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Comparison of 0.46% calcium diets with and without added anions with a 0.7% calcium anionic diet as a means to reduce periparturient hypocalcemia

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ABSTRACT

Most studies demonstrating that diets with low dietary cation-anion difference (DCAD) reduce hypocalcemia in cows add enough anions to the diet to reduce urine pH below 7.0. One objective of these experiments was to determine whether there is any benefit to periparturient plasma Ca concentration if diet anion addition results in a lesser degree of acidification of the cow and urine pH does not go below 7.0. Another method for reducing hypocalcemia involves feeding a prepartal diet that is Ca deficient. This places the cow in negative Ca balance before calving, stimulating parathyroid hormone (PTH) and 1,25-dihydroxyvitamin D secretion before calving and thus promoting Ca homeostasis at calving. As practiced in the field, low-Ca diets are often about 0.5% Ca. Our second objective was to determine whether a 0.46% Ca diet would be sufficiently low in Ca to stimulate PTH secretion before calving. A meta-analysis of the literature suggests that a 0.5% Ca, low-DCAD diet will reduce hypocalcemia better than a 0.7% Ca diet. A third objective was to compare periparturient plasma Ca in cows fed 0.46 or 0.72% Ca diets with similar DCAD. In experiment 1, anions (primarily chloride) or anions plus Ca were added to a 1.4% K basal diet to create the following diets: 0.46% Ca and +167 mEq/kg of DCAD, 0.46% Ca and −13 mEq/kg of DCAD, and 0.72% Ca and −17 mEq/kg of DCAD. In experiment 2, the same amounts of anion were added to a 2.05% K basal diet to create the following diets: 0.46% Ca and +327 mEq/kg of DCAD, 0.46% Ca and +146 mEq/kg of DCAD, and 0.72% Ca and +140 mEq/kg of DCAD. In experiment 1, cows fed the diet with 0.46% Ca and +167 mEq/kg of DCAD had significantly lower plasma Ca concentration after calving than cows fed the 0.46 or 0.72% Ca diets with anions. Periparturient plasma Ca concentrations did not differ in cows fed the low-DCAD diets with 0.46 or 0.72% Ca. Urine pH was reduced from 8.27 in the diet with 0.46% Ca and +167 mEq/kg of DCAD to 7.07 and 7.41 in the 0.46 and 0.72% Ca anion diets, respectively. Precalving plasma PTH and 1,25-dihydroxyvitamin D concentrations were similar in cows fed the 0.46% Ca diets and the 0.72% Ca diets, suggesting that the 0.46% Ca diets were not low enough in Ca to place the cow in negative Ca balance before calving. In experiment 2, adding the anion supplements to a 2.05% K diet did not reduce urine pH below 8.0. Periparturient plasma Ca concentrations did not differ in cows in any group in experiment 2. Precalving diets that are 0.46% Ca fed ad libitum are too high in Ca to stimulate Ca homeostasis before calving. Adding anions to a diet can benefit periparturient cow plasma Ca concentration, but only if it alters acid–base status enough to reduce urine pH below 7.5.

Key words: hypocalcemia, anion, dietary cation-anion difference, milk fever

INTRODUCTION

Ender et al. (1971) elegantly demonstrated that adding anions to the precalving diet of cows to reduce DCAD, which they defined as (mEq of Na + mEq of K) − (mEq of Cl + mEq of S) in the diet, improved Ca status and effectively prevented milk fever. Numerous studies (Block, 1984; Oetzel et al., 1988; Gaynor et al., 1989) have corroborated and refined those observations. Cows fed high-DCAD prepartum diets are in a state of compensated metabolic alkalosis. This reduces the sensitivity of target tissues such as bone and kidney to parathyroid hormone (**PTH**), compromising Ca homeostasis (Goff et al., 2014). Adding sufficient anions creates a lower DCAD diet and induces a compensated metabolic acidosis in the cow, improving tissue sensitivity to PTH. This restores the competency of Ca homeostatic mechanisms and facilitates a rapid return to normocalcemia after blood Ca concentration decreases at the onset of lactation. However, several practical questions remain about the best practices to use when adding anions to diets to prevent hypocalcemia.

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One question revolves around the proper level of Ca to include in the diet of the cow fed anions. Many studies using anions fed before parturition to reduce milk fever incidence used Ca chloride and Ca sulfate as sources of anions (Dishington, 1975; Block, 1984; Oetzel et al., 1988; Beede et al., 1991). Meta-analyses of factors associated with hypocalcemia at calving suggest that the highest incidence of milk fever occurs when diet Ca is 0.8 to 1.3% Ca (Oetzel, 1991; Lean et al., 2006). The Lean et al. (2006) model suggests that milk fever incidence should increase from 1 to 2% of cows when diet Ca increases from 0.5 to 0.7%. Interestingly, many of the early successful anionic diet studies used diets that were between 0.75 and 1.3% Ca (Dishington, 1975; Oetzel et al., 1988; Gaynor et al., 1989). One of the objectives of these studies was to test the hypothesis that a 0.7% Ca anionic diet would not reduce hypocalcemia at calving as well as a 0.5% Ca anionic diet.

Another strategy for reducing periparturient hypocalcemia involves limiting diet Ca (or diet Ca availability) to levels that place the cow in negative Ca balance for 7 to 14 d before calving. Negative Ca balance stimulates the parathyroid gland to secrete PTH within 3 to 4 d of the diet Ca reduction, and it remains elevated until calving (Goings et al., 1974). This prolonged exposure to high levels of PTH is able to overcome the tissue resistance to PTH caused by metabolic alkalosis (Goff et al., 1986). This initiates Ca conservation and Ca mobilization mechanisms, such as renal production of 1,25-dihydroxyvitamin D [**1,25-$(OH)_2$D**] and bone resorption, before the onset of lactation (Boda and Cole, 1954; Goings et al., 1974; Green et al., 1981). Prolonged PTH exposure causes osteoclast numbers to increase in the bone before calving even when the cow is in metabolic alkalosis. Recruitment of osteoclasts is a process that can take several days of exposure to elevated PTH (Goff et al., 1986; Liesegang et al., 1998). The cow's homeostasis mechanisms are primed by the low-Ca diet and ready to respond to the Ca demands of lactation. There is some debate in the field about the diet Ca concentration that constitutes a low-Ca diet.

Utilizing NRC (2001) calculations, a 600-kg dry cow in late gestation can meet maintenance and fetal requirements for Ca by absorbing 17 g of Ca from her diet each day. The availability of Ca in forages may be as low as 30%. To meet this cow's requirements, the diet would have to contain at least 57 g of Ca if forage supplied all the Ca. This same cow is estimated to consume about 14 kg/d of DM in the weeks before calving. Distributing 57 g of Ca across 14 kg of DM indicates that a diet can be as low as 0.41% Ca—all from forage—and still meet the Ca requirement of the animal. The original studies in which milk fever was prevented using the low-Ca precalving diet strategy had total diet Ca below 18 g/d (Boda and Cole, 1954; Goings et al., 1974; Green et al., 1981; Kichura et al., 1982) and likely supplied just 5 to 10 g of absorbable Ca each day (NRC, 2001). It is difficult to feed prepartum diets that are this low in Ca using typical forages used in the United States, and when cows in the United States are fed ad libitum, their DMI will often ensure that their Ca requirements in late gestation are met or exceeded. Yet diets with more than 0.41% Ca continue to be promoted by some nutritionists as a low-Ca diet approach to milk fever prevention. Perhaps the NRC (2001) calculations are inaccurate. One objective of these studies was to test the hypothesis that a diet that was about 0.46% Ca could effectively stimulate PTH and 1,25-$(OH)_2$D production before calving and reduce hypocalcemia.

Another practical question revolves around the degree of acidification needed to reduce the degree of hypocalcemia experienced by a cow. Urine pH generally reflects blood pH status and has been used to assess the acidifying activity of the anion diet being fed. Most of the published studies have had an anion-supplemented group of cows with urine pH below 7.0 and usually closer to 6.0 (Jardon, 1995; Charbonneau et al., 2006). The periparturient Ca status of those cows was typically contrasted with that of a control "no anion" group. Control cows were generally in a state of metabolic alkalosis, with urine pH $\geq$8.0. Another objective of these studies was to determine whether anion addition improves periparturient plasma Ca concentration, even if there is no or only partial acidification of the cow.

MATERIALS AND METHODS

All procedures used on the cows in this study were approved by the Iowa State University (Ames) Institutional Animal Care and Use Committee.

Experiment 1

Sixty Holstein cows from the Iowa State University dairy herd entering their second or greater lactation were enrolled in this study to test the hypothesis that a 0.46% Ca diet would stimulate Ca homeostatic mechanisms before calving. It also tested the hypothesis that anion supplementation will still benefit periparturient Ca status even if urine pH remains above recommended levels of 6.5 or less (Goff, 2014). Twenty cows were sequentially assigned to each of 3 treatments: 0.46% Ca and +167 mEq/kg of DCAD (**LC**), 0.46% Ca and −13 mEq/kg of DCAD (**LC+A**), or 0.72% Ca and −17 mEq/kg of DCAD (**HC+A**). Cows were assigned