efficacy of mixing rumen contents, which reduces the amounts of VFA near the rumen wall. Consequently, VFA absorption is reduced (Allen and Beede, 1996). As rumen motility and mixing decline so does rumination, which reduces the amount of saliva flowing into the rumen. The secretion of buffers in the

saliva is equivalent to more than 7 lbs of sodium bicarbonate and 2 lbs of disodium phosphate (Erdman, 1988).

When NFC present in the rumen increases abruptly and rapidly, the production of VFA increases proportionately. If these VFA are not absorbed rapidly they will accumulate in the rumen. Different problems are caused by rumen acidosis depending on the quantity of acids that accumulate in the rumen. When there is a mild accumulation, DMI and production can decline in spite of the cow appearing to be in good health (Owens et al., 1998). Also, the milk produced could have reduced fat. Certain species of bacteria and protozoa in the rumen are extremely sensitive to acidosis and release endotoxins and histamine. These substances can contribute to laminitis. As the acid accumulation continues more severe problems can occur with DMI and production and also with overall health (Nocek, 1996).

It appears that this subclinical rumen acidosis is insidious and omnipresent in transition cows as well as cows in early lactation. It is beyond the scope of this presentation to adequately describe this subject area, however, there are numerous publications dealing with its description and detection (Yokoyama and Johnson, 1988; Nordlund, 1994; Allen and Beede, 1996; Hall, 1999).

THE NEED FOR DCAD BY THE LACTATING COW

Based on the above discussion it appears prudent to begin to transition the dry cow to her lactating ration as soon as possible. Early lactation cows should be fed rumen buffers and higher DCAD diets. Higher DCAD diets seem to help offset some of the above transition problems including: abomasal displacement, reduced rumination, irritation to the rumen wall decreased feed consumption, low milk fat test, laminitis and reduced milk production. We feed buffers and raise the DCAD to help offset the acidosis and reduce the associated problems.

Research on DCAD for the lactating cow is more limited than that for the dry cow. However, recent research has shown that DCAD can affect milk production and intake of the lactating cow. It appears that DCAD in the range of +30 to +45 maximizes milk production and blood bicarbonate (the blood buffer that counteracts acid in the blood).

NUTRITIONAL FACTORS RELATED TO CATION-ANION INTERRELATIONSHIPS

Leach (1979) and Mongin (1980) reviewed nutritional concepts related to cation-anion interrelationships. Historically, nutritionists intuitively knew it was difficult to evaluate the effect of one macromineral without considering the influences of others. Early concepts evaluated total ash, mineral ratios, and differences between two or more of the macrominerals.

Acid or Alkaline Ash. Nutritionists first investigated the alkalinity and acidity of the diet under the acid- or alkaline-ash concept (Shohl, 1939). It was recognized that human food

either had an acid or alkaline ash. When food is metabolized in the body, organic anions, such as acetate, citrate, malate, etc., are oxidized. Inorganic cations originally associated with these organic anions remain. Because organic anions can buffer H^+ ions generated through metabolism, a food with a large amount of organic anions (and thus inorganic cations) was considered alkaline. The pH of the ash represented the acid or alkaline nature of human food.

Much of the current research on feeding macrominerals to dairy cows centers on the effects of the cation and anion charges. As you think about these charges envision common salt or sodium chloride in a glass of water. The salt is quickly solubilized in water because it ionizes into Na^+ and Cl^- ions. As ions these macrominerals affect the concentration of hydrogen (H^+) and bicarbonate (HCO_3^-) ions in body fluids.

When the positively charged ions or cations (i.e., Na^+ , K^+ , Ca^{++} , and Mg^{++}) increase in blood, HCO_3^- also increases (to counteract the positive charges). Because HCO_3^- is a base we say that the cations (Na^+ , K^+ , Ca^{++} , and Mg^{++}) as basic and these help to neutralize acid. When the negatively charged ions or anions (i.e., Cl^- , S^- , P^-) ions increase in blood, H^+ also increases to counteract the negative charges. Because H^+ is acid we refer to the anions (Cl^- , S^- , P^-) as acidic and these increase acid. Figure 1 (below illustrates this phenomenon).

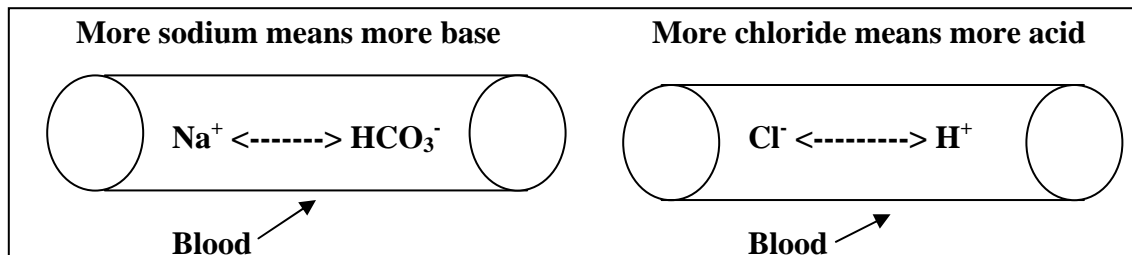


Figure 1. Effect of cations (i.e., Na) and anions (i.e., Cl) on blood acid-base status.

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 milliequivalents (**meq**) of 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 **dietary 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.

Nutritionists have used the effects of these ions on H^+ and HCO_3^- concentrations in body fluids to predict the effect of the diet on the balance of acids and bases in the blood. When a diet contains more mineral cations than anions the diet is basic and blood and urine pH are increased. When a diet contains more mineral anions than cations the diet is acidic and blood

and urine pH are decreased. Thus the expression dietary cation-anion difference or DCAD was developed to help nutritionists determine if the diet is acidic or basic.

Expressed in its fullest form, DCAD would be written as:

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

Equation 1

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:

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

Equation 2

This expression was considered superior for non-ruminant 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:

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

Equation 3

A more appropriate equation biologically would be one that discounts the bioavailability value for S (Tucker et al., 1991) to approximately 60% of the value of the monovalent ions Na, K and Cl. However, because S generally does not vary greatly in lactation rations (as compared to close-up rations) this discounting factor is often ignored.

CALCULATING DCAD

To actually calculate DCAD using Equation 3 mineral concentrations are first converted to meq as follows (note - atomic weights come from the periodic table (Table 3):

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

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:

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

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 $(0.10\% \text{ Na}/0.023) + (0.65\% \text{ K}/0.039) - (0.2\% \text{ Cl}/0.0355) - (0.16\% \text{ S}/0.016) = +\mathbf{5.4 \text{ meq/100g diet DM.}}$

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%).

EFFECTS OF DCAD ON POSTPARTUM LACTATIONAL PERFORMANCE

Below is a review of the latest research on the effects of DCAD on lactational performance. In studies where S was not reported, I discuss the results relative to the Na + K – Cl expression (Table 2) and, in studies where S was reported, I discuss the results relative to the Na + K – Cl – S expression (Table 4). As a general rule of thumb, when S equals 0.2% of the diet dry matter (DM), S provides approximately –12 meq/100g of calculated anionic charge. Part of the confusion with the effect of S on acid-base status is related to its incomplete dissociation, and thus, reduced physiological ion effect.

Studies Using the Three Element (Na + K - Cl) DCAD Equation. Kentucky researchers (Tucker et al., 1988; Figure 2) were the first to conduct a study specifically designed to evaluate the effect of DCAD as (Na + K - Cl) on acid-base status and lactational performance of dairy cattle. They compared diets formulated with -10, 0, +10 or +20 DCAD. A diet with a +20 DCAD improved dry matter intake (**DMI**) 11% and milk yield (**MY**) 9% compared with a diet with -10 DCAD. Blood HCO_3^- increased linearly with increasing DCAD, which indicated an improvement in acid-base status with high DCAD compared with low DCAD. They concluded that responses to increasing DCAD were independent of specific Na, K or Cl effects. Because lactation diets typically contain greater DCAD than +20, these results were initially more theoretical than practical. For example, the NRC (1989) minimum Na, Cl, and S requirements indicate that DCAD should be greater than about +25 DCAD. The next question that had to be answered was whether or not responses would continue to increase with diets above +20 DCAD.

West et al. (1991; Figure 2) in Georgia answered part of this question when they evaluated diets with up to +40 meq/100 g of diet DM. Their study used two 4 x 4 Latin squares blocked by environmental temperature (cool vs. hot). Separate squares included four Holstein and four Jersey cows. Diets contained +2.5, +15, +27.5 or +40 DCAD. No effect of environment was reported, but increasing DCAD from +2.5 to +27.5 increased DMI, MY and blood bicarbonate (HCO_3^-). These findings suggested that performance was depressed with lower DCAD. At +27.5 DCAD, negative effects were overcome. Above +27.5 DCAD no additional improvement was attained.

In another study by this group (West et al., 1992; Figure 2), diets with even higher DCAD (+10, +21.7, +33.4 and +45.1) were fed to a total of 16 lactating dairy cows during hot weather. Source of cation (Na or K) used to manipulate DCAD also was compared. Increasing DCAD increased DMI linearly, independent of Na or K source. Yield of 3.5% FCM was not affected by DCAD or cation source. Milk fat concentration was greater with Na- compared with K-manipulated diets (3.92 vs. 3.62%). Blood pH increased linearly; whereas blood HCO_3^- increased curvilinearly, there was no effect due to cation source on acid-base status. Their results indicated that increasing DCAD improved DMI and acid-base status in a manner consistent with other studies. In general, DCAD was independent of a specific Na or K effect.

The influence of Na, K and Cl at constant DCAD was evaluated by Tucker and Hogue (1990). Diets were formulated to provide +32 DCAD in either: a basal diet (adequate in dietary Na, K, and Cl), a basal diet containing an additional 1.17% NaCl, or a basal diet containing an additional 1.56% KCl. Fifteen mid lactation cows were assigned to replicated 3 x 3 Latin squares. The KCl-fed cows consumed more DM and had lower milk fat percentage than NaCl-fed cows, but there were no differences in MY. It was concluded that dietary DCAD was a more important determinant of dietary impact on systemic acid-base status than actual dietary concentrations of Na, K, and Cl.

To model the effect of multiple concentrations of DCAD across a variety of diets and management conditions, Sanchez et al. (1994b) assembled a large database from 10 years of studies with mid lactation cows in Florida. Combining data from many studies into one

analysis verified the curvilinear response to DCAD. Dry matter intake, MY and FCM yield were all maximized at +38 DCAD (Na + K - Cl)/100 g. These models were validated by comparing them to independent data of Tucker et al. (1988), West et al. (1991) and West et al. (1992). The DCAD models developed from the data base predicted results very well. For DMI, the DCAD model and independent data set predictions differed only by an average of 2.87% (range .19 to 12.27%). For MY, they differed only by an average of 2% (range .13 to 7.94%). Absolute deviations between the data base DCAD models and independent data set predictions (corrected for experiment effects) ranged from .24 to 1.22 kg/d for DMI and .07 to .60 kg/d for MY. A summary of studies that used the Na + K - Cl expression is presented in Table 1.

Studies Using the Four Element DCAD [(Na + K) - (Cl + S)] Equation. A summary of studies that used the (Na + K) - (Cl + S) expression is presented in Table 2. A large study with 48 cows and 15 dietary treatments was conducted by Sanchez et al. (1994a) to investigate lactational and acid-base responses to DCAD as [(Na + K) - (Cl + S)]. Treatments consisted of combinations of Na, K and Cl, so that DCAD ranged from 0 to +50 [(Na + K) - (Cl + S)]/100g DM. The basal diet was 54.5% concentrate, 5.5% cottonseed hulls and 40% corn silage (DM basis). Dry matter intake and MY were highest when DCAD [(Na + K) - (Cl + S)] was between +17 to +38 and +25 to +40 respectively (Figure 3). There was one odd treatment (a low Cl, high K, and high Na treatment combination) that may have caused a Cl deficiency. Had that treatment not been included the regression line would have shifted to the right. Blood HCO_3^- (P=.09) also responded quadratically to increasing DCAD.

Blood HCO_3^- was maximized with +38 DCAD [(Na + K) - (Cl + S)]. In support of conclusions of Tucker et al. (1988) and West et al. (1991 and 1992) results of this study indicated that feeding diets with less than +20 DCAD depressed blood HCO_3^- and should not be fed. Note that with most DCAD studies (this one included) dietary carbonate and bicarbonate concentrations are confounded with DCAD. Because these salts are used to elevate DCAD, DCAD effects cannot be separated from well known ruminal and systemic buffering effects of carbonate and bicarbonate salts.

Studies using the Four Element [(Na + K) - (Cl + S)] Equation Throughout Different Phases of Lactation. Three switchback experiments (Delaquis and Block, 1995) were conducted with 12 cows each in early, mid, and late lactation (Table 2). Each experiment compared two DCAD levels calculated as [(Na + K) - (Cl + S)]/100g DM. Increasing DCAD from +5.5 to + 25.8 in early lactation and from +14.0 to + 37.3 in mid lactation increased DMI and milk production. These effects were not observed in late lactation (with either +20.0 or +37.5 DCAD). Concentration of blood HCO_3^- was decreased in early lactation and excretion of carbonate ions in urine was reduced by a lower DCAD at all stages of lactation. Responses in this study are consistent with the effect of DCAD on acid-base status observed in other studies; however, this study supports the concept that response to DCAD is affected by stages of lactation.

Differences between K and Na as the Source of Increased DCAD. In the study by West et al. (1992), the source of cation (Na or K) used to manipulate DCAD also was compared. In these mid-lactation cows, no difference between Na and K was observed.

Tucker and Hogue (1990) evaluated the influence of Na, K and Cl at constant DCAD. This study thus compared effects of Na to K. Diets were formulated to provide +32 DCAD in either: a basal diet (adequate in dietary Na, K, and Cl), a basal diet containing an additional 1.17% NaCl, or a basal diet containing an additional 1.56% KCl. Fifteen mid lactation cows were assigned to replicated 3 x 3 Latin squares. The KCl-fed cows consumed more DM and had lower milk fat percentage than NaCl-fed cows, but there were no differences in milk yield. Again, for these midlactation cows, it was concluded that dietary DCAD was a more important determinant of dietary impact on systemic acid-base status than actual dietary concentrations of Na, K, and Cl.

Perhaps the most complete study on the effects of DCAD on early lactation dairy cows conducted to date is an unpublished trial by Elliott Block and associates from McGill University (E. Block, personal communication, 1999). Block fed a control diet with no added Na or K (+18 DCAD) and two higher (+25 and +52) DCAD diets to early lactation (0 to 10 weeks in milk) Holstein cows. Within the higher DCAD diets he manipulated the source of DCAD (by using either sodium bicarbonate or potassium carbonate alone or a combination of both) to determine the individual or combined effects of Na and K. Block determined that the combination of Na and K yielded the best response in DMI and milk production and that the +52 DCAD diet yielded the highest milk production response (Figure 3). The combinations of Na and K also resulted in the highest blood bicarbonate concentrations (Figure 3).

THE UNIQUE ROLE OF POTASSIUM

The above positive responses observed with combinations of Na and K point to the unique role that dietary K plays in in early lactation. A similar role has been noted for cows in heat stress. Heat stressed cows lose K via sweat and milk is high in K. Thus the heat stressed dairy cow is often K deficient. Research conducted by Joe West in Texas, and Griffel and Sanchez in Idaho where potassium carbonate was the source of dietary K, indicates that there is a linear response to dietary K during summer in Texas and Idaho (West et al., 1986, West et al., 1987a,b, Griffel et al., 1997) at up to 2.1 % dietary K. Figure 4 shows the fat-corrected milk responses to varying dietary K in mid and early lactation heat-stressed and non-heat stressed cows.

We recently conducted two studies to verify if additional K was warranted for lactating cows in commercial herds. In the first trial potassium carbonate replaced potassium chloride and in the second trial potassium carbonate replaced some of the sodium bicarbonate. Milk yield was significantly increased in both studies (3 and 2.5 lbs./day, respectively) so we feel confident that the data from the university studies have practical applications on commercial dairies.

The additional K required in early lactation and during heat stress only increases dietary K needs from 1.0 to 1.5% K (dry matter basis) depending on intake. We are not completely

sure why cows continue to respond to levels closer to 1.8% K. It may be that NRC (1989) calculations for intake are higher than possible, which then would require higher concentrations of K in the diet. There also appears to be other effects of K related to ion pumping, protein metabolism, and hormone (i.e., insulin) physiology. Hopefully research in this area will continue to investigate effects that impact the early lactation cow.

UPPER LIMIT OF DIETARY POTASSIUM

In 1989, the National Research Council published 3% K as the maximum tolerable level (NRC, 1989). The Canadian researchers, Fisher et al. (1994) conducted more-recent research on the effect of high levels of dietary K (Figure 4). They fed diets with 1.6, 3.1 and 4.6% K (low, medium and high) to lactating cows. Their study involved 15 early lactation Holsteins housed in free-stall barns and fed grass-haylage based-diets. The reported DCAD values for these diets were very high at +36.6, + 73.5, and + 108.1 meq [(Na + K) - (Cl + S)]/100 g dietary DM. In terms of palatability effects, the authors noted that 'several cows preferred the 3.1% K diets to the others'. No significant differences were noted between diets on total DMI (23.0, 23.82, and 22.3 kg/day for the low, medium and high K diets, respectively). However, when expressed as a percent of body weight, cows fed the medium K diets had the highest intakes (3.54, 3.59, and 3.36 % of body weight respectively). Milk yields were reduced for cows fed the high K diets but similar for cows fed the low and medium K diets (31.6, 31.5, and 29.8 kg/day for the low, medium and high K diets, respectively). Milk fat concentrations were higher for the medium and high K diets (3.6, 3.72, and 3.79%, respectively).

EFFECT OF ADDITIONAL POTASSIUM ON MAGNESIUM ABSORPTION

When adding additional dietary K, the absorption of dietary Mg will be reduced. Therefore, Mg needs to be increased due to the effect K has on reducing absorption of dietary Mg. When feeding high levels of dietary K optimal levels of dietary Mg appear to be in the 0.35 to 0.38% of diet DM range. Another rule of thumb is to maintain less than a 5:1 ratio of dietary K to Mg (the ratio is calculated with minerals on a percent of diet DM basis).

SOURCES OF DIETARY POTASSIUM

Potassium chloride is a common source of K when both K and Cl are needed but does not increase DCAD. Excess Cl appears to be detrimental to feed intake and milk yield (Sanchez et al., 1994b) particularly in summer, so excess KCl is not recommended. Potassium carbonate is a source of K that increases DCAD. Note that potassium carbonate must be handled and mixed carefully in feed mills and on the farm. Follow manufacturers mixing directions closely.

DIETARY K AND NUTRIENT MANAGEMENT PLANS

Any program that increases the amount of K fed to the lactating cows must consider the overall effect of K on the nutrient management plan on the dairy. This of course is due to the fact that feeding extra K to pre-calving dry cows can contribute to milk fever problems. Therefore, a nutrient management plan that considers both manure and purchased fertilizer K is needed to avoid growing forages with excessively high K. We are aware of two whole-farm potassium balance case histories that help illustrate the importance of nutrient management plans to control excess K in soils and harvested forages.

In the first case, The Cornell University Dairy Farm monitored N, P, and K balances over 25 years (Wang et al., 1999). Table 3 provides the details of the mass balance of K in 1979 and 1994. Feed imports of K increased greatly but because fertilizer sources of K were drastically reduced and the amount of K captured in milk increased, the net balance of K on the farm was reduced by 30%.

The second case history comes from the Washington State University Dairy at Puyallup (Joe Harrison personal communication, 1999). At their dairy they noticed grass-forage K concentrations increasing to levels as high as 6%. Therefore, they eliminated potassium in their purchased fertilizer (they still had K coming in from manure). After a three-year period, grass-forage K concentrations returned to normal. Based on new soil samples, they re-introduced purchased K at a reduced level back into their fertilization program.

VARIABILITY OF FEEDSTUFFS IN MACROMINERAL CONTENT

Obviously to use DCAD in the formulation strategy, one needs values for macrominerals in the diet. Recent research and field observations have demonstrated that the macrominerals can be highly variable in feedstuffs. DePeters et al., 2000 published nutrient values for various byproduct feedstuffs and found large variations in the macromineral content of these feeds. Schauff et al., 2000 reported values for forages and also found that the macrominerals varied in content and were highly different from NRC book values. These types of reports support the need to analyze the minerals in byproducts and forages so that accurate DCAD values can be calculated and formulated.

CONCLUSIONS AND RECOMMENDATIONS

The most recent literature on macrominerals and DCAD for the postpartum cow indicates that:

- The optimal DCAD for mid lactation cows is between +27.5 and +40 meq [(Na + K) - (Cl + S)]/100g DM.

- The optimum DCAD for early lactation cows may be as high as +50 but until more field data on these higher levels becomes available, increasing DCAD to +40 is currently the most practical strategy.
- The combination of Na and K is better than Na or K as the sole source of the increased DCAD - dietary K appears to have a unique role independent of its effect on DCAD.
- When feeding high levels of dietary K optimal levels of dietary Mg appear to be in the 0.35 to 0.38% of diet DM range. Another rule of thumb is to maintain less than a 5:1 ratio of dietary K to Mg (calculated with minerals on a percent of diet DM basis).

These guidelines for macrominerals and DCAD can be affected by numerous factors including production level, feeding management, level of intake depression (i.e., heat stress, etc.) and acid producing potential of the diet.

Church & Dwight will continue to research these and other factors as we strive to provide the latest information on DCAD nutrition for the lactating dairy cow.

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