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Effect of continued metabolic acidification into the first three days of lactation on blood calcium status in postpartum dairy cattle: a randomized controlled trial

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Abstract

Although the incidence of clinical hypocalcemia in postpartum dairy cows is low on US dairies, subclinical hypocalcemia post calving is common and has been associated with metabolic and infectious disease. It is widespread farm practice to feed a diet rich in anions to prepartum dairy cattle to support calcium homeostasis. However, this diet is typically discontinued at parturition when calcium needs are still high. The objective of this trial was to determine the effects of extending metabolic acidification into the first three days of lactation in multiparous Holstein cows with the use of magnesium chloride ($MgCl_2$) hexahydrate drenches on blood ionized calcium concentrations.

Adult Holstein cows at a commercial dairy in their 2nd or higher lactation with a urine pH of 6.8 or less on the day of calving were randomly assigned to either treatment or control groups, resulting in 13 cows in the treatment group and 14 cows in the control group. Treatment cows received 480 g oral $MgCl_2$ hexahydrate once daily for three days for continued acidification starting on the day of calving while cows in the control group received no treatment. Urine pH was measured daily for 5 days starting on the day of calving (0 DIM) to assess acidification status; blood was collected on day of calving, 2 DIM and 4 DIM calving and analyzed for ionized calcium concentrations. Differences in blood ionized calcium and urine pH were compared using longitudinal data analysis.

Urine pH was lower in treatment cows when compared to control cows 1, 2 and 3 DIM. Blood ionized calcium concentrations were different from baseline taken at enrollment at 2 and 4 DIM in both treatment and control cows. However, there was no difference between treatment and control cows at 2 or 4 DIM with respect to blood ionized calcium concentrations.

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Oral supplementation with $MgCl_2$ hexahydrate resulted in the desired acidification of urine pH in the treatment group similar to feeding of an anionic close-up diet. Continued acidification of dairy cows until 2 DIM did not result in clinically meaningful higher blood calcium concentrations compared to controls, and further research to identify physiological reasons for this finding are needed.

INTERPRETIVE SUMMARY

Effect of continued metabolic acidification after calving on blood calcium concentration in postpartum dairy cattle: *By Maier et al.* Milk fever is a disease of dairy cattle caused by low blood calcium before or after calving. Cows were fed a low DCAD diet prior to calving to increase calcium release from bone and absorption in the intestine to reduce hypocalcemia. We drenched cows with anions the first 3 days of lactation to determine if extending acidification into early lactation improved blood calcium status. Despite lower urine pH there was no improvement in blood calcium concentration.

Keywords

DCAD; hypocalcemia; postpartum; dairy cows

INTRODUCTION

Milk fever has been recognized as a disease of dairy cattle for over 200 years (Murray et al. 2008). The disease is caused by inadequate blood calcium concentrations necessary for proper nerve and muscle function and is of particular concern in the periparturient cow in which the onset of lactation often overwhelms her homeostatic capabilities (Goff 2008). The reported incidence of clinical milk fever in US dairy herds is low with an estimated 2.8% based on the 2014 survey by the National Animal Health Monitoring System (USDA 2018). However, the prevalence of subclinical hypocalcemia is 25% and 47% in primiparous and multiparous cows respectively (Reinhardt et al. 2011). Subclinical hypocalcemia has been defined in the literature as a serum total calcium concentration of < 2.15 mmol/L (8.60 mg/dL) (Martinez et al. 2012) or blood ionized calcium < 1.0 mmol/L (Martinez et al. 2014). Hypocalcemia in dairy cattle is an area of ongoing research and cut points have evolved based on new knowledge, including considering factors such as days in milk and parity (Neves et al. 2018). Subclinical hypocalcemia has been associated with decreased feed intake and less time spent chewing and ruminating, reduced concentration of neutrophils in the blood, impaired neutrophil function and an increased risk of a number of postpartum diseases (Hansen et al. 2003; Martinez et al. 2012). There is a higher risk of developing metritis or displaced abomasum in cows with subclinical hypocalcemia in the first three to four days in milk following calving compared to normocalcemic cows (Martinez et al. 2012; Neves et al. 2018). Cows with blood calcium concentrations < 2.2 mmol/L in the first week postpartum had a higher incidence of displaced abomasum than cows with higher blood calcium concentrations (Chapinal et al. 2011). Cows with retained fetal membranes had significantly lower plasma calcium concentrations (1.79 mmol/L± 0.09) soon after calving compared to cows without retained fetal membranes (2.19 mmol/L +/− 0.07) (Melendez et al. 2004).

Inducing a mild metabolic acidosis in dairy cows in the close-up period before parturition by increasing the dietary anions $SO4^{2-}$ and Cl^{-}· relative to the dietary cations Na^{+} and K^{+} (DCAD; dietary cation anion difference) remains the most accepted preventive measure for milk fever (Murray et al. 2008). Urine pH is correlated with blood pH and blood HCO_3 and decreases with lower DCAD values (Luebbe et al. 2011). Urine pH is often used as a surrogate for metabolic acidification and as a monitoring tool for dairy cows in the close-up pen due to its relative ease of sample collection and low cost. Metabolic acidosis has several effects on the function of parathyroid hormone (PTH) released during a state of hypocalcemia and its receptor. The conformation of the PTH receptor is more sensitive to PTH at a lower pH and the kidneys have higher reabsorption of calcium from the glomerular filtrate and function better at converting 25-hydroxyvitamin D into 1,25-dihydroxyvitamin D, the active form of vitamin D, which is required for intestinal uptake of calcium (Goff 2008). A negative DCAD is typically fed during the last two to three weeks of pregnancy (Goff 2014). Once a cow has calved, she is usually moved to a different pen where the addition of acidifying salts to the diet is discontinued and DCAD becomes positive.

The objective of our study was to evaluate the effect of an extension of negative DCAD into the first three days of lactation on the blood calcium concentration of postpartum multiparous dairy cows to evaluate this technique as an approach to reduce postpartum subclinical hypocalcemia. Our hypothesis was that cows receiving continued metabolic acidification into the first three days of lactation would have higher blood calcium concentrations than those that did not. The primary outcome was mean blood ionized calcium concentrations 2 DIM and 4 DIM and the secondary outcome was mean urine pH measured once daily between 1 DIM and 4 DIM for cows in the treatment and control groups.

MATERIALS AND METHODS

Our study was approved by the UC Davis Institutional Animal Care and Use Committee (protocol #18395). A sample size calculation resulted in 14 cows per group to detect a difference of 0.1 mmol/L with a standard deviation of 0.09 mmol/L of ionized calcium between groups with a power of 80% and alpha at 0.05. We enrolled 27 multiparous Holstein dairy cows on a commercial dairy on the day of calving (0 DIM) after they had calved and after measuring their urine pH with a digital urine pH-meter (Horiba Scientific, Piscataway, NJ). Cows were randomly assigned to either control or treatment groups. Randomization was conducted using a list of random numbers generated in a spreadsheet program (Excel, Microsoft, Redmond, WA). Cows in the study were not administered any routine postpartum supplementation.

Cows with a urine pH above 6.8 were considered not acidified and were not eligible to be enrolled in the study. Likewise, we excluded cows from enrolment that appeared visually depressed, based on demeanor, ear carriage, and appetite or had retained fetal membranes, as these cows were more likely to have decreased feed intake or to suffer from low blood calcium concentrations during the study period, which would have confounded results. After determining eligibility of a cow to be enrolled in the study, the investigator was told by the assistant whether the cow was in the treatment or control group based on the random

numbers list, but no special concealment measure for allocation was put in place. At enrollment, the investigator determined the body condition score of the enrolled study cows using the 1 to 5 body condition score system with 1 being emaciated and 5 being obese (Edmonson et al. 1989) as well as measured their rectal temperature using a digital thermometer on the day of calving. For practical reasons, study cows were moved to the early lactation cow pen and received the same diet as other postpartum cows in the herd. Composition of pre- and postpartum diets are described in Table 1.

Cows were enrolled twice daily either in the morning between 6 and 7 am or in the afternoon between 3 and 4 pm so that the first sample (0 DIM) was collected in a window between 0 – 14 h post calving. Subsequent samples were collected only in the mornings and correspond to the following postcalving time windows: between 14 – 38 h at 1 DIM, between 38 – 62 hours at 2 DIM, between 62 – 86 hours at 3 DIM and between 86 and 110 hours at 4 DIM. Cows in the treatment group were administered 480 g of $MgCl_2$ hexahydrate (Sierra Chemical, West Sacramento, CA) dissolved in approximately 3 L of water and delivered via Magrath cattle pump (Springer Magrath Co., Glencoe, MN) with a 152 cm orogastric tube according to manufacturer's instructions once at enrollment (morning or afternoon) and two more times in the morning starting on the day of calving. Cows in the control group did not receive acidification interventions.

DCAD was calculated for treatment and control groups with the formula DCAD = (% sodium × 43.48) + (% potassium × 25.64) – (% Chlorine × 28.17) – (% Sulfur × 62.5) (Lean et al. 2006). The target DCAD for the treatment group was < 0 meq / 100 g DM. In both treatment and control groups, we collected urine samples via perineal stimulation on the day of calving (morning or afternoon, 0 DIM) and on the following 4 d (1 – 4 DIM) in the morning and measured urine pH via a handheld pH meter (Horiba Scientific, Piscataway, NJ). Prior to each use we calibrated the pH meter at pH 4, pH 7 and pH 10. On the day of calving (0 DIM), and at 2 and 4 DIM, we collected 10 mL of blood from a coccygeal vessel into an evacuated collection tube containing heparin and analyzed via a point of care handheld blood analyzer (iStat, Abaxis, Union City, CA) for ionized calcium within 2 hours of collection.

Cows that required calcium supplementation (IV calcium gluconate) during the study period (0 – 4 DIM) because of clinical signs of hypocalcemia were censored, i.e. only data collected up to calcium supplementation were analyzed. A total of 17 cows were enrolled during December 2014, while 10 cows were enrolled in March 2015.

We performed the statistical analysis with SAS software (SAS version 9.4, SAS Institute Inc., Cary, NC). Linear mixed models with urine pH or blood ionized calcium concentrations as the response variable included sampling timepoints (0, 1, 2, 3 and 4 DIM for urine pH and 0,2 and 4 DIM for blood ionized calcium concentration), treatment group and the interaction of sampling timepoints and treatment group with repeated measures by cow. Baseline values for urine pH and ionized calcium concentration as well as parity were offered to models as possible confounders and remained in the models if $P < 0.05$. Residuals for the models with outcome variables urine pH and ionized calcium concentration were evaluated for normality. Results including confidence intervals were unadjusted for

multiplicity. Model fit was assessed with the Akaike Information Criterion (AIC) for the covariance structures compound symmetry, first order autoregressive, unstructured and Toeplitz. The Kenward-Roger method was used to estimate degrees of freedom. Contrasts were used to compare least square means between different sampling timepoints using the diff function in SAS. The number of subclinically hypocalcemic cows between groups was compared with a Chi square test for a difference in proportions or a Fisher's exact test for small cell counts (50% of cells with expected counts less than 5). We used a cutoff of < 1.0 mmol/L for subclinical hypocalcemia.

RESULTS

The DCAD for the early lactation cow TMR was 22 meq / 100 g DM (0.49% sodium × 43.5) + (1.55% potassium × 25.6) – (0.74% chlorine × 28.2) – (0.29% sulfur × 62.5). Assuming a DMI of 19.3 kg of early lactation cow diet, adding 480 g of $MgCl_2$ hexahydrate ($MgCl_2 \cdot 6H_2O$) with a dry matter content of 225 g, lowered DCAD for the same diet to −2.4 meq / 100 g DM (0.48% sodium × 43.5) + (1.53% potassium × 25.6) – (1.59% chlorine × 28.2) – (0.29% sulfur × 62.5).

Baseline characteristics and urine pH of study cows are listed in table 2. No cows had to be excluded based on high urine pH. Study groups were similar in terms of lactation number, body condition score, rectal temperature, blood ionized calcium, or urine pH. One cow in the treatment group developed mild diarrhea at 2 DIM and did not receive a third $MgCl_2$ hexahydrate treatment. Another cow in the treatment group developed clinical signs of milk fever at 3 DIM and was treated with and responded to IV calcium gluconate after a blood sample was obtained. The data for the two cows that were excluded after study start was used until the day of study exclusion. No blood sample was obtained at 4 DIM for this cow. Furthermore, we were unable to obtain urine from one cow in the treatment group at 4 DIM. Apart from these exceptions, the results from all enrolled cows were used in the analysis.

Change of mean urine pH over the study period is depicted in Figure 1. Mean urine pH at enrollment was 5.84 (± 0.09) in control cows and 6.00 (± 0.1) in treatment cows ($P = 0.52$). On subsequent study days, urine pH in control and treatment cows, respectively, was 7.22 (± 0.2) and 6.38 (± 0.2) at 1 DIM ($P < 0.001$), 7.91 (± 0.1) and 6.77 (± 0.3) at 2 DIM ($P < 0.001$), 7.95 (± 0.07) and 6.78 (± 1.6) at 3 DIM ($P < 0.001$), and 7.97 (± 0.05) and 7.64 (± 0.2) at 4 DIM ($P = 0.24$) where the interaction between study group and study day had a P-value of 0.001. The covariance structure with the lowest AIC was first order autoregressive, which was used for the final model. Baseline urine pH was not a significant covariate and was excluded from the final model.

The change in blood ionized calcium for the two groups is illustrated in Figure 2. Mean blood ionized calcium on day of calving was 1.05 (± 0.02) for controls and 1.09 (± 0.03) for treatment cows ($P = 0.22$). At 2 DIM, ionized calcium concentrations were 1.12 (± 0.02) for controls and 1.17 (± 0.02) for treatment cows ($P = 0.12$). Finally, at 4 DIM, ionized calcium was 1.17 (± 0.02) for controls and 1.18 (± 0.02) for treatment cows ($P = 0.82$). There was an increase in ionized calcium concentrations over time ($P < 0.001$). Blood ionized calcium concentrations were different from baseline (1.07 ± 0.02) in both treatment and control cows

at 2 DIM (1.14 ± 0.02, $P < 0.001$) and 4 DIM (1.17 ± 0.01, $P < 0.001$), but not between treatment groups ($P = 0.16$) and the slopes for the change in ionized calcium concentrations over time between study groups were not different ($P = 0.16$). A model that included baseline calcium concentrations did not converge. Lactation number was significantly associated with the outcome (P=0.02) and was included in the final model to adjust for its effect. The unstructured covariance structure for the repeated measures analysis had the lowest AIC and was used for the final model. At 0 DIM, there were two subclinically hypocalcemic cows (ionized calcium concentration < 1 mmol / L) in the treatment group compared to three in the control group. There was one subclinically hypocalcemic cow in the control group at 2 DIM. All other ionized calcium concentrations measured were ≥ 1 mmol / L and a Fisher's exact test showed no association between group and subclinical hypocalcemia (P=0.52).

DISCUSSION

Cows in the current study responded to treatment with oral $MgCl_2$ hexahydrate with a lowered urine pH during the treatment period (0 – 2 DIM) The urine pH of cows in the treatment group quickly returned to the urine pH of control cows by 4 DIM, after acidification with $MgCl_2$ hexahydrate had been discontinued. There was no difference in blood ionized calcium associated with this difference in urine pH, indicating that acidifying cows in the early postpartum period may not contribute to a reduction in early postpartum subclinical hypocalcemia, although a modified study design where continued acidification is achieved through diet may lead to different results. The blood ionized calcium level tended to be higher in treatment cows at 2 DIM ($P = 0.08$) but the absolute difference of 0.04 mmol/L blood ionized calcium between groups may not be clinically relevant. Clinically relevant differences in blood ionized calcium may be an order of magnitude larger, as demonstrated in a study that found differences between normocalcemic cows and cows with experimentally induced subclinical hypocalcemia with respect to appetite, metabolism, and immune function, where mean blood ionized calcium differed by 0.49 mmol/L (Martinez et al. 2014).

Acidogenic diets have been thought to be less palatable than those without addition of acidogenic salts and lead to depressed DMI (Oetzel et al. 1988). We expected that the delivery of $MgCl_2$ hexahydrate via oral drench once per d could circumvent the problem of lowered palatability and subsequently lowered DMI. However, a recent study showed that the decrease in DMI in diets with negative DCAD is attributable to the metabolic acidosis and not to the addition of acidogenic salts containing chloride (Zimpel et al. 2018). Cows in our study that received the treatment may have had lower DMI than those in the control group because of their acid/base status, which could have negated any advantages in calcium mobilization brought about through lower blood pH. The DCAD achieved in our study was −2.4 meq / 100 g DM compared to the −5.0–15.0 meq / 100 g DM recommended for prepartum cows by the National Research Council (NRC 2001). Calculation of DCAD was based on an assumed feed intake of 19.3 kg for the early lactation cow diet fed in this herd. The actual intake on the day of calving may have been lower than on subsequent days and hence DCAD for the first treatment was likely more negative than what we calculated.

Since magnesium can act as a cathartic in cattle, we wanted to limit the amount of $MgCl_2$ hexahydrate administered to cows to a level that did not cause diarrhea in study cows. Only one of the cows developed mild diarrhea during the trial. Larger doses of $MgCl_2$ hexahydrate may have led to a more negative DCAD, but also could have caused gastrointestinal upset in more study cows, which could have interfered with calcium absorption from the diet. The ideal prepartum DCAD for optimal performance is unknown (Zimpel et al. 2018), but the DCAD achieved in this experiment may have been inadequate in improving calcium mobilization at a clinically relevant level.

We did not observe a drop in blood calcium during the first 2 DIM in the study cows as has been seen elsewhere (Martinez et al. 2012) and few animals had blood ionized calcium concentrations < 1.0 mM during the study period. Since one of the criteria for enrollment was a urine pH of ≤ 6.8, it is conceivable that cows in this study were able to meet their increased postpartum calcium demands because of prepartum acidification. A larger effect may be seen in cows that are less acidified at calving and are less able to mobilize calcium and are therefore more prone to subclinical hypocalcemia. It is also possible that the blood calcium nadir in this herd was at 1 DIM and differences between groups were missed because we did not sample on that day. The cow that showed clinical signs of hypocalcemia and responded to treatment with IV calcium gluconate was in her 5^{th} lactation and more prone to hypocalcemia than lower lactation cows as lactation number is one of the risk factors for clinical postpartum hypocalcemia (Goff, 2008). Her blood ionized calcium level was 1.16 mmol/L, which is within the normal range. The sample was taken the same day but prior to the calcium gluconate infusion. We did not monitor blood magnesium concentrations, but hypermagnesemia can impair nerve conduction, which could have been an unintended side effect of the treatment.

Limitations of this study are the use of an oral drench to achieve negative DCAD versus addition of acidogenic salts to the diet, because for practical reasons dairy farmers would likely prefer adding acidogenic salts to a diet as opposed to the drenching of each cow multiple times. Due to logistical reasons, separating study cows from other animals in the herd during the immediate postpartum period was not feasible and the present solution, while not practical on a large scale, was able to achieve acidification in the study cows. Further limitations are our inability to measure DMI in study animals, limited blood sampling timepoints, and no placebo treatment for control cows, again due to logistical reasons. Measuring DMI could have answered the question whether the lack of differences in blood ionized calcium concentrations were due to differences in DMI. Finally, the researcher conducting the field work was not blinded to treatment allocation in this study, which may have introduced some bias although care was taken to avoid any differential treatments of study groups.

CONCLUSION

To our knowledge, this is the first study evaluating the effect of continued metabolic acidification on ionized blood calcium concentrations in Holstein cows in the first few days of lactation. Although no difference was seen in blood ionized calcium concentrations between treatment and control groups, further research into the possible benefits of this

technique in a more controlled environment, or with additional sampling timepoints may be warranted.

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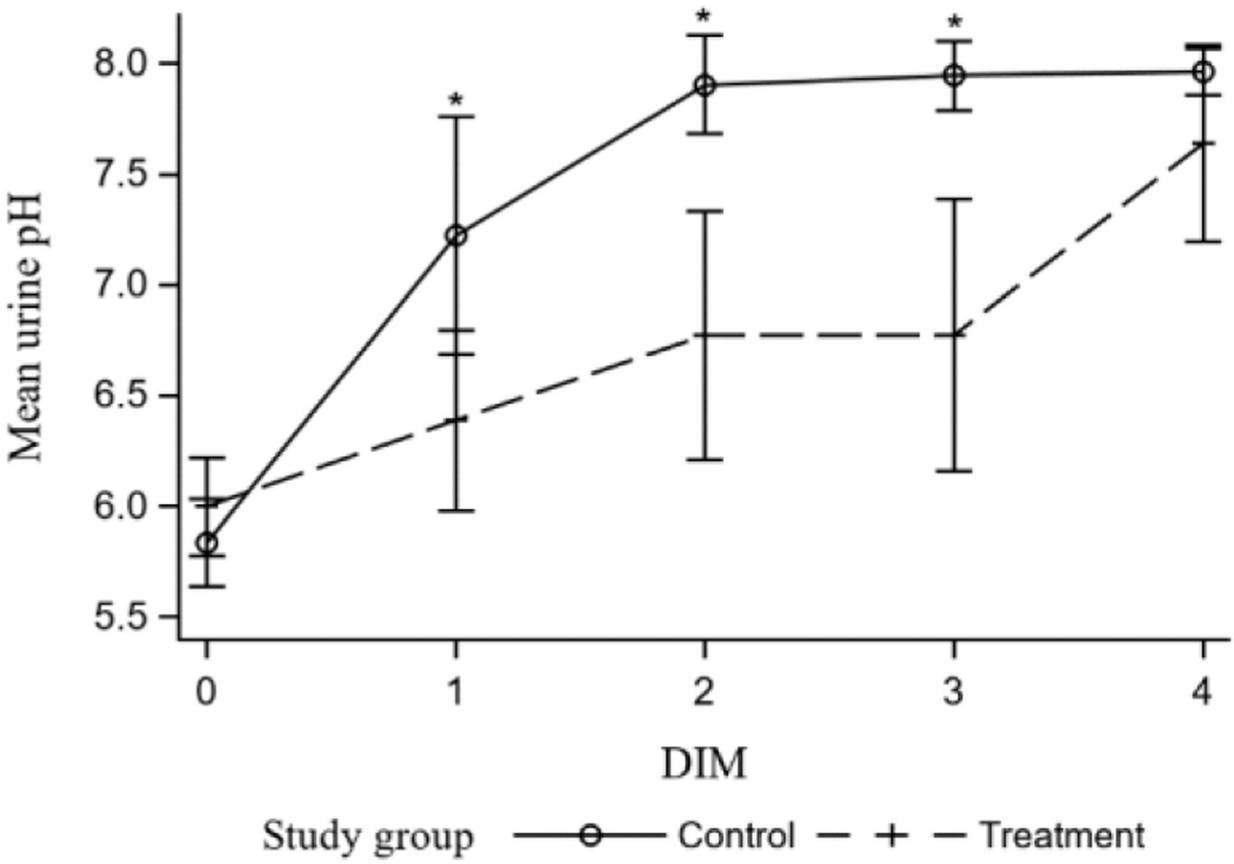

Figure 1.

Mean urine pH (± 95% CI) for 27 multiparous Holstein dairy cows on day of calving and during the first four days postpartum. The treatment group (N = 13) was treated with 480 g $MgCl_2$ hexahydrate as an oral drench for the first three days (d 1, 2, and 3) after calving, while cows in the control group (N = 14) received no treatment. Data points marked with a star differ between groups ($P < 0.05$).

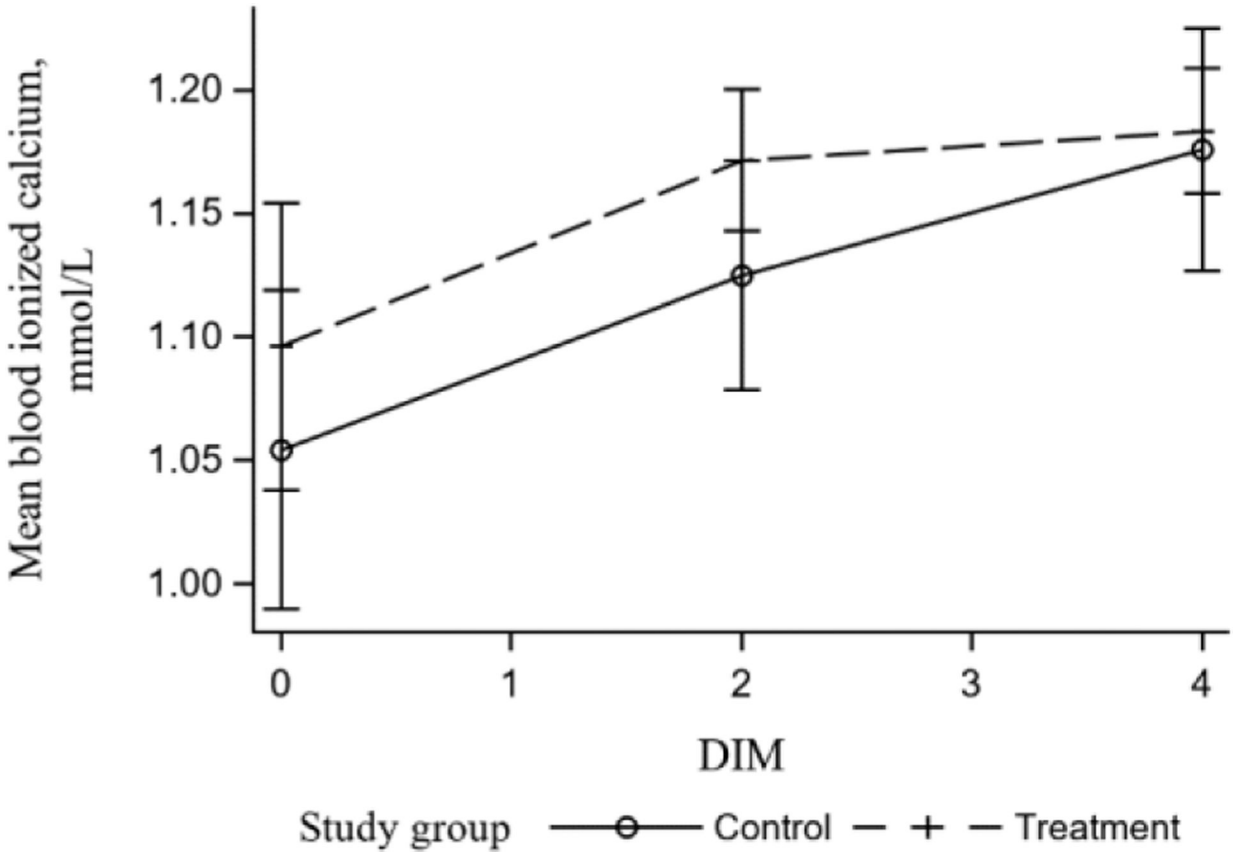

Figure 2.

Mean ionized blood calcium concentration in mmol/L (± 95% CI) in 27 multiparous Holstein dairy cows on day of calving and during the first four days postpartum. The treatment group (N = 13) was treated with 480 g $MgCl_2$ hexahydrate as an oral drench for the first three days (d 1, 2, and 3) after calving, while cows in the control group (N = 14) received no treatment.

Table 1:

Pre- and postpartum diet composition for 27 multiparous Holstein cows that either received oral $MgCl_2$ hexahydrate for 3 d postpartum (treatment) or no treatment (control).

Items, % DM basis	Prepartum[1]	Early lactation[2]
Nutrients		
DM	57.6	53.1
CP	15.4	16.9
ADF	26.6	24.5
NDF	38.4	35.9
Lignin	4.8	4.8
Fat	2.6	3.5
Starch	13.9	17.7
Ash	9.6	9.2
NE[l], MJ/kg	6.6	6.8
NEm, MJ/kg	7.1	7.6
NEg, MJ/kg	4.3	4.6
DCAD[3] meq / 100 g	−12.7	21.8
Sodium	0.18	0.49
Chloride	1.3	0.74
Potassium	1.5	1.55
Sulfur	0.35	0.29
Magnesium	0.38	0.36
Calcium	1.2	1.03
Ingredients		
Corn Silage	38	30
Alfalfa Hay	27	19
Corn	4.6	14
Wheat Straw	4.0	1.6
Beet Pulp Pellets	5.4	5.3
Soy Hulls Pellets	3.8	4.1
Wheat Mill Run	3.4	3.2
Canola Meal	3.2	4.3
Dry Corn Distillers Grains	2.5	1.2
Molasses Cane	0.13	0.51
Whole Cottonseed		7.1
Soybean Meal		5.4
Soy Best		1.6
Blood Meal		0.75
Supplement[1]	8.85	2.65

[1]Prepartum Supplement: Calcium carbonate, calcium chloride, magnesium sulfate 7H20, calcium sulfate dihydronate, magnesium oxide, vitamin E, white salt, Rumensin 90.7 (Elanco® Greenfield, IN), Prequel 21 (Virtus Nutrition LLC Corcoran), Alimet (Novus International, Inc Saint Charles, MO), Celmanax SCP (Arm & Hammer Animal Nutrition Princeton, NJ), Diamond V XPC (Diamond V Cedar Rapids, Iowa), Clarifly

0.67% (Central Garden & Pet Company, Schaumburg, IL), Biochlor (Arm & Hammer Animal Nutrition Princeton, NJ), Animate (Phibro Animal Health Corporation Teaneck, NJ), Reashure Choline (Balchem Corporation New Hampton, NY), PDS L55 Mintrex Premix (Progressive Dairy Solutions, Inc. Oakdale, CA)

[2]Early lactation supplement: Magnesium oxide, Vitamin E, oyster shell ground, sodium bicarbonate, white salt, Zinpro4 Plex C (Diamond V Cedar Rapids, Iowa), Rumensin 90.7 (Elanco® Greenfield, IN), Prequel 21 (Virtus Nutrition LLC Corcoran), Alimet (Novus International, Inc Saint Charles, MO), Celmanax SCP (Arm & Hammer Animal Nutrition Princeton, NJ), Diamond V XPC (Diamond V Cedar Rapids, Iowa), Clarifly 0.67% (Central Garden & Pet Company, Schaumburg, IL), PDS L45 TM (Progressive Dairy Solutions, Inc. Oakdale, CA)

[3]DCAD was calculated as (Na + K) − (S + Cl) using the DCAD calculator, Arm & Hammer Animal and Food Production (https://ahfoodchain.com/calculators/dcad-calculator)

Table 2:

Mean (standard deviation) or median (interquartile range) and range for baseline characteristics on day of calving in 27 multiparous Holstein cows that either received oral $MgCl_2$ hexahydrate for 3 d postpartum (treatment) or no treatment (control).

Baseline characteristic	Treatment (N=13)	Control (N=14)
Lactation number[1]	3.0 (2.0–4.0)	3.5 (2.0–5.0)
Range	2 – 5	2 – 6
Body condition score[1,3]	3.0 (2.75–3.5)	3.0 (3.0–3.25)
Range	2.5 – 3.5	2.75 – 3.75
Rectal temperature, ° F[2]	101.6 (1.06)	101.6 (1.04)
Range	99.7 – 103.8	98.7 – 103.0
Whole blood ionized calcium, mmol/L[2]	1.10 (0.10)	1.05 (0.11)
Range	0.92 – 1.24	0.80 – 1.22
Urine pH[2]	6.00 (0.37)	5.84 (0.33)
Range	5.47 – 6.60	5.39 – 6.40

[1]Median (interquartile range)

[2]Mean (standard deviation)

[3]Body condition score was measured on a scale from 1 – 5, with 1 being emaciate and 5 being obese.