EFFECTS OF VARYING DCAD AND CALCIUM CONCENTRATION IN PREPARTUM DIETS ON CALCIUM STATUS, HEALTH AND PERFORMANCE IN COWS AND ON NEWBORN CALF ACID-BASE BALANCE

By

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(Under the Direction of John Bernard)

ABSTRACT

The objective of this trial was to determine the effects of feeding a partial or fully acidified diet (-3 compared with -22 mEq/100 g DM) supplemented with 1.3 or 1.8% calcium (Ca) during the prepartum period on blood Ca, postpartum performance and health as well as newborn calf acid-base status. Feeding the fully acidified diet or 1.3% Ca decreased dry matter intake (DMI) prepartum. Milk yield increased after 45 days in milk (DIM) for cows fed the fully acidified diet. Plasma and urine minerals were altered to improved Ca status by 3 DIM for cows fed fully acidified diets, while 1.8% Ca supplementation improved plasma calcium concentration at 1 DIM only. Feeding 1.8% Ca decreased colostrum quality compared to 1.3% Ca. There was no effect on blood mineral or gas concentrations of calves from feeding a partial or fully acidified diet prepartum.

INDEX WORDS: DCAD, calcium, milk yield, colostrum, plasma minerals

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B.S., Cornell University, 2015

A Thesis Submitted to the Graduate Faculty of The University of Georgia in Partial Fulfillment of the Requirements of the Degree

MASTER OF SCIENCE

ATHENS, GA

2017

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DEDICATION

I would like to dedicate this thesis to my parents, who have taught me to always follow my dreams and provided continuous support throughout all my endeavors. Thank you for everything!

ACKNOWLEDGEMENTS

I would like to acknowledge the following individuals and groups for helping make this research trial possible. First, I would like to acknowledge and thank my major professor Dr. John Bernard for his support and guidance during my time completing my degree. I greatly appreciate all his efforts to make me successful during my time at the University of Georgia and in preparing me to enter the dairy nutrition industry. Secondly, I would like to thank Phibro Animal Health Corporation for providing partial funding in support of this project. Lastly, I would like to thank everyone involved in the trial at the University of Georgia Research Dairy Center and in the laboratory.

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CHAPTER 1

INTRODUCTION

The transition period for dairy cattle is marked from late gestation to the early onset of lactation. It is characterized by dramatic changes in endocrine status that influences metabolism (NRC, 2001). During this time, cows are at greatest risk for metabolic diseases which cause health complications and decreased production, or death. Hypocalcemia is a life-threatening disorder caused by a delay of Ca absorption and mobilization after calving. This results in a dramatic decrease in blood calcium concentrations. Hypocalcemia, also known as milk fever, occurs almost exclusively in dairy animals due to the high selectivity for increased milk production (Horst et. al., 2005). Cows that develop milk fever also have a greater risk for developing additional health disorders such as displaced abomasum and retained placenta (Curtis et al., 1985). Disorders like these are associated with low blood Ca because of its action on decreasing muscle contraction.

Addressing issues during the transition period is multifaceted, however nutrition and diet manipulation during this time has proven to decrease the incidence of hypocalcemia, improve production and overall health status. One common diet manipulation is feeding a negative dietary cation-anion difference (DCAD) diet. This induces a mild systemic metabolic acidosis which improves calcium availability and reduces the incidence of hypocalcemia. Inclusion of the proper concentration of Ca

supplementation during the transition period also aids in preparing the mechanisms needed to meet the extreme demand immediately postpartum.

The main objective of this study was to determine the effect of feeding a neutral or fully acidified DCAD diet differing in supplemental Ca during the close-up dry period on plasma Ca status, health, and postpartum performance. The second objective of this study was to determine the effects of feeding a prepartum DCAD diet on newborn calf acid-base status and health.

CHAPTER 2

LITERATURE REVIEW

Hypocalcemia

Hypocalcemia or “milk fever” is a production disease observed nearly exclusively in dairy cows from selectivity for high production (Horst et al., 2005). This results from an imbalance in blood calcium due to the sudden increase in Ca demand immediately postpartum for colostrum and milk production and the lack of adaptation or delay in calcium homeostasis. Goff and Horst (1997) stated that the change in Ca demand can increase from 11 g/d during pregnancy to support the mother and fetus to 23 g/d immediately after calving to support colostrum and milk synthesis. Two varying states of hypocalcemia have been identified and classified as clinical and subclinical hypocalcemia. Clinical hypocalcemia is defined as a blood Ca concentration below 8.0 mg/dL while subclinical hypocalcemia has been defined as a blood Ca concentration below 8.5 mg/dL (Goff, 2008; Martinez et. al., 2012). Symptoms of clinical hypocalcemia include dullness, cold ears, dry nose, uncoordinated walking or inability to stand, and loss of consciousness (Horst et al., 2005). Due to the effect on muscle tone and contractility of the cardiovascular and gastrointestinal systems, if left untreated 60% to 70% of clinical hypocalcemia cases are fatal (Hibbs, 1950). Cows with subclinical hypocalcemia have decreased blood Ca concentrations resulting from the rapid increase of Ca demand but may not show clinical signs. Despite the possibility of no symptoms, subclinical hypocalcemia is associated with decreased DMI and gut function (Martinez et

al., 2014), negative energy balance (Chamberlain et al., 2013), and reduced immune function (Kimura et al., 2006).

Factors of parity and past incidences of milk fever will predispose cows to developing hypocalcemia. Heifers and younger cows are less susceptible because of their high efficiency of Ca absorption. It is thought that younger animals are more able to mobilize Ca from reserves compared to their older counterparts (van Mosel et. al, 1993). Factors for older cows seem to be multi-faceted. One is the sheer quantity of colostrum and milk secreted during early lactation, which increases their demand for Ca. Another is the lack in stimulation of 1,25 dihydroxyvitamin D which decreases the number of receptors in the small intestine reducing the efficient of Ca absorption (Mayer et al., 1969; Horst et al., 1990). Past incidences of milk fever for an individual animal also predisposes them to subsequent incidences and may also be the cause of loss in receptor function as stated previously.

Calcium

Calcium is a macro-mineral required for many physiological processes including muscle contraction, nerve transmission, blood clotting, osmotic pressure and acid-base balance. Approximately 98% of the total body Ca is found in bone which is not readily available while the other 2% is found in extracellular fluid and is readily available (Martin-Tereso & Martens, 2014). Within this readily available fraction, about 50% of Ca is bound to organic or inorganic compounds and 48% is ionized or free (Goff, 2014). As much as 23 grams of Ca are secreted into colostrum alone, which is nearly 6 times greater than what is available in the extracellular pool (Goff et al., 1987). The three locations Ca can be absorbed in the body is the kidney, small intestine and the bone. Calcium enters

the cell via trans-epithelial transport by passive transport of channels TRPV5 in the kidney (Kahnal & Nemere, 2008) and TRPV6 in the intestines (Suzuki et al., 2008). The amount that is passively absorbed by the intestines is expected to be less than 50% due to the dilution effect of the rumen (NRC, 2001). Bouillon et al., 2003 determined these passive transport channels were actually under the regulation of calcitriol. Once Ca enters the body, it undergoes intracellular diffusion mediated by the binding protein calbindin. Calcium moves into the extracellular compartment by active transport of the Ca/ATPase or Na/Ca pump, which are also both regulated by calcitriol (Hoenderop et al, 2005).

Blood Calcium Homeostasis Mechanisms

Calcium concentrations in the body are tightly regulated. Normal plasma Ca concentration in the body is maintained between 8.5 to 10 mg/dL (Cahn & Line, 2005). Parathyroid hormone and 1, 25 dihydroxyvitamin D (calcitriol) are responsible for controlling serum Ca concentrations (Breves et al., 2010). These control gastrointestinal absorption, renal tubular reabsorption and bone turnover of Ca in response to serum calcium concentrations. Excessive Ca loss during late pregnancy and initiation of lactation indicate the need of adaptive changes in Ca status to reduce absorption and increase excretion of Ca to maintain homeostasis (Horst et al., 2005). Gastrointestinal reabsorption of Ca is the first limiting mechanism in prevention of low serum calcium. As 1,25 dihydroxyvitamin D (calcitriol) release from the kidney increases, it stimulates the Ca reabsorption from dietary sources. This gastrointestinal adaptation takes 24 to 48 h to become fully active and driven by the amount of Ca being supplied in the diet (Goff et al., 1989). Renal tubular reabsorption and bone turnover is controlled by parathyroid hormone (PTH). Increased PTH concentrations increase Ca reabsorption from the kidney

which decreases urinary calcium loss. This adaptation occurs in a matter of minutes but can not correct for large deficits in plasma Ca concentration (Martin-Tereso and Martens, 2014). Within bone, PTH causes the rapid turnover of Ca from the blood fluid pool to the extracellular pool (Horst et al., 2005). This adaption also takes around 48 h to become fully active. In order to achieve successful Ca homeostasis, the kidney and bone must be capable to respond to PTH and 1, 25 dihydroxyvitamin D (calcitriol).

Dietary DCAD and Calcium Supplementation

Calcium from mineral supplements is more available than that found in common forages and feedstuffs, but availability is limited by Ca solubility of each mineral supplement (Hansard et al., 1957). Calcium carbonate is commonly fed because the biological value is approximately 75%. The current NRC recommendation for a mature, pregnant Holstein cow is 0.4-0.5% Ca if fed in a non-acidified diet or positive DCAD diet or 1.0-1.5% Ca if fed an acidified diet or negative DCAD (NRC, 2001). It is still debated whether or not to feed above or below the concentration recommended by NRC. Some evidence indicates that limiting Ca to less than 20 g/d may be advantageous in stimulating Ca homeostasis to prevent milk fever (Thilsing-Hansen et. al., 2002). The theory behind this is that the Ca restriction increases Ca absorption mechanisms prior to calving. However, achieving inclusion rates less than 20 g/d is difficult with most common forages and feedstuffs used in the dairy industry.

Miller (1983) concluded that feeding Ca above 1.0% DM may decrease DMI and performance while Beede and Shearer (1991) fed Ca at inclusion rates up to 1.8% with no negative effects on intake or performance. Later, Goff and Horst (2012) observed no increase in blood Ca concentrations of cows fed 1.5% vs 0.5% Ca prepartum while on

diets either -6.1 or -6.8 meq/100 g DM DCAD. Chan et. al. (2006) also reported no change in blood Ca concentration when supplementing diets with 0.99% (moderate) or 1.5% (high) Ca while feeding a diet with -6 meq/100g DM DCAD. Conversely, Oba et. al. (2011) observed that feeding higher Ca concentrations (0.9% vs 0.3%) with anion supplementation improved blood Ca concentrations. Research suggests that when feeding a negative DCAD diet, the inclusion rate of 1.0% Ca is sufficient for maintaining blood calcium (Shire and Beede, 2013).

Dietary Cation Anion Difference (DCAD)

Ender and Dishington (1970) first documented the ability of mineral acids to decrease the incidence of hypocalcemia. Additional studies demonstrated the increase in blood Ca resulted from the addition of anionic salts (Ender et. al., 1971; Dishington, 1975). Mongin (1981) suggested this mechanism of action based on the alteration of acid-base status. Stewart (1983) supported this and further illustrated that acid-base homeostasis was determined by the number of cations and anions available for absorption and reflected in blood. Block (1984) proposed this action as the DCAD theory to be used in transition cows in the dairy industry. In the diet, cations such as K, Na, Ca, and Mg induce a metabolic alkalosis state while anions such as Cl and S induce a metabolic acidosis state. The DCAD influences both acid-base status and calcium metabolism; where negative or low DCAD diets alter Ca status to prevent milk fever (Block 1984, Oetzel 1991). Effectiveness of a low or negative DCAD via anion supplementation was achieved by feeding the diet 2 to 3 wk pre-partum (Oetzel, 1988). Furthermore, Leach (1979) and Goff et al. (2004) concluded that the main anionic action came from excessive Cl inclusion compared to other anions like sulfates because of Cl higher acidifying activity.

The mechanism of DCAD is not completely understood. It is thought that PTH and 1,25 dihydroxyvitamin D (active vitamin D) stimulate bone resorption of Ca, renal reabsorption of Ca and gastrointestinal absorption of Ca, increase ostoclastic activity, resorption of Ca and allows for increased tissue responsiveness via the reduction in blood pH (Goff & Horst, 2003). Manipulation of DCAD using anionic salts may be most effective in inducing an acidotic state (Martin-Tereso and Verstegen, 2001).

Calculating DCAD

The equation for DCAD used most commonly in the dairy industry is defined as (Na + K) – (Cl + S) meq/kg DM. This equation initially was proposed by Ender et al. (1971) and further supported by Block (1984). There have been additional variations proposed to better calculate DCAD. These variations have included using additional ions such as Mg and Ca (Tucker et al., 1991; Horst et al., 1997) and the use of absorption coefficients (Goff, 1997b). Tucker et al., (1991) and Horst et al., (1997) proposed the addition of cations Ca, Mg, and P based on their involvement in acid-base balance. Goff (1997b) proposed the use of coefficients to indicate the alkalization or acidification potential of each ion. In a meta-analysis by Charbonneau et. al. (2006), the addition of 0.6S to the equation correlated better with milk fever incidence and prediction of acid-base status. This change would account for the reduction in acidifying effect by S. The effectiveness of the addition of the 0.6 coefficient for S seems dependent on sources of S in different diets. Another meta-analysis by Lean et al. (2006) reported no additional benefits by including additional ions to the model. It was concluded that the best model for predicting milk fever incidence and bringing DCAD below 0 is the equation (Na + K)

– (Cl + S). It is thought that with this equation, the optimal DCAD is -10 to -15 meq/100g DM to accommodate biological variation within individual cows.

DCAD Effects on Intake and Performance

Some researchers have observed decreases in prepartum DMI because of poor palatability of the anionic salts used to acidify the diet (Otezel, 1993; Moore et al., 2000; Charbonneau et al., 2006), while others reported no change in DMI of diets formulated for -13 versus -7.6 meq/100g DM (Block, 1984; Oetzel 1988). Despite possible lower prepartum DMI effects from anionic diets, DeGroot et al. (2010) reported improved DMI postpartum in cows fed anionic diets. Leno et al. (2017) also reported an improved response in DMI from improved calcium homeostasis early postpartum.

Effects of DCAD are also observed in lactation performance of cows fed anionic diets prepartum. Block (1984) reported a 6.7% increase in milk yield of cows fed -13 compared with +33 meq/100g DM. Research by DeGroot et. al. (2010) also noted increased lactation performance where cows fed -10 or -12 meq/100 g DM prepartum had increased milk yield of 6.5 kg per cow per day compared to cows fed +22 meq/100 g DM. Leno et al. (2017) indicated that lower DCAD diets improve milk yield specifically in the third week of lactation. However, Moore et. al., (2000) and Ramos-Nieves et. al., (2009) observed no change in milk yield or composition by feeding -15 compared with +15 meq/100g DM or -15 compared with +10 meq/100g DM respectively.

Effects of prepartum DCAD on post-partum performance is commonly researched from 21 d to 14 d prepartum. Research has supported that the implementation of a negative DCAD diet should be between 21 d and 28 d prepartum. DeGaris et al. (2008) indicated that increasing the number of days cows were fed negative DCAD diets

prepartum increased milk yield, especially at 22 and 25 d pre-partum. Wu et al. (2014) reported minor differences in blood metabolites with extended feeding of negative DCAD diets and indicated feeding longer than 21 d prepartum has no effect on postpartum health and performance.

Blood and Urine Parameters

Alteration of DCAD is reflected in both blood and urine pH and metabolites. Tucker et al., (1988) observed that as DCAD increases from -10 to 20 meq/100g DM, blood pH increased linearly. These results support the alkalosis induced when feeding high DCAD levels. Findings by West et al. (1991) supported prior research that blood pH and bicarbonate were lowest in cows being fed diets with a -11 meq/100g DM DCAD. Block (1984) initially indicated lowest Ca and P concentrations in cows fed 33 meq/100g DM compared with -12 meq/100g DM DCAD. Further research reported that feeding negative DCAD increased total plasma Ca (Siciliano-Jones et al., 2008) and ionized Ca (iCa) (Moore et. al., 2000).

Vagoni and Otzel (1998) reported reductions in urinary pH and bicarbonate excretion in cow fed negative DCAD diets. Jardon (1995) stated systemic metabolic acidosis was achieved when urine pH was lower than 7.0. Moore et. al. (2000) supported these findings by observing that urine pH decreased from 7.3 to 6.0 when feeding 0 compared with -15 meq/100g DM DCAD diets. Charbonneau et. al. (2006) demonstrated that decreasing pH lower than 6.8 did not result in additional reduction in incidence of hypocalcemia. Leno et al. (2017) reported that maintaining a urine pH between 5.5 and 6.0 results in additional benefits in Ca status. Feeding anionic salts to achieve a negative DCAD diets has been reported to result in greater anion excretion (Cl and S) compared to

cations (Na and K). Tucker et al. (1988) indicated that urinary mineral excretion is the more responsive to DCAD manipulation than plasma mineral concentrations. Urine excretion of Ca increased from 0.5 g/d to 5-6 g/d in cows fed negative DCAD diets (Goff, 2014a), which indicates the mechanism of action being to stimulate increased Ca transport prior to calving.

Acid-Base Status in Calves

Respiratory and metabolic acidosis are key measures of the metabolic state of a calf. There has been a strong correlation of both measures with the incidence of calf mortality (Szenci, 1985). In normal situations, calves will be in a brief hypoxic state during the birthing process. This often lowers pH and pCO_2 which results in a mild acidosis state (Garry, 1993).

Research has been limited in terms of examining the effects of feeding a negative DCAD diet pre-partum to the dam on the acid-base balance of their calves. Joyce and Sanchez (1994) and Guy et al. (1996) reported that calves born to cows fed anionic diets were affected by respiratory or metabolic acidosis which may affect the apparent efficiency of absorption of IgG from colostrum. Conversely, Tucker et al. (1992) reported no effect on acid-base status or plasma mineral concentration in calves born to cows fed diets with either -30 or +90 meq/kg DM DCAD. These results are consistent with those of Quigley and Drewry (1998) who reported that calves born to dams fed a negative DCAD diet had higher rates of respiratory acidosis, which may impair the acquisition of passive immunity via colostrum. Conversely, results from Morrill et. al. 2010 reported similar IgG concentration and apparent efficiency of absorption in calves born to dams fed anionic salts prepartum compared to calves born to dams not fed anionic salts

prepartum. However, it has been suggested that research is needed to better define the relationship between the anion content of the diet and the incidence of respiratory or metabolic acidosis in neonates and absorption of IgG for passive immunity.

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CHAPTER 3

EFFECTS OF VARYING DCAD AND CALCIUM CONCENTATION IN PREPARTUM DIETS ON CALCUIM STATUS, HEALTH AND PERFORMANCE IN MULTIPARIOUS COWS[1]

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ABSTRACT

Between January and October 2016, eighty-two multiparous Holstein cows were enrolled 28 d prior to calving and remained on trial through 63 days in milk (DIM). Cows were assigned to 1 of 4 dietary treatments in a randomized block design experiment with a 2 X 2 factorial arrangement of treatments. Dietary treatments provided two dietary cation-anion concentrations (DCAD): -22 (NEG) or -3 mEq/100 g DM (NEU); and two dietary Ca concentrations: 1.3% or 1.8% of DM. Cows were individually fed and dry matter intake (DMI) recorded daily. Urine and blood samples were collected once weekly prior to calving. Additional blood samples were collected 2 d prior to expected calving date, immediately after calving, and on 1, 2, and 3 DIM. Urine pH was lower for NEG compared with NEU and tended to be lower for 1.8% compared with 1.3% Ca. Prepartum concentrations of urine Ca were higher whereas urine creatinine and urine K were lower for NEG compared with NEU. Fractional excretion of Ca and Mg was greater for NEG than NEU. Plasma bicarbonate was lower and plasma total P was higher for NEG compared with NEU. Plasma total P and blood urea nitrogen (BUN) to creatinine ratio was higher for 1.3% compared with 1.8% Ca prepartum. From 0 to 3 DIM ketone and ratio of ionized Ca/ionized Mg were higher for NEG compared with NEU whereas total protein, albumin, total Mg, ionized Mg were higher for NEU compared with NEG. Interactions of DCAD and DIM were observed for plasma total Mg and ionized Mg, as concentrations were similar on 0 and 3 days in milk but were higher for NEU on d 1 and 2. An interaction of Ca and DIM was observed for plasma total Ca due to higher concentration at d 1 for 1.8%, whereas concentrations were not different at d 2 or 3. After calving, cows were milked 3 times daily, yield recorded at each milking and samples

collected once weekly for analysis of components. Body weight and body condition score were recorded 21 d prior to calving, and 0, 21, and 63 DIM. No interactions were observed between DCAD and Ca for any variables measured. No differences were observed in body weight or body condition score due to DCAD or Ca. Prepartum dry matter intake (DMI) was lower for NEG compared with NEU and lower for 1.8% compared with 1.3% Ca. Postpartum DMI was not different among treatments. Daily milk yield was not different among treatments but an interaction was observed for DCAD and DIM due to higher milk yield after 45 DIM for NEG compared with NEU. No differences were observed in milk component percentage or yield among treatments. These results suggest that feeding -22 mEq/100 g DM DCAD prepartum alters plasma and urine mineral concentrations, compared to feeding -3 mEq/100 g DM DCAD and also supports increased milk yield after 45 DIM. Feeding 1.8% Ca prepartum improved plasma Ca at 1 DIM. Feeding -22 mEq/100 g DM compared with -3 mEq/100 g DM DCAD, or 1.8% Ca compared to 1.3% Ca reduced DMI prepartum.

KEY WORDS: DCAD, Ca, milk yield, milk composition

INTRODUCTION

During the transition period, cows are at highest risk for metabolic diseases and health complications. Hypocalcemia often arises from the increase Ca demands paired with the delay in Ca absorption and mobilization after calving, resulting in low blood Ca concentrations. Cow with hypocalcemia have lower milk production and increased risk of other health disorders like mastitis, displaced abomasum, and retained placenta (Curtis et. al. 1985). Block (1984) and Oetzel (1991) determined that feeding anions to create a negative DCAD diet prepartum effectively altered Ca homeostasis to decrease the incidence of milk fever and improve production and health compared to positive DCAD.

Proper Ca supplementation during the prepartum period aids in meeting requirements for the cow and calf and also prepares mechanisms needed to meet the extreme Ca demand immediately postpartum. Cows fed negative DCAD diets prepartum have a Ca requirement of 1.0-1.5% (NRC, 2001). However, inclusion within the NRC recommendation to further alter Ca homeostasis has been debated by researchers and consultants given the higher milk yield of modern dairy cows. Mixed results have been reported on intake and blood Ca concentration due on inclusion rates. Some have observed that inclusion rates above 1.0% decrease DMI (Miller, 1983) while others have fed rates up to 1.8% with no effects on DMI (Beede & Shearer, 1991). Chan et. al 2006 reported no change in blood Ca concentration when diets were supplemented 0.99% compared with 1.5% calcium in diets with a -6 meq/100g DCAD. Oba et. al. (2011) demonstrated improved blood Ca concentration feeding 0.9% compared to 0.3%. Based on these results, 1.0% Ca prepartum with a negative DCAD diet seems sufficient for maintaining blood Ca during the immediate postpartum period. The objective of this trial

was to determine the effect of feeding a neutral or fully acidified diet varying in Ca concentration during the prepartum period on plasma Ca status, health and postpartum performance.

MATERIALS AND METHODS

Animals and Feeding Management

All methods were reviewed and approved by the University of Georgia Animal Care and Use Committee prior to conducting the trial. Eighty-two multiparous Holstein cows were enrolled in a randomized block design experiment from January to October 2016. Cows were assigned to 1 of 4 dietary treatments in a randomized block design experiment with a 2 X 2 factorial arrangement of treatments. Enrollment was 28 ± 4 d prepartum and conclusion at 63 DIM. Cows were blocked on expected calving date and parity. Dietary treatments provided two dietary cation-anion concentrations (DCAD): -22 (NEG) or -3 mEq/100 g DM (NEU); and two dietary Ca concentrations: 1.3% or 1.8% of DM, providing a total of four experimental diets: NEU-1.3 (n=20), NEU-1.8 (n=21), NEG-1.3 (n=20) and NEG-1.8, referring to the level of DCAD and Ca supplementation.

Animals were trained to eat behind Calan gates (American Calan, Northwood, NH) prior to initiation of the trial. Cows were housed in a 4-row sand bedded freestall barn with equipped with fans and misters for evaporative cooling. Cows had with free access to a dry lot 24 h/d. Cows were fed once daily and had access to water at all times. Experimental diets and a common lactating diet were formulated to meet or exceed NRC requirements with 5% refusals. Base ingredients were initially mixed using a Khun mixer wagon to form a base mix. The base mix was then added to premix supplements and

concentrate mix as outlined in Table 3.1 and individually fed using a DataRanger. Amounts offered and refused were measured and recorded daily. Feed was manually pushed up by hand two times daily.

Sampling

Samples of individual feed ingredients, experimental diets, and the common lactating diet were collected 3 times each week. Dry matter was determined using a forced air drying oven set at 55°C for 48 h. Samples were ground to pass through a 6mm screen and then a 2mm screen using a Wiley mill (Thomas Scientific, Swedesboro, NJ) and composited by week. Samples were analyzed for protein (LECO FP-5258 Nitrogen Analyzer, St. Joseph, MO), ADF (AOAC International, 2000), NDF adjusted for ash (Van Soest et al., 1991) , ether extract (AOAC International, 2000), and minerals (AOAC International, 2000). Body weight measurements were recorded on 3 consecutive days at the beginning of the trial and averaged to determine initial BW. A BCS was assigned by one individual as described by Wildman et al. (1982) to maintain consistency throughout the trial. Body weight and BCS was measured again immediately post calving and at 21 and 63 DIM.

Blood samples were collected from the coccygeal vessel once per week at -3, -2, -1 wk, and -2 d prior to expected calving date, immediately after calving (D0), at 1 (D1), 2 (D2), 3 (D3) DIM, and 1 and 2 wk postcalving. Blood was analyzed for selected metabolites: total protein, albumin, globulin, urea N, creatinine, total bilirubin, glucose, AST, CK, GGT, Ca, Mg, Na, K, Cl, bicarbonate, anion gap, (Siemens Medical Solutions USA, Inc., Malvern, PA), iCa, and ionized Mg (iMg) (pHOx Ultra, Nova Biomedical, Waltham, MA). Serum was separated from the second sample for analysis of NEFA

concentrations (Waco Chemicals USA Inc., Richmond, VA). Concentrations of beta-hydroxybutyrate (BHB) concentrations were determined using a Nova Max Ketone Strop and a Nova Max Plus reader (Nova Biomedical, Waltham, WA). Pre-partum urine samples were collected once per week prior to calving, -3 wk for pH analysis and -2 and -1 wk for pH analysis of minerals (Ca, Mg, Na, K, and Cl) and creatinine concentrations as described previously. Additionally, pH was assessed using urine strips at the beginning of the trial to ensure proper acidification level via DCAD was being met.

After calving, cows were milked 3 times per day at 0800, 1600, and 2400 h. Milk weights were electronically recorded each milking (Alpro, Deleval , Kansas City, MO) and summed daily. Milk samples were collected once each week at three consecutive milking. Samples were refrigerated and shipped for next day delivery to Dairy One Cooperative (Ithaca, NY) for analysis of fat, protein, lactose, MUN and SCC using a Foss 400 instrument (Foss North America, Eden Prairie, MN) as described by AOAC International (2000).

Statistical Analysis

Data were subjected to repeated analysis of variance using PROC MIXED procedures of SAS (SAS Institute, Cary, N. C.). The model included block, DCAD treatment, Ca treatment, interaction of DCAD and Ca, week and the interactions of week and treatments. The first-order autoregressive covariance structure was used according to Littell et al. (1998). Previous lactation ME milk, fat and protein yield along with average lactation SCC were used as covariates in the analysis of production data. Significance was declared at $P < 0.05$ and a trend when $P > 0.05$ and < 0.1.

RESULTS

Diet Composition

Chemical composition of experimental diets and the common postpartum diet is presented in Table 3.2. The DCAD [(Na + K) – (Cl + S)] was -2.37 and -21.18 mEq/100 g DM for NEU and NEG, respectively. The calcium inclusion rate was 1.3 and 1.8% DM.

Pre-and Postpartum Body Weight and Intake

No differences ($P > 0.10$) were observed due to DCAD, Ca, or interaction of treatments on BW or BCS for both the pre-and postpartum periods. Prepartum BW and BCS averaged 754.8 kg and 3.36, respectively. Postpartum body weight at calving, 21 and 63 DIM averaged 707.2, 654.9, and 638.3 kg, respectively. Corresponding BCS for at calving, 21 and 63 DIM were 3.18, 3.03, and 2.92, respectively.

Prepartum DMI was effected by both DCAD (P=0.0096) and Ca concentration (P=0.0038). These results are presented in Figure 3.1. Cows fed NEG had lower DMI compared with NEU; 14.2 kg/d and 16.0 kg/d, respectively. Cows fed 1.3% calcium had higher DMI compared to 1.8% calcium; 16.2 kg/d and 14.0 kg/d, respectively. No differences ($P > 0.10$) were observed in postpartum DMI due to DCAD, Ca, or their interaction which averaged 23.1 kg/d (Table 3.3).

Prepartum Blood and Urine Metabolites

Selected blood and urine metabolites and urinary fractional excretion of minerals are presented in Table 3.4. Cows fed NEG had lower concentrations of Na ($P = 0.0646$) and bicarbonate ($P = 0.0011$) whereas Cl ($P = 0.0519$) was higher. Increasing supplemental Ca from 1.3 to 1.8% reduced concentrations of P and BUN: creatinine ratio which averaged 7.35 mg/dl and 31.89 and 6.76 mg/dl and 27.81,

respectively. Interactions of Ca and DIM were observed for albumin (P = 0.0297), Na (P = 0.0363), and urinary creatinine (P = 0.0500). Albumin was generally lower for 1.8% compared to 1.3% Ca during the prepartum period, especially -7 and -2 d prior to calving (Figure 3.2). Plasma Na increased throughout the prepartum period and was generally higher for 1.3% compared to 1.8% Ca (Figure 3.3). Urinary creatinine increased with 1.8% Ca whereas for 1.3% Ca it decreased during the prepartum period (Figure 3.4) An interaction of DCAD and day was observed for plasma creatinine kinase (P = 0.0031) and bicarbonate (P = 0.0599). Creatinine kinase was lower at -21 d, then higher at -14 d, and not different at -7 or -2 d prepartum for NEG compared with NEU (Figure 3.5). Bicarbonate tended to be lower for NEG compared to NEU throughout the prepartum period, but generally increased at -7 and -2 d prepartum for both groups (Figure 3.6).

Urine pH was lower (P < 0.0001) for cows fed NEG compared to NEU and averaged 6.03 and 7.05, respectively (Table 3.4). Urine pH also tended to be lower (P = 0.06) for 1.8% Ca compared to 1.3% Ca; 6.75 and 6.33, respectively. Feeding NEG compared to NEU resulted in higher (P = 0.02) concentration of Ca (42.31 and 29.22 mg/dl, respectively) and lower (P < 0.01) concentration of creatinine (62.81 and 131.01 mg/dl, respectively) and K (92.75 and 167.20 mg/dl, respectively) in the urine. Fractional excretion of Ca (P = 0.0156) and Cl (P < 0.0001) were higher for NEG compared to NEU, while excretion of Na (P = 0.0262) and K (P = 0.0375) was higher for 1.3% compared to 1.8% Ca.

Postpartum Blood Metabolites

Postpartum blood metabolites from 0-3 DIM are shown in Table 3.5 and metabolites from 7 and 14 DIM are shown in Table 3.6. Ketone concentrations and ratio

of iCa/iMg were higher (P < 0.05) for NEG compared with NEU whereas total protein, albumin, total Mg, iMg were higher (*P* < 0.05) for NEU compared with NEG. No differences (*P* > 0.01) were observed between 1.3% and 1.8% Ca from 0 to 3 DIM. Interactions of DCAD and DIM (*P* < 0.05) were observed for plasma total Mg (Figure 3.7) and iMg (Figure 3.8) which were similar on 0 and 3 DIM but were higher for NEU on 1 and 2 DIM compared to NEG. An interaction was observed for Ca and DIM for plasma total Ca (*P* = 0.0030) due to higher concentrations at 1 DIM for 1.8%, whereas concentrations were not different at 2 or 3 DIM (Figure 3.9). An interaction was observed for lactation and DIM for total plasma Ca, total plasma Mg, and ratio of iCa/iMg (*P* < 0.05). Total plasma Ca was higher 0 through 3 DIM for 2nd lactation compared to 3+ lactation (Figure 3.10). Total plasma Mg was higher for 3+ lactation 0 and 1 DIM compared to 2nd lactation but then were similar 2 and 3 DIM (Figure 3.11). Ratio of iCa/iMgwas higher for 2nd lactation 0 and 1 DIM compared to 3rd lactation but then were similar 2 and 3 d postpartum (Figure 3.12).

Milk Yield and Composition

No differences were observed in daily milk yield among treatments, but there was an interaction observed for DCAD and DIM (*P* = 0.0125), shown in Figure 3.13. Higher milk yield was observed for cows fed NEG compared with NEU specifically from 45 DIM through the end of the trial. Cows fed NEG compared to NEU averaged 2.6 kg/d greater during this period. No differences (*P* > 0.10) were observed in milk component percent or yield due to treatment (Table 3.3).

Health Performance

The number of health events and hypocalcemia incidence rates are listed in Table 3.6. The least number of cows having health events were in treatment group NEU-1.3, while the least number of multiple health events were in the cows from treatment group NEG-1.3. Subclincial hypocalcemia rates (< 8.6 mg/dL) were reduced in cows fed NEU and 1.8% Ca prepartum d 1 postpartum while NEG and 1.8% Ca showed greatest recovery by d 14 compared to NEU. Clinical hypocalcemia rates (< 8.0 mg/dL) were reduced in cows fed NEG and 1.8% Ca prepartum d 1 postpartum while NEG showed complete recovery compared to NEU which still had 10.53% incidence of clinical hypocalcemia by d 14.

DISCUSSION

Pre- and Postpartum Body Weight and Intake

No differences were observed in pre- or postpartum BW or BCS for among treatments. Prepartum DMI was effected by both DCAD and Ca concentration, where increased acidification or higher Ca supplementation decreased DMI. This is consistent with the observations reported by Otezel (1993), Moore et. al. (2000), and Charbonneau et. al. (2006) where anionic diets decreased palatability and reduced prepartum DMI. A similar effect was observed with higher inclusion of Ca (Miller, 1983) who concluded that the additional mineral content decreased palatability. Treatment NEG-1.8 combined both parameters and we observed the lowest DMI from this group. Despite significant effects on DMI, cows in all treatment groups were eating above recommended values (NRC, 2001). No difference was observed in postpartum DMI which is in contrast with

the reports of DeGroot et. al. (2010) and Leno et. al. (2017), where prepartum anionic diets improved postpartum DMI.

Prepartum Urine and Blood Metabolites

Urine pH for was lower for NEG compared to NEU. Our findings are consistent with previous research that state that increasing acidification to create a mild metabolic acidosis will decrease prepartum urine pH (Jardon, 1995; Vagoni and Otzel, 1998; Moore et. al. 2000; Charbonneau et. al. 2006). In addition, we observed a trend for lower urine pH for 1.8% compared to 1.3% Ca. Calcium is a cation, therefore would increase a metabolic alkalosis and should raise pH. The reason for the lower pH is not apparent as there were no significant differences in concentrations of anions in the blood or urine. Higher urinary concentrations of Ca were observed for NEG compared to NEU in addition to greater fractional excretion of Ca and Cl. These results support observations of Leno et. al. 2017 where Ca excretion increased as urine pH decreased. Animate, the anionic supplement used to achieve negative DCAD contains high concentrations of Cl which contributed to the increased Cl concentrations observed in plasma as well as the higher fractional excretion.

Postpartum Blood Metabolites

The most significant observation was the interaction of Ca and DIM on total plasma Ca. Total plasma Ca was higher at 1 DIM for cows fed 1.8% compared with 1.3% Ca prepartum. This timepoint is important because it falls within the 12-24 h that hypocalcemia is most likely to occur. Shire and Beede (2013) suggested that 1.0% Ca prepartum is sufficient to maintain blood Ca. Based off our results, we would agree that optimum Ca supplementation is between 1.0 and 1.3% during the prepartum period.

Multiparous cows that were entering their second lactation generally had improved Ca status compared to the cows entering their third or subsequent lactation. Parity is the key factor that will predispose cows to developing hypocalcemia (van Mosel et. al., 1993).

Both total and ionized Mg concentrations were 3 and 5% higher for cows fed NEU compared with NEG, respectively. The concentration of Mg in the diet was slightly higher for NEU compared with NEG (0.51 and 0.54 % of DM for NEU and NEG, respectively). Due to decreased DMI immediately prepartum the amount available for absorption for the NEG compared to NEU was lower which could be the reasoning behind the differences we observed. There may also be minor differences in homeostasis control of Mg, specifically in terms of PTH release. Minor decreases in Mg concentration increases PTH secretion, suggesting the minor decrease between NEG and NEU could be due to increased PTH secretion to maintain Ca homeostasis. However, Mg excretion postpartum or hormone levels were not measured in the current trial in order to support these possibilities.

Milk Yield and Composition

Milk yield did not differ among treatments but was improved from 45 through 63 DIM for cows fed NEG compared to NEU prepartum. Milk yield was 2.6 kg/d greater for NEG compared with NEU during this time period. These results are consistent with the observations of DeGroot et. al. (2010) and Leno et. al. (2017); however, they reported increases earlier in lactation than we observed in our current trial. No differences were observed in milk component yield or percentage which is consistent with prior research from Moore et. al, (2000) and Ramos-Nieves et. al. (2009).

Health Performance

Ketosis, retained placenta, and mastitis occurred most often in all treatment groups. The cut off for clinical hypocalcemia used was < 8.0 mg/dL while the cut off for subclinical hypocalcemia used was < 8.6 mg/dL. Based off the health event incidences, subclinical hypocalcemic cows had decreased gut function and reduced immune function like what has been observed previously (Martinez et. al. 2014; Kimura et. al., 2006), resulting in common disease. Subclinial hypocalcemia rates were numerically higher for NEG and 1.3% Ca at 1 DIM but the greatest recovery made was in cows fed NEG prepartum. A larger proportion of cows calved during periods of heat stress which may have been a contributing factor in health status as the trial ran though the peak heat stress months.

CONCLUSIONS

Results of this trial indicate that feeding fully acidified diet (NEG) as defined by maintaining a urine pH of 5.5 to 6.0 alters plasma and urine mineral concentrations to successfully improve plasma Ca concentrations by 3 DIM and increased milk yield from 45 through 63 DIM, compared to nonacidified diet (NEU). Feeding 1.8% Ca prepartum improved total plasma Ca at 1 DIM only compared to 1.3% Ca, but reduced prepartum DMI. Postpartum DMI and milk composition was not different among Ca treatments.

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Table 3.1. Ingredient composition of diets differing in DCAD and calcium concentration prepartum and common postpartum diet (% of DM).

DCAD	NEU[1]		NEG		
Ca	1.3%	1.8%	1.3%	1.8%	Postpartum
Ingredient					
Tift 85	15.00	15.00	15.00	15.00	4.32
Corn silage	43.33	43.33	43.33	43.33	38.04
Brewers grains, wet	9.17	9.17	9.17	9.17	10.38
QLF-dairy 28 molasses	8.33	8.33	8.33	8.33	4.32
Ground corn	2.50	2.50	2.50	2.50	12.54
Soybean hulls					5.19
Corn gluten feed					4.76
Citrus pulp					5.19
Cottonseed, whole					2.16
Cottonseed hulls[2]	4.22	2.98	1.83	0.60	
Soybean meal, 48%	5.00	5.00	5.00	5.00	1.30
AminoPlus[3]	5.00	5.00	5.00	5.00	2.59
Prolak[4]	3.33	3.33	3.33	3.33	3.03
Urea					0.17
Mepron[5]					0.05
AjiPro-L[6]					0.26
Megalac[7]					1.73
Omnigen AF[8]	0.38	0.38	0.38	0.38	0.20
Procreatin-7[9]	0.03	0.03	0.03	0.03	0.01
Rumensin	0.33	0.33	0.33	0.33	0.26
Salt	0.13	0.13	0.13	0.13	0.35
Sodium sequinate					1.04
Calcium carbonate[2]	1.27	2.50	1.17	2.40	0.86
Magnesium oxide[2]	0.18	0.18			0.35
Animate[2,10]	1.50	1.50	4.17	4.17	
DCAD Plus[11]					0.52
Dynamate[12]					0.09
TM-Vit premix	0.30	0.30	0.30	0.30	0.30

[1] NEG= negative DCAD, NEU= neutral DCAD

[2] Ingredients for treatment premix added individually to mixer prior to feeding

[3] Ruminally protected soybean meal, Ag Processing, Inc. Omaha, NE

[4] Marine and animal rumen undegradable protein supplement, H. J. Baker & Bros., Inc. Westport, CT

[5] Ruminally protected methionine, Envonik, Theodore, AL

[6] Ruminally protected lysine, Ajinomoto, Itasca, IL

[7] Rumen bypass fat, Arm & Hammer Animal Nutrition, Princeton, NJ

[8] Immune stimulant, Phibro Animal Health, Corp., Teaneck, NJ

[9] Yeast supplement, Phibro Animal Health, Corp., Teaneck, NJ

[10] Anionic mineral supplement, Phibro Animal Health, Corp., Teaneck, NJ

[11] Potassium and magnesium sulfate mineral supplement, The Mosaic Company, Plymouth, MN

[12] Potassium mineral supplement, Arm & Hammer Animal Nutrition, Princeton, NJ

Table 3.2. Chemical composition of diets differing in DCAD and calcium concentration prepartum and common postpartum diet (Mean ± SD).

DCAD	Neutral		Negative		
Ca	1.3	1.8	1.3	1.8	Postpartum
DM, %	52.5 ± 4.4	52.7 ± 3.8	53.2 ± 3.9	53.8 ± 3.9	54.7 ± 3.7
	---------- % of DM ----------				
CP	21.4 ± 1.3	21.2 ± 1.5	21.8 ± 1.0	21.3 ± 1.3	17.4 ± 1.4
ADF	17.9 ± 0.6	17.8 ± 1.8	18.9 ± 1.5	18.1 ± 0.5	18.3 ± 2.3
NDF	38.1 ± 1.7	37.3 ± 1.2	39.9 ± 1.2	40.1 ± 1.2	36.5 ± 3.0
EE[1]	2.9 ± 0.6	2.3 ± 0.2	2.9 ± 0.5	2.7 ± 0.6	5.6 ± 1.6
Ash	8.5 ± 0.4	9.7 ± 0.4	8.7 ± 0.4	9.9 ± 0.4	7.5 ± 0.5
Ca	1.31 ± 0.07	1.81 ± 0.07	1.30 ± 0.07	1.80 ± 0.07	1.05 ± 0.08
P	0.41 ± 0.01	0.40 ± 0.01	0.42 ± 0.01	0.42 ± 0.01	0.42 ± 0.02
Mg	0.50 ± 0.06	0.51 ± 0.06	0.53 ± 0.01	0.54 ± 0.06	0.38 ± 0.02
K	1.60 ± 0.08	1.59 ± 0.08	1.60 ± 0.08	1.59 ± 0.08	1.46 ± 0.05
Na	0.11 ± 0.04	0.11 ± 0.04	0.12 ± 0.04	0.12 ± 0.04	0.42 ± 0.07
S	0.42 ± 0.03	0.42 ± 0.03	0.57 ± 0.12	0.57 ± 0.03	0.26 ± 0.01
Cl	0.77 ± 0.09	0.77 ± 0.09	1.11 ± 0.09	1.11 ± 0.09	0.41 ± 0.03
DCAD[2]	-2.30 ± 3.23	-2.45 ± 3.23	-21.11 ± 3.23	-21.26 ± 3.23	27.10 ± 2.34

[1] EE= crude fat

[2] Calculated as DCAD = (Na + K) – (Cl + S) mEq/100g DM

Table 3.3. Dry matter intake, milk yield and composition of cows fed diets differing in prepartum DCAD and Ca concentrations

DCAD	NEU		NEG			P		
Ca, %	1.3%	1.8%	1.3%	1.8%	SE	DCAD	Ca	DCAD x CA
Prepartum DMI, kg/d	17.3	14.8	15.1	13.0	0.7	0.0096	0.0038	0.6042
% of BW	2.06	1.83	2.30	1.96	0.09	0.0051	0.0046	0.5406
Postpartum DMI, kg/d	22.5	23.0	24.0	23.0	0.9	0.4288	0.7628	0.4435
% of BW	3.31	3.31	3.72	3.46	0.16	0.2544	0.8304	0.1486
Milk, kg/d	39.7	38.9	40.6	41.0	1.6	0.6040	0.8787	0.8777
Fat, %	4.32	4.22	4.25	4.04	0.14	0.3980	0.2765	0.7117
Fat, kg/d	1.72	1.64	1.73	1.66	0.06	0.5883	0.2303	0.8268
Protein, %	2.68	2.64	2.68	2.65	0.04	0.9028	0.4506	0.8741
Protein, kg/d	1.07	1.03	1.09	1.09	0.04	0.3197	0.6277	0.5640
Lactose, %	4.62	4.61	4.57	4.66	0.05	0.9675	0.4059	0.3609
Lactose, kg/d	1.84	1.79	1.86	1.91	0.08	0.4093	0.8926	0.4678
SNF, %	8.33	8.30	8.33	8.32	0.06	0.9084	0.7334	0.8455
SNF, kg/d	3.31	3.23	3.38	3.41	0.13	0.3502	0.8565	0.6221
ECM, kg/d	43.3	41.8	44.0	43.2	1.6	0.3347	0.4225	0.9592
MUN, mg/dL	10.06	9.56	8.90	9.56	0.37	0.1194	0.8308	0.1360
SCC, cells x 1,000/ml	383	356	389	250	120	0.6743	0.4922	0.6542
Efficiency, ECM/DMI	1.81	1.79	1.74	1.83	0.07	0.7985	0.5751	0.4536

Table 3.4. Prepartum (-28 through -2 d) blood and urine metabolites and fractional excretion of cows fed diets differing in prepartum DCAD and Ca concentrations

DCAD	NEU		NEG			P		
Ca	1.3%	1.8%	1.3%	1.8%	SE	DCAD	Ca	DCAD x Ca
Plasma								
Ketone	0.73	0.73	0.76	0.75	0.14	0.6223	0.8804	0.9134
Total protein, g/dL	7.02	6.97	6.86	6.78	0.12	0.1611	0.6228	0.9010
Albumin, g/dL	3.48	3.43	3.42	3.38	0.04	0.1102	0.2191	0.8641
Globulin, g/dL	3.53	3.54	3.44	3.41	0.12	0.3437	0.9091	0.8619
A/G ratio	1.05	1.00	1.02	1.01	0.03	0.8345	0.3490	0.5333
BUN, mg/dL	22.98	19.47	21.57	20.13	0.85	0.6623	0.0056	0.2317
Creatinine, mg/dL	0.70	0.73	0.70	0.75	0.03	0.6395	0.1191	0.6417
BUN/Creatinine	32.14	27.97	31.65	27.65	1.62	0.8023	0.0150	0.9603
Total Bilirubin, mg/dL	0.05	0.10	0.08	0.17	0.03	0.1859	0.0552	0.4851
Glucose, mg/dL	55.40	57.46	52.92	54.55	1.64	0.1017	0.2630	0.8968
AST, U/L	69.01	74.99	70.18	77.79	4.16	0.6332	0.1093	0.8454
Creatinine Kinase, U/L	177.62	229.49	193.27	170.74	31.67	0.4942	0.6437	0.2456
GGT, U/L	16.90	14.96	16.76	16.73	1.16	0.4850	0.4043	0.4189
Ca, mg/dL	9.24	8.98	10.78	9.00	1.28	0.5383	0.4293	0.5562
P, mg/dL	7.27	6.66	7.44	6.86	0.17	0.2938	0.0013	0.9402
Mg, mg/dL	2.41	2.35	2.37	2.41	0.04	0.7876	0.7938	0.1210
Na, mEq/L	143.55	142.95	142.90	142.37	0.33	0.0646	0.0896	0.9040
K, mEq/L	5.65	4.81	4.99	5.20	0.37	0.7110	0.3865	0.1562
Cl, mEq/L	100.58	103.62	104.39	105.15	1.36	0.0519	0.1668	0.4028

Bicarbonate, mmol/L	27.38	27.69	25.83	27.81	0.41	0.0011	0.1486	0.6797
Anion Gap, mmol/L	17.26	16.85	17.10	16.81	0.52	0.8428	0.5068	0.9117
iCa, mmol/L	1.27	1.24	1.26	1.26	0.01	0.7996	0.5791	0.2249
iMg, mmol/L	0.60	0.61	0.60	0.62	0.01	0.2585	0.1778	0.5931
iCa/iMg	2.07	2.08	2.09	2.05	0.03	0.8589	0.5998	0.4334
Urine								
Ca, mg/dL	28.41	30.03	44.60	40.02	5.43	0.0207	0.7908	0.5862
Cl, mmol/L	124.99	118.21	131.65	115.26	13.24	0.8908	0.3941	0.7249
Creatinine, mg/dL	103.05	82.44	69.60	56.03	9.43	0.0027	0.0813	0.7187
K, mmol/L	176.34	158.05	141.55	120.47	13.40	0.0096	0.1542	0.9195
Mg, mg/dL	50.55	48.61	31.80	33.31	15.53	0.2840	0.9895	0.9160
Na, mmol/L	29.23	22.15	24.83	22.54	5.67	0.7287	0.4220	0.6915
pH	7.45	6.66	6.05	6.01	0.21	<0.0001	0.0628	0.0973
Fractional Excretion, %								
Ca	0.13	0.14	0.21	0.17	0.02	0.0156	0.7033	0.2757
Mg	15.06	12.68	13.78	13.69	0.89	0.8798	0.1757	0.2027
Na	0.27	0.14	0.30	0.21	0.05	0.3477	0.0262	0.6807
K	34.78	28.77	35.68	32.07	2.24	0.3460	0.0375	0.5937
Cl	1.05	1.01	1.52	1.42	0.09	<0.0001	0.4209	0.7244

Table 3.5. Postpartum blood metabolites (0 – 3 d) of cows fed diets differing in prepartum DCAD and Ca concentrations

DCAD	NEU		NEG			P		
Ca, %	1.3%	1.8%	1.3%	1.8%	SE	DCAD	Ca	DCAD x Ca
Ketone	0.92	0.98	1.06	1.19	0.08	0.0548	0.3108	0.7160
Total protein, g/dL	6.78	6.80	6.46	6.46	0.12	0.0065	0.9619	0.9243
Albumin, g/dL	3.58	3.56	3.51	3.38	0.05	0.0070	0.1253	0.2266
Globulin, g/dL	3.21	3.23	3.30	3.08	0.25	0.8956	0.6864	0.6231
A/G ratio	1.17	1.14	1.20	1.12	0.04	0.8714	0.1704	0.5733
BUN, mg/dL	20.19	16.43	16.41	15.67	1.24	0.0722	0.0744	0.2291
Creatinine, mg/dL	0.87	0.75	0.71	0.76	0.03	0.0342	0.3121	0.0165
BUN/Creatinine	22.47	23.06	25.26	21.93	1.94	0.6698	0.4816	0.3147
Total Bilirubin, mg/dL	0.39	0.46	0.47	0.56	0.06	0.0937	0.1664	0.8440
Glucose, mg/dL	58.79	59.11	57.53	52.70	4.11	0.3542	0.5851	0.5334
AST, U/L	108.04	127.94	127.52	138.38	26.89	0.5800	0.5692	0.8671
Creatinine Kinase, U/L	496.70	685.99	377.66	609.19	329.03	0.7669	0.5246	0.9490
GGT, U/L	21.76	13.71	15.02	15.79	2.73	0.3952	0.8482	0.1102
Ca, mg/dL	8.12	8.24	7.98	8.23	0.15	0.6035	0.2375	0.6672
P, mg/dL	5.75	5.97	5.59	5.67	0.30	0.4492	0.6019	0.8139
Mg, mg/dL	2.45	2.43	2.31	2.30	0.06	0.0235	0.7229	0.9003
Na, mEq/L	142.98	142.51	143.42	142.98	0.38	0.2342	0.2314	0.9680
K, mEq/L	5.69	5.97	5.94	5.80	0.22	0.8136	0.7919	0.3173
Cl, mEq/L	103.34	102.58	103.58	103.26	0.45	0.3074	0.2343	0.6273
Bicarbonate, mmol/L	29.60	30.19	29.88	29.34	0.46	0.5356	0.9574	0.2215
Anion Gap, mmol/L	15.72	0.16	15.95	16.21	0.43	0.4303	0.7227	0.7976
iCa, mmol/L	1.15	1.16	1.14	1.17	0.01	0.6868	0.2254	0.4201
iMg, mmol/L	0.62	0.61	0.06	0.59	0.01	0.0196	0.7141	0.5115
iCa/iMg	1.90	1.92	2.01	2.02	0.05	0.0464	0.7201	0.8777

Table 3.6. Postpartum blood metabolites (7 and 14 d) of cows fed diets differing in prepartum DCAD and Ca concentrations

DCAD	NEU		NEG			P		
Ca, %	1.3%	1.8%	1.3%	1.8%	SE	DCAD	Ca	DCAD x Ca
Ketone	1.67	1.30	1.56	1.80	0.20	0.3322	0.7505	0.1383
Total protein, g/dL	6.93	7.03	6.72	6.61	0.13	0.0162	0.9418	0.4202
Albumin, g/dL	3.48	3.40	3.37	3.25	0.06	0.0340	0.0841	0.7575
Globulin, g/dL	3.46	3.63	3.35	3.37	0.13	0.1590	0.4602	0.5486
A/G ratio	1.06	0.97	1.28	0.99	0.16	0.4508	0.2383	0.5154
BUN, mg/dL	16.44	13.87	13.04	12.07	1.37	0.0588	0.1941	0.5572
Creatinine, mg/dL	0.93	0.68	0.62	0.64	0.09	0.0481	0.1901	0.1217
BUN/Creatinine	20.04	20.37	23.01	19.19	1.56	0.5656	0.2624	0.1826
Total Bilirubin, mg/dL	0.43	0.37	0.43	0.42	0.08	0.7863	0.6263	0.7598
Glucose, mg/dL	47.96	43.14	41.65	38.85	4.56	0.2440	0.4013	0.8225
AST, U/L	171.76	139.77	150.00	146.35	23.71	0.7470	0.4497	0.5474
Creatinine Kinase, U/L	684.78	296.58	363.96	203.76	235.18	0.3767	0.2422	0.6254
GGT, U/L	23.22	21.07	16.94	18.41	19.53	0.1371	0.9079	0.5437
Ca, mg/dL	9.14	9.16	8.95	9.00	0.12	0.1343	0.7374	0.8668
P, mg/dL	5.36	5.69	5.70	5.41	0.23	0.9021	0.9219	0.1641
Mg, mg/dL	2.27	2.23	2.15	2.18	0.06	0.1787	0.9842	0.5111
Na, mEq/L	138.49	139.31	139.51	140.39	0.53	0.0500	0.1093	0.9475
K, mEq/L	5.54	5.61	6.23	5.21	0.63	0.8151	0.4472	0.3889
Cl, mEq/L	97.12	97.83	95.57	97.68	0.79	0.2775	0.0740	0.3708
Bicarbonate, mmol/L	29.71	30.28	31.34	30.96	0.59	0.0516	0.8697	0.4140
Anion Gap, mmol/L	17.28	16.76	17.90	16.86	0.60	0.5420	0.1901	0.6622
iCa, mmol/L	1.26	1.25	1.23	1.24	0.01	0.1390	0.6930	0.2416
iMg, mmol/L	0.59	0.58	0.56	0.57	0.01	0.1045	0.9191	0.4912
iCa/iMg	2.15	2.18	2.21	2.22	0.05	0.3140	0.7362	0.7704

Table 3.7. Postpartum health events and hypocalcemia incidence of cows fed diets differing in prepartum DCAD and Ca concentrations[1]

DCAD	NEU		NEG	
Ca	1.3%	1.8%	1.3%	1.8%
n=	20	21	20	21
Health Events, # of cows				
Twins	2	1	2	1
Retained placenta	2	4	3	5
Metritis	0	0	2	0
Hypocalcemia	1	1	1	1
Ketosis	5	3	3	5
Displaced abomasum	1	1	0	2
Mastitis	1	3	1	2
Lame	1	2	1	0
Died	1	1	2	1
Total cows with disease	8	10	9	9
Total cows with multiple disease events	5	5	2	5
Subclincial Hypocalcemia, Ca < 8.6 mg/dL				
Days postpartum, % of cows				
0	97.74	80.95	89.47	73.68
1	90.00	42.10	70.00	75.00
2	52.63	47.60	50.00	60.00
3	26.30	28.30	45.00	52.38
7	15.79	15.00	31.56	38.11
14	15.79	10.50	10.50	5.00
Clinical Hypocalcemia, Ca < 8.0 mg/dL				
Days postpartum, % of cows				
0	57.79	42.86	73.68	50.00
1	45.00	26.30	55.00	35.00
2	21.00	23.81	25.00	42.86
3	10.53	23.81	25.00	19.05
7	5.26	5.00	21.05	4.76
14	0.00	10.53	0.00	0.00

[1]Data represent means for individual treatments and were not subjected to statistical analysis.

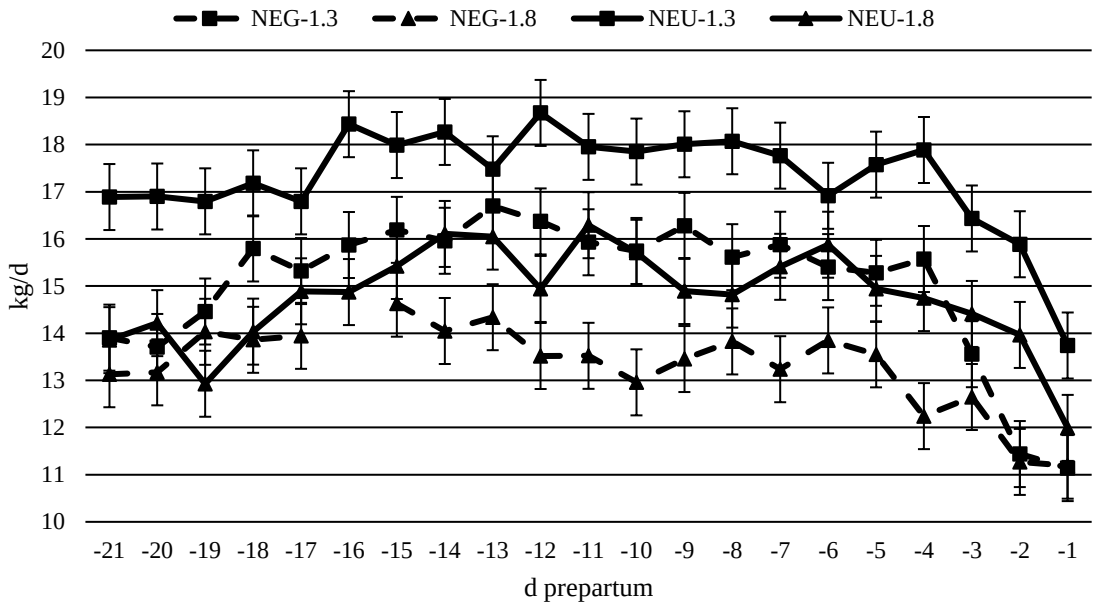

Figure 3.1. Prepartum DMI of cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD, $P = 0.0096$; Ca, $P = 0.0038$)

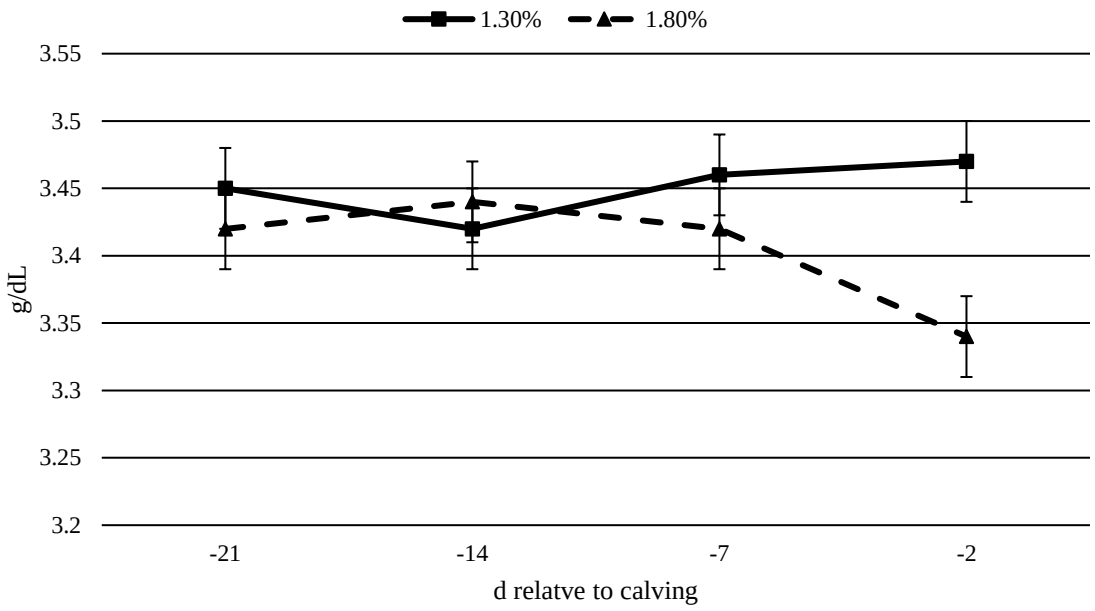

Figure 3.2. Prepartum plasma albumin of cows fed diets differing in prepartum DCAD and Ca concentrations (Ca x d, $P = 0.0297$)

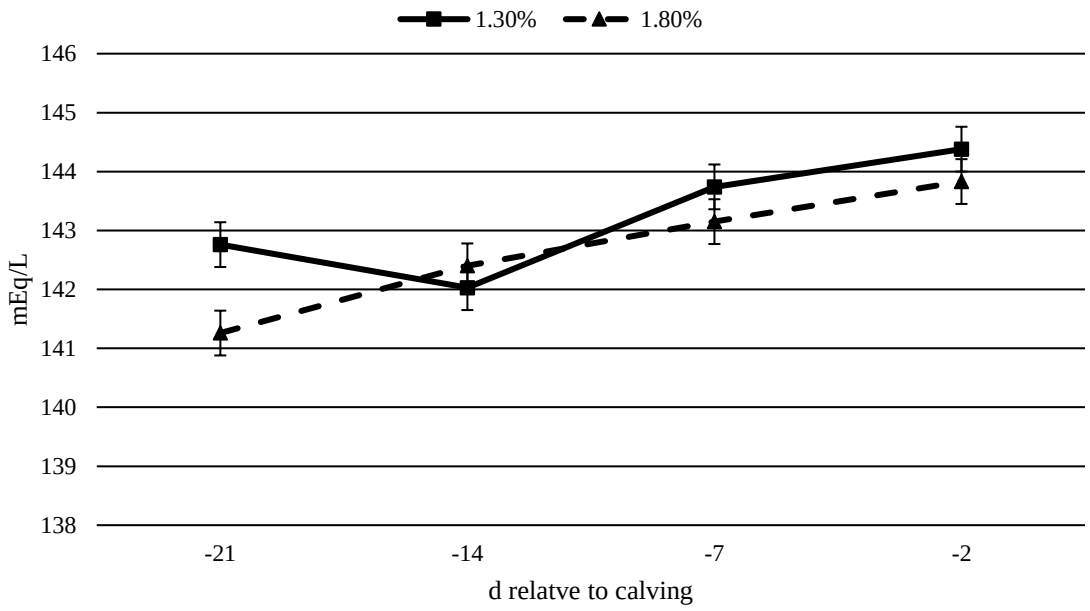

Figure 3.3. Prepartum plasma Na of cows fed diets differing in prepartum DCAD and Ca concentrations (Ca x d, $P = 0.0363$)

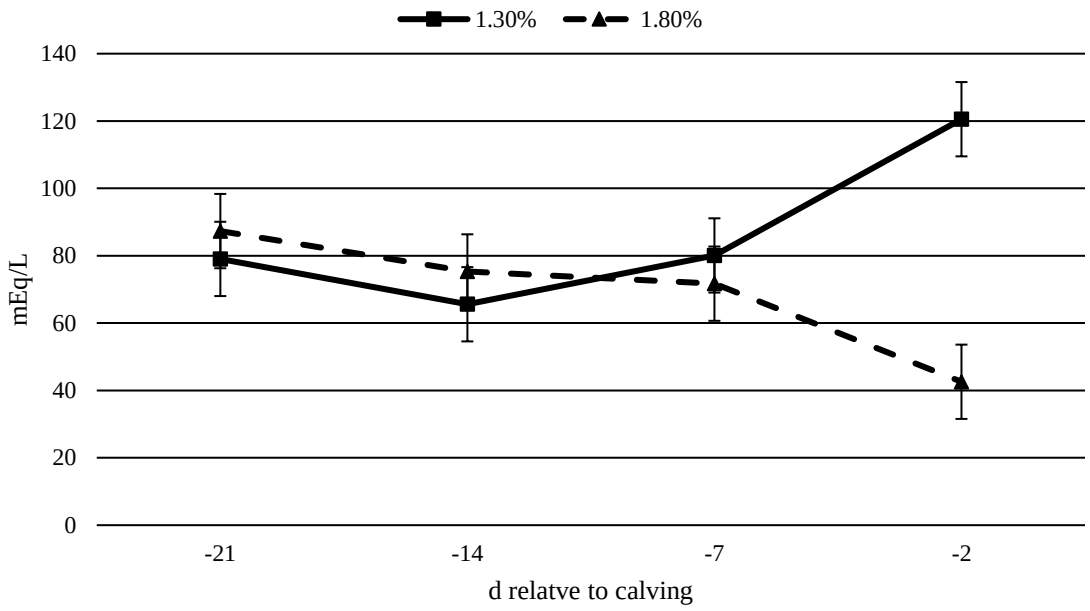

Figure 3.4. Prepartum urinary creatinine of cows fed diets differing in prepartum DCAD and Ca concentrations (Ca x d, $P = 0.0500$)

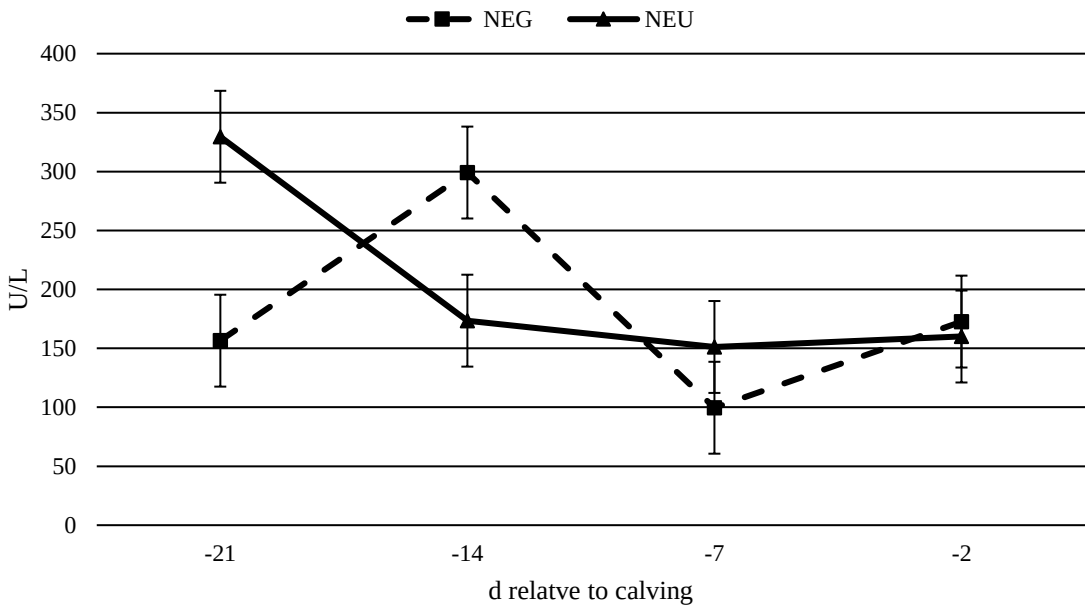

Figure 3.5. Prepartum plasma creatinine kinase of cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD x d, $P = 0.0031$)

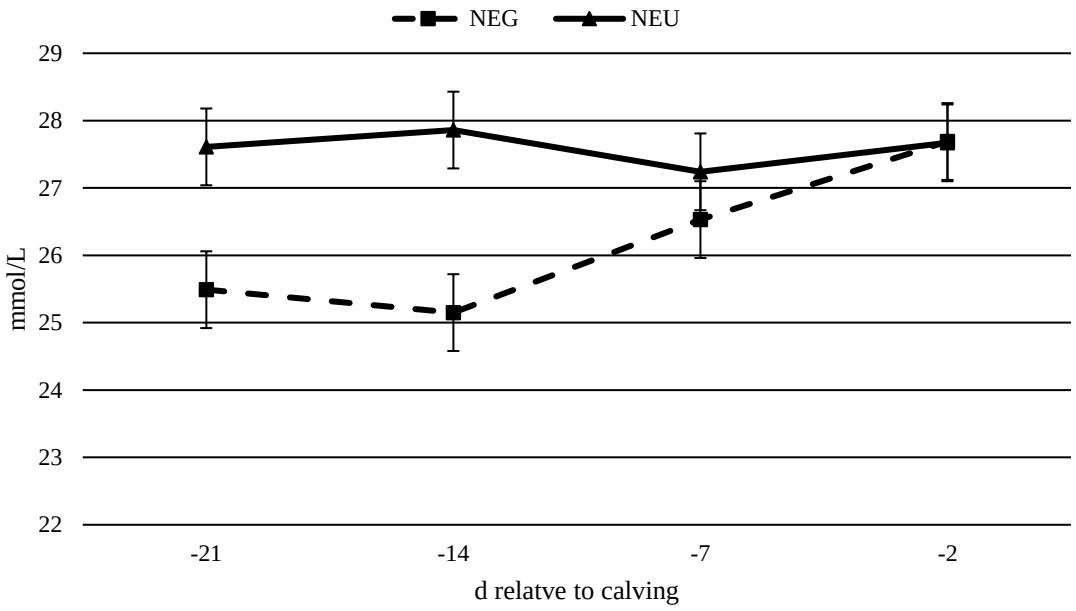

Figure 3.6. Prepartum plasma bicarbonate of cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD x d, $P = 0.0599$)

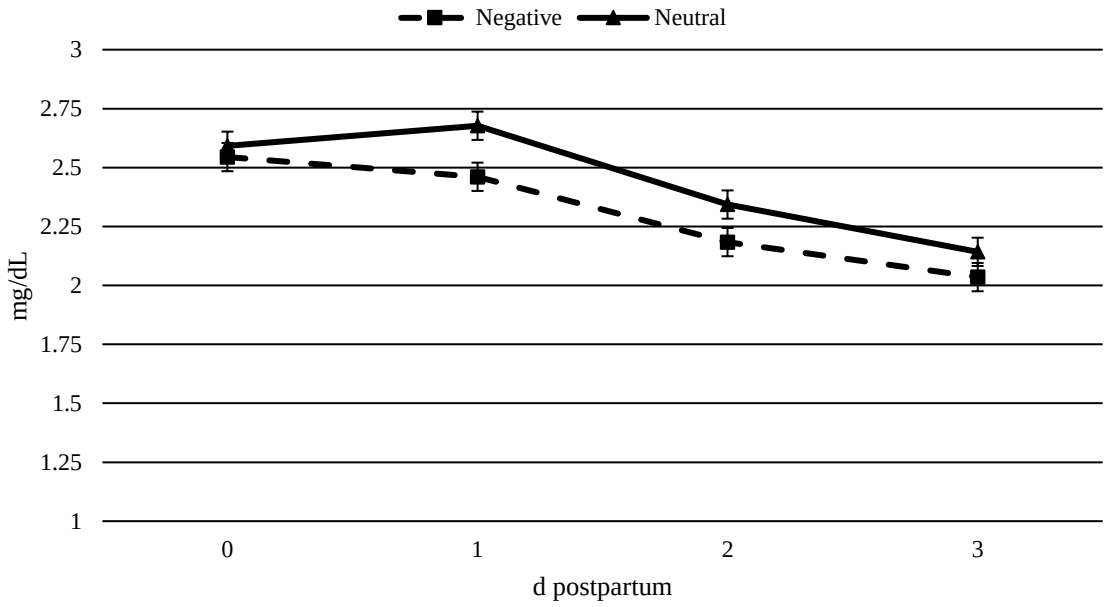

Figure 3.7. Plasma total Mg from 0 to 3 d postpartum of cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD x d, $P = 0.0355$)

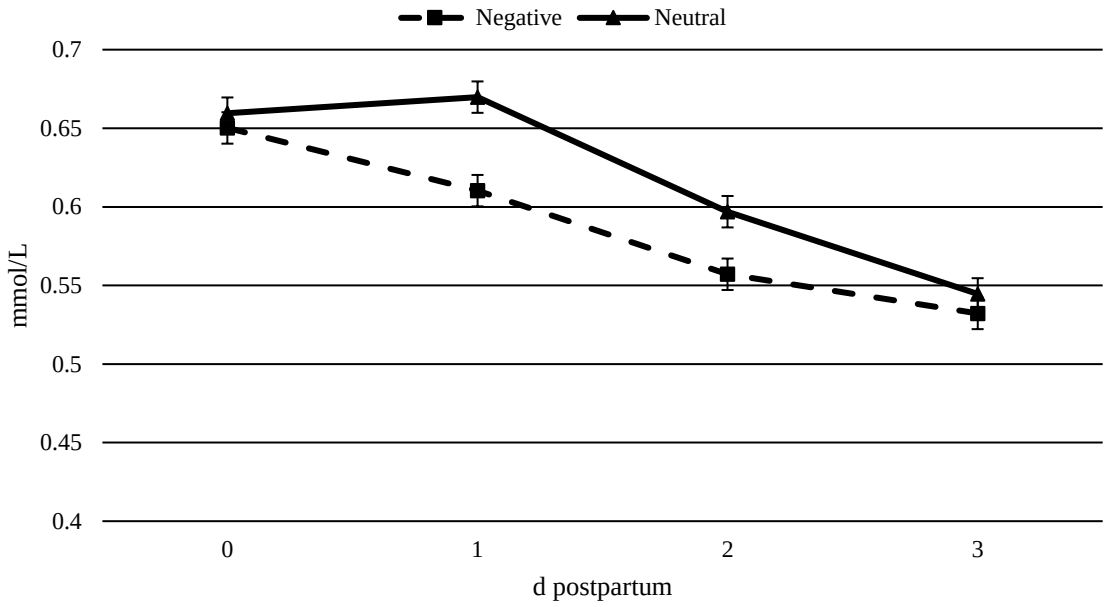

Figure 3.8. Plasma ionized Mg from 0 to 3 d postpartum of cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD x d, $P = 0.0020$)

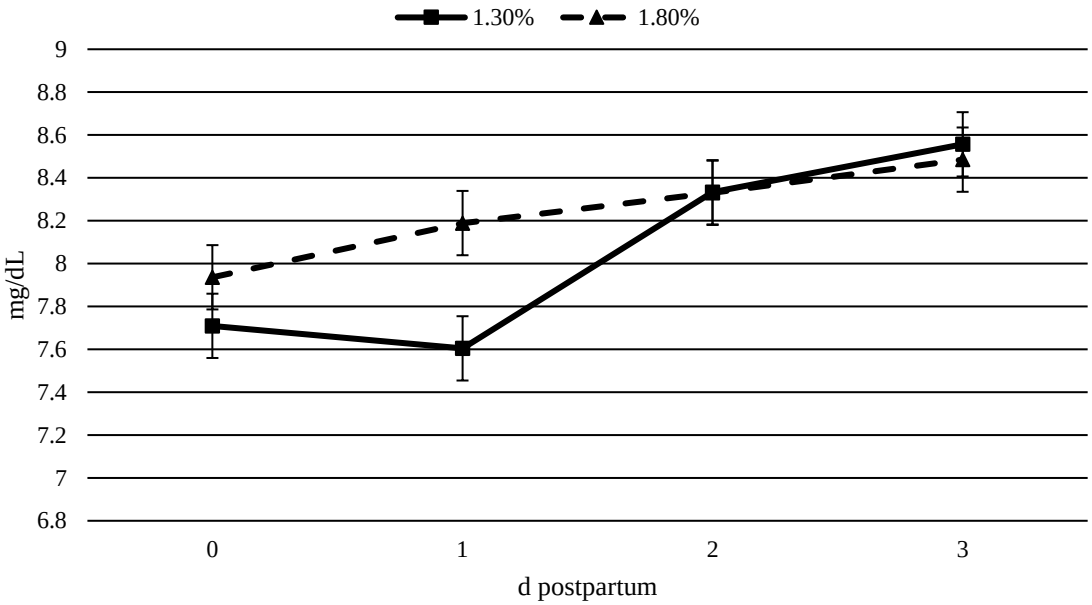

Figure 3.9. Total plasma Ca from 0 to 3 d postpartum for cows fed diets differing in DCAD and Ca concentrations (Ca x d, $P = 0.0030$)

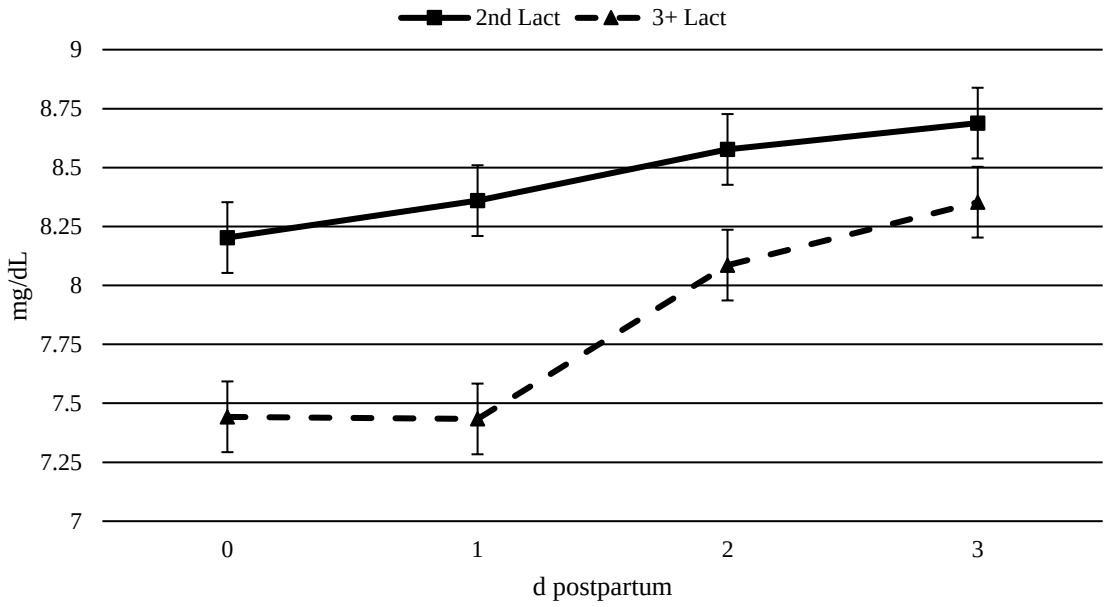

Figure 3.10. Plasma total Ca from 0 to 3 d postpartum by lactation of cows fed diets differing in prepartum DCAD and Ca concentrations (Lact x d, $P = 0.0403$)

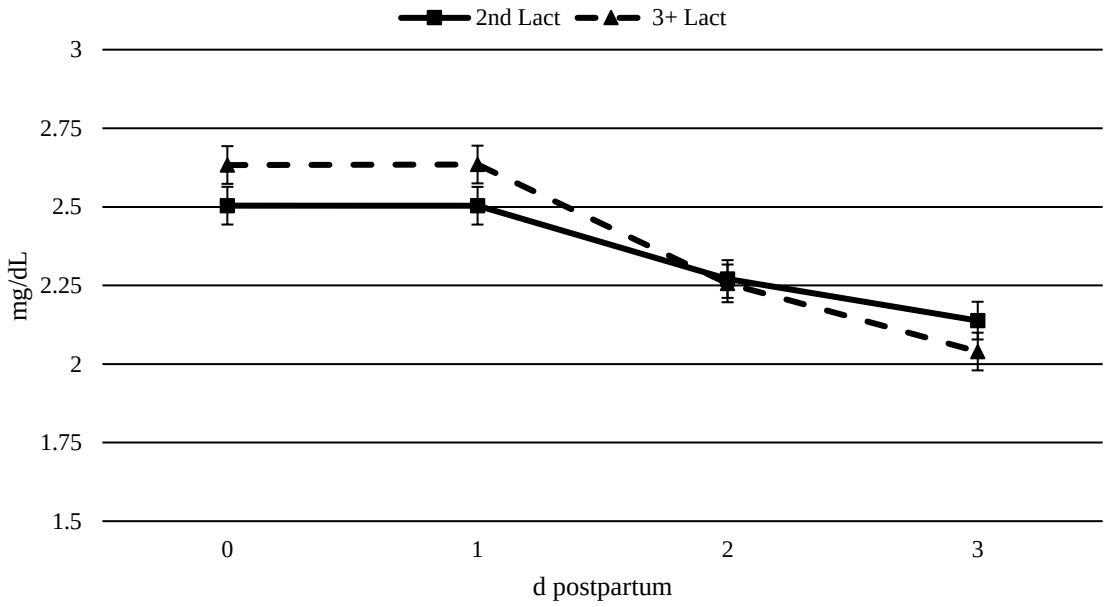

Figure 3.11. Total plasma Mg from 0 to 3 d postpartum by lactation of cows fed diets differing in prepartum DCAD and Ca concentrations (Lact x d, $P = 0.0205$)

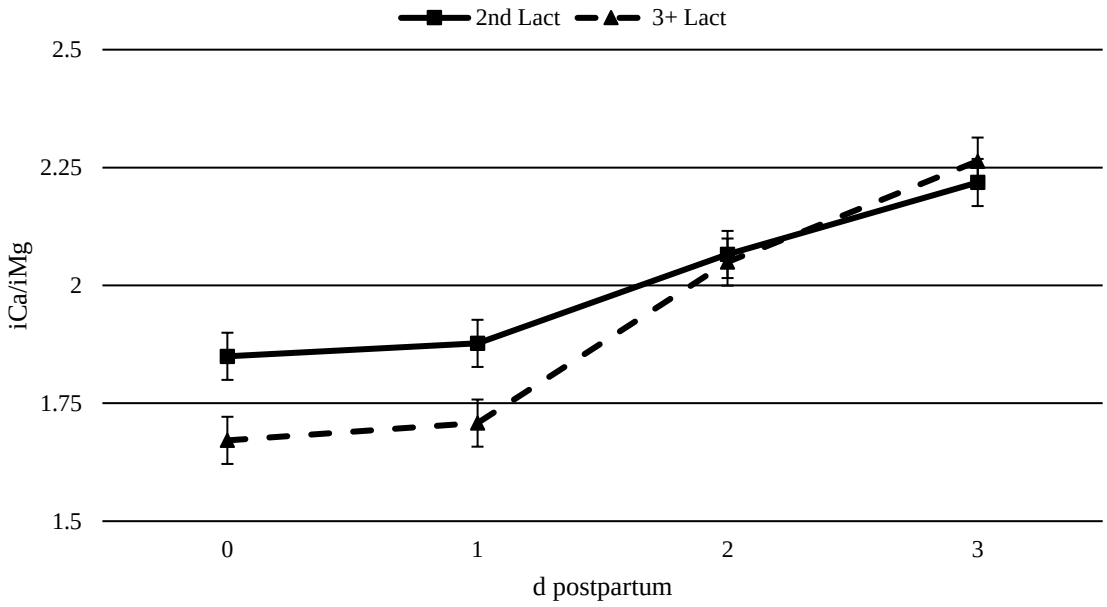

Figure 3.12. Ratio of iCa/iMg from 0 to 3 d postpartum by lactation of cows fed diets differing in prepartum DCAD and Ca concentrations (Lact x d, $P = 0.0068$)

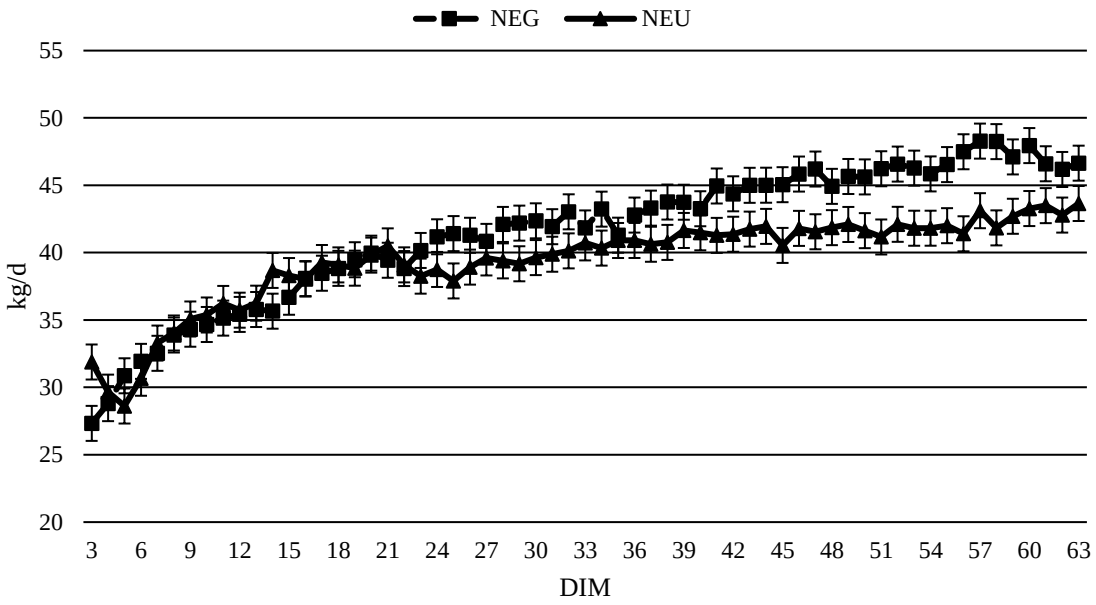

Figure 3.13. Daily milk yield for cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD x DIM, $P = 0.0125$)

CHAPTER 4

EFFECT OF VARYING DCAD AND CALCIUM CONCENTRATION ON COLOSTRUM QUALITY AND NEWBORN CALF BLOOD MINERAL AND GAS CONCENTRATION[2]

[2] Diehl, A. L. and J. K. Bernard. To be submitted to *J. Dairy Sci.*

ABSTRACT

Eighty-two multiparous Holstein cows were fed diets differing in dietary cation-anion difference (DCAD) and Ca concentrations in a randomized block design experiment beginning 4 wk prior to anticipated calving to determine the effects on colostrum yield and quality and acid-base balance of calves. Treatments were arranged as a 2 X 2 factorial to provide two DCAD (-22 mEq/100 g DM [NEG] or -3 mEq/100 g DM [NEU]) and two Ca concentrations (1.3 or 1.8% of DM). Calves were fed a commercial colostrum replacer to provide 200 g IgG within 4 h of birth. Blood samples were collected before and 24 to 36 h after the initial colostrum replacer feeding for analysis of IgG, plasma minerals and blood gases. Cows were milked within 2 to 8 h after calving and colostrum yield recorded. Colostrum samples were collected for immediate Brix analysis and for IgG concentrations by radial immunodiffusion. Birth weight and dystocia score were not different among treatments and averaged 42.7 kg and 1.12, respectively. No differences were observed in colostrum yield among treatments, which averaged 8.75 kg. Colostrum quality, as measured using a Brix refractometer, was not affected by prepartum DCAD but was higher (P = 0.0442) for 1.3 compared with 1.8% Ca: 21.58% and 19.87%, respectively. Concentrations of IgG were higher (P = 0.0034) for cows fed NEG compared with NEU and for 1.3 compared with 1.8% Ca. No differences were observed in plasma concentrations of Ca, P, K, Cl, anion gap, or whole blood pH, pO2, pCO2, or SO2 of calves due to treatment. Plasma Mg (P = 0.0391) and lactate (P = 0.0591) were higher for calves born to cows fed 1.3% compared with 1.8% Ca. Interactions of DCAD and Ca were observed for plasma Na (P = 0.0232), plasma Cl (P = 0.0619) and whole blood HCO3 (P = 0.0515) due to higher concentrations observed with

NEG and 1.3% Ca compared with NEG and 1.8% Ca. Feeding prepartum diets with 1.8% compared with 1.3% Ca concentrations reduced plasma Mg and lactate concentrations in calves immediately after birth and reduced Brix value of colostrum. Results of this trial indicate that feeding a fully acidified diet prepartum may support higher IgG concentrations and does not alter blood mineral or gas concentrations of calves compared with feeding a neutral DCAD diet. Feeding 1.8% decreased colostrum quality as measured by Brix and IgG concentrations.

KEY WORDS: DCAD, Ca, colostrum, blood minerals

INTRODUCTION

Feeding a negative DCAD prepartum diet has become prevalent within the dairy industry as a means of reducing the incidence of milk fever. This dietary manipulation creates a mild metabolic acidosis in order to alter calcium homeostasis mechanisms. With increased prevalence for feeding acidified diets prepartum, there is also an increased curiosity of possible effects on calves from the industry. There has been limited research regarding the response of calves born to dams fed negative DCAD diets prepartum. Metabolic and respiratory acidosis are key parameters in measuring the metabolic state of claves. Both measures are strongly correlated with increased incidence of calf mortality (Szenci, 1985). Past observations show no effect on plasma mineral concentrations of calves from dams fed negative versus positive DCAD diets (Tucker, 1992). Others have reported negative effects on IgG absorption in calves whose dam were fed negative DCAD diets (Joyce and Sanchez, 1994; Guy et. al., 1996; Quigley and Drewry, 1998). The objectives of this trial were to determine the effects of prepartum DCAD and dietary calcium concentration on colostrum quality, newborn calf blood mineral and gas concentration, and calf health.

MATERIALS AND METHODS

Animals and Calving Management

All methods were reviewed and approved by the University of Georgia Animal Care and Use Committee prior to conducting the trial. Eighty-two multiparous Holstein calves born to cows fed diets differing in DCAD (-3 [NEU] or -22 [NEG] mEq/100 g DM) and Ca (1.3 or 1.8% of DM) were used in the trial. Cows calved in a dry lot or were

brought into a dry calving pen when parturition was near. Time of birth, sex, and dystocia score were recorded at calving. Calves were separated within 4-6 h after birth and fed a colostrum replacer to provide 200 g IgG (Bovine IgG, Land O Lakes® Animal Milk Products Co., Shoreview, MN) via bottle or tube if necessary. Calves were individually housed in a calf barn or in individual calf hutches on sand. Calves received vaccinations and an ear tag and were fed a commercial milk replacer (Nuture Processional 26-17, Vigortone Ag Products, Brooksville, OH) at the rate of 0.68 kg solids per day twice daily through 6 wk of age. During wk 7, the amount of milk replacer fed was reduced by 50% and calves were weaned at the beginning of wk 8. Water and calf starter were offered for free choice daily.

Sampling

Colostrum was harvested and a sample collected within 2-8 h of calving. Colostrum analysis via Brix refractomer was conducted immediately after colostrum harvest (MISCO DD-1 Refractometer, MISCO, Solon, OH) to indicate quality and a aliquot frozen for IgG analysis using radial immunodiffusion (Bovine IgG Test Kit, Triple J Farms, Bellingham, WA). Blood samples were collected immediately after birth and prior to colostrum feeding and again at 24 h after birth for analysis of lactate, pH, and blood gases to include pO2, pCO2, pSO2, Ca, Mg, Na, K, and Cl (Siemens Medical Solutions USA, Inc., Malvern, PA). Body weights were recorded prior to the calf entering individual calf pens on the day of birth. Dystocia scores for the cow-calf pair were also recorded after calving. Calf mobility, mortality, and health events were recorded and monitored for the following 8 wk.

Statistical Analysis

PROC MIXED procedures of SAS were used to analyze the data. The model included block, DCAD treatment, calcium treatment, interaction of DCAD and calcium, week, interactions of week and treatments, and sex of calf. Significance was declared when $P < 0.05$ and trends were declared when $P > 0.05$ and ≤ 0.10. When an interaction was detected, the PDIFF option was used for mean separation.

RESULTS

Birth weight and dystocia score were not different among treatments ($P > 0.10$) and averaged 42.7 kg and 1.12, respectively. Colostrum yield and selected characteristics are shown in Table 4.1. Yield was not different among treatments ($P > 0.10$) and averaged 8.75 kg. However, colostrum quality measured using Brix refractometer was not different among DCAD treatment but was higher (P=0.0442) for 1.3% Ca compared to 1.8% Ca: 21.58% and 19.87% respectively. Concentrations of IgG via radial immunodiffusion (Figure 4.1) were different for prepartum DCAD (P = 0.0034) and Ca (P = 0.0120). The prepartum NEG diet had higher IgG concentration compared to NEU which averaged 9,982.1 mg/dl and 5,395.0 mg/dl, respectively. Inclusion of 1.3% Ca also supported higher IgG concentrations compared to 1.8% Ca: 9,558.5 mg/dl and 5,818.4 mg/dl, respectively.

Blood metabolites of calves are shown in Table 4.2. No differences ($P > 0.10$) were observed among treatments in plasma concentrations of calcium, phosphorus, potassium, anion gap, or whole blood pH, pO2, pCO2, or pSO_2 of calves. Calves born to cows fed 1.3% calcium had higher concentrations of plasma Mg (P=0.0391) and plasma

lactate (*P*=0.0591) compared with 1.8% calcium. An interaction was observed between DCAD and calcium for plasma Na (*P*= 0.0232), plasma Cl (*P*= 0.0619) and whole blood HCO_3 (*P*= 0.0515) which were higher for NEG and 1.3% calcium compared with NEG and 1.8% calcium. An interaction of DCAD and day was observed for serum CO_2 (*P* = 0.0062) and whole blood HCO_3 (0.0237). Serum CO_2 increased greater from d 0 to d 1 in calves from cows fed the NEU compared with NEG (Figure 4.2). Whole blood HCO_3 decreased from d 0 to d 1 (27.42 to 26.88 mmol/L) in calves born to cows fed NEG while it increased (26.60 to 28.29 mmol/L) in calves from cows fed NEU (Figure 4.3).

Morbidity measures were recorded for the incidence of scours and respiratory illness. There were 7, 5, 4, and 10 cases of scours and 0, 1, 4, and 4 cases of respiratory illness was observed for NEG-1.3% (n=21), NEG-1.8% (n=19), NEU-1.3% (n=20) and NEG-1.8% (n=22) respectively. Mortality rates by treatment were 14.3%, 5.3%, 10% and 22.7% respectively.

DISCUSSION

Research on the effects of prepartum DCAD and calcium on calves and colostrum has been limited. No differences were observed in colostrum quantity from cows fed either a NEU or NEG diet prepartum. However, colostrum quality was effected by both acidification level and calcium concentration of prepartum diets. Colostrum quality was initially measured by Brix refractometer immediately after harvesting colostrum. The Brix refractometer has been proven to be a valid on-farm tool to detect colostrum quality (Bartier et. al., 2015). Research using the Brix refractometer have correlated a Brix score between 18% and 23% to be equivalent to the concentration of 5,500 mg/dL IgG,

indicating high quality colostrum (Jones and Heinrichs, 2011). Our Brix measures indicated quality was reduced for cows fed 1.8% Ca versus 1.3% Ca prepartum. Brix scores averaged 19.87 and 21.58%, respectively. A subsample was frozen and further used to measure IgG content using radial immunodiffusion. These results indicated quality was negatively affected by feeding either a neutral DCAD (NEU) and or increased Ca (1.8% Ca). Colostrum quality is most often effected by dry period length, dry period, age of cow, breed and volume of first milk (dilution effects). However, in this trial these factors remained constant among the treatment groups. Our findings need to be repeated in order to determine if these effects are consistent due to treatment and if so, may have a large impact on future calf feeding management and strategies. No differences were observed in IgG concentration in the serum of calves from cows fed NEU or NEG diets prepartum. Joyce and Sanchez, 1994 and Guy et. al. (1996) reported that calves from cows fed anionic diets had lower serum IgG concentration. Results from our current trial indicate there was no effect on the apparent efficiency of IgG absorption in calves based on the acidification level of prepartum diets fed to the cow.

Calf metabolic status is measured by the incidence of respiratory and metabolic acidosis. Normal calves with little to no respiratory distress will have a serum pCO_2 of 55 ± 3 mmHg (Szenci, 1985). Our results indicate the average pCO_2 concentrations of calves for treatments were greater than 55 ± 2 mmHg, meaning little to no respiratory distress was observed. Serum pH is used in combination with pCO_2 as an indicator for metabolic acidosis. Serum pH was not effected by prepartum DCAD which is consistent with Tucker (1992) who concluded that feeding negative DCAD diets prepartum has no effect on acid-base status or blood minerals of calves.

CONCLUSION

Results of this trial suggests that feeding a fully acidified diet to cows prepartum does not alter blood mineral or gas concentrations in calves compared with calves born to dams fed a non-acidified diet. Feeding a fully acidified diet did result in higher quality colostrum as indicated by IgG concentrations. However, increasing the calcium concentration of the diet to 1.8 compared with 1.3% may negatively affect colostrum quality and result in lower Brix and IgG concentrations which could affect calf health when fed in the production setting.

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Table 4.1. Colostrum yield and characteristics of cows fed diets differing in prepartum DCAD and Ca concentrations

DCAD	NEU		NEG			P		
Ca, %	1.3%	1.8%	1.3%	1.8%	SE	DCAD	Ca	DCAD x Ca
Yield, kg/d	10.1	7.9	8.4	8.6	1.2	0.6092	0.4567	0.3085
BRIX[1], %	21.42	19.16	21.74	20.58	0.81	0.2792	0.0442	0.4914
IgG[2], mg/dL	6135.9	4654.0	12981.0	6982.8	1171.0	0.0034	0.0120	0.1153

[1] Measured by Brix Refractometer (MISCO DD-1 Refractometer)

[2] Measured by radial immunodiffusion (Bovine IgG Test Kit)

Table 4.2. Serum metabolites of calves born to cows fed diets differing in prepartum DCAD and Ca concentrations

DCAD	NEU		NEG			P		
Ca, %	1.3%	1.8%	1.3%	1.8%	SE	DCAD	Ca	DCAD x Ca
Serum								
IgG, mg/dL	1151.9	1018.6	1036.1	1103.7	267.13	0.9547	0.9006	0.7063
Ca, mg/dL	11.05	11.28	11.05	11.23	0.24	0.9095	0.3447	0.9045
P, mg/dL	6.98	6.98	6.89	6.85	0.14	0.4361	0.8940	0.8982
Mg, mg/dL	2.40	2.28	2.41	2.33	0.05	0.5843	0.0391	0.6907
K, mEq/L	6.92	7.14	7.77	6.54	0.49	0.8029	0.3047	0.1517
Na, mEq/L	142.48	139.25	140.60	143.47	1.29	0.3668	0.8845	0.0232
Cl, mEq/L	100.43	98.48	100.04	102.01	1.02	0.1259	0.9948	0.0619
CO_2, mmol/L	27.47	29.20	28.03	28.41	0.61	0.8515	0.0816	0.2773
Anion gap, mmol/L	20.71	18.62	19.45	19.32	0.71	0.6911	0.1131	0.1825
Whole blood								
pH	7.26	7.26	7.24	7.27	0.02	0.7214	0.2102	0.2751
pO_2, mmHg	45.54	39.25	43.21	42.84	4.19	0.8799	0.4119	0.4806
pCO_2, mmHg	60.60	64.03	66.54	58.18	3.24	0.9887	0.4415	0.0770
HCO_3, mmol/L	26.89	28.00	28.01	26.30	0.70	0.6726	0.6613	0.0515
SO_2, mmol/L	62.11	58.17	59.68	61.58	2.93	0.8659	0.7214	0.3311
Lactate, mg/dL	6.74	5.78	6.93	6.13	0.47	0.5698	0.0591	0.8670

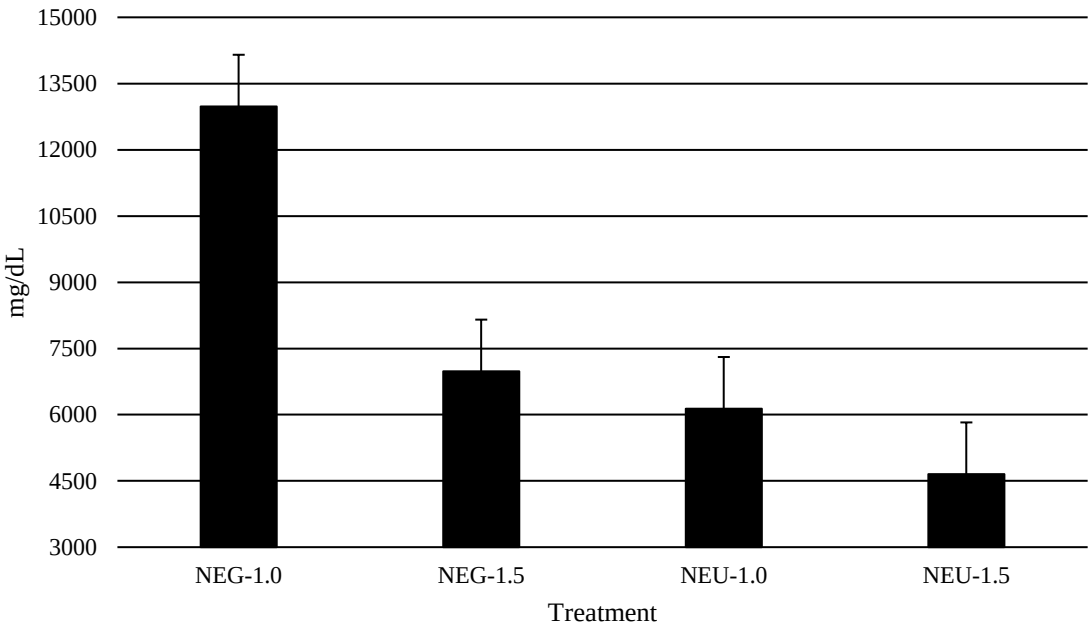

Figure 4.1. Colostrum IgG concentration of cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD, $P = 0.0034$; Ca, $P = 0.0120$)

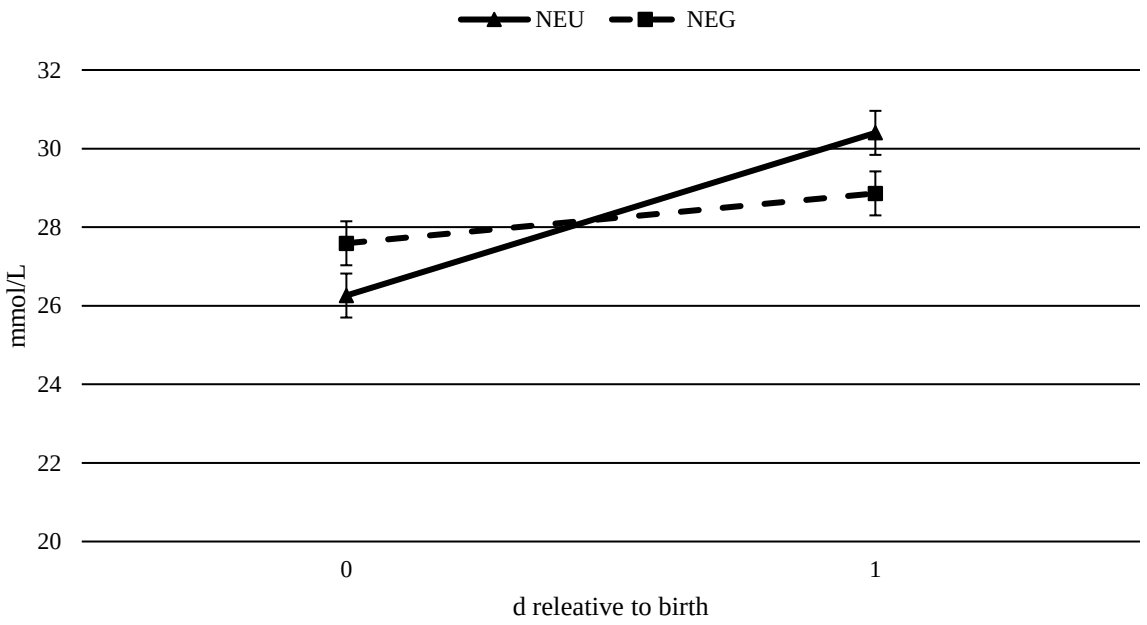

Figure 4.2. Serum CO_2 0 and 1 d after birth of calves from cows fed diets differing in prepartum DCAD and Ca concentrations (DCAD x d, $P = 0.0062$)

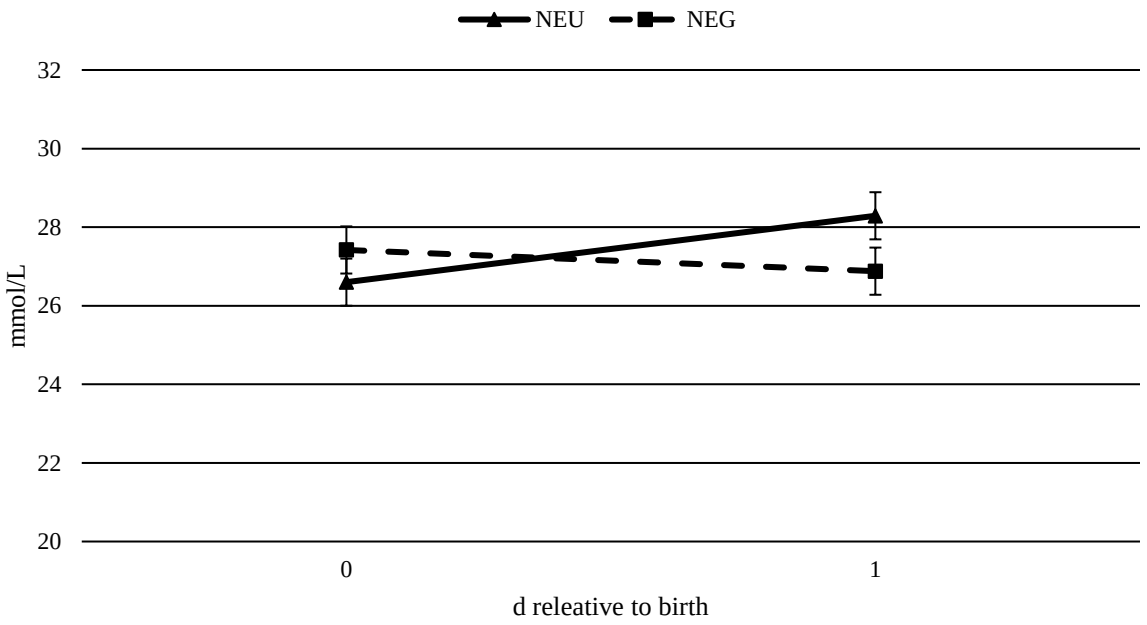

Figure 4.3. Whole blood HCO_3 0 and 1 d after birth of calves from cows fed diets differing in prepartum DCAD and Ca concentrations. (DCAD x d, $P = 0.0237$)

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CONCLUSIONS

Results of this trial indicate that feeding fully acidified diet (-22 mEq/100 g DM DCAD) altered plasma and urine mineral concentrations to successfully improve Ca status by 3 DIM and increased milk yield from 45 through 63 DIM compared with a neutral diet (-3 mEq/100 g DM DCAD). Feeding 1.8% Ca prepartum improved total plasma Ca at 1 DIM compared to 1.3% Ca, but also decreased prepartum DMI. No differences were observed for postpartum DMI or milk composition among treatments. Colostrum quality as measured by Brix and IgG was greater for cows fed diets supplemented with 1.3% Ca compared with that of cows fed 1.8% Ca and for NEG compared to NEU. No differences were observed in concentrations of IgG or acid base balance of calves born to cows fed fully acidified or neutral DCAD diets supplemented with 1.3 or 1.8% Ca. These results suggest feeding a fully acidified diet (-22 mEq/100 g DM DCAD) and 1.3% Ca prepartum is sufficient for preventing hypocalcemia immediately postpartum and does not alter blood mineral or gas concentrations, or IgG absorption efficiency in calves.