

Update on DCAD for Dry and Lactating Cows

T. R. Overton
Department of Animal Science
Cornell University

Introduction

The concept of managing the dietary cation-anion difference for improved health and performance of dairy cattle has existed for more than 30 years (Horst et al., 1997; NRC, 2001). It is well-established that decreasing the dietary cation-anion difference in the diet fed during the prepartum period improves calcium status and decreases risk of hypocalcemia during the immediate postpartum period (Goff, 2014). In addition, a meta-analysis conducted by Hu and Murphy (2004) suggested that increasing the dietary DCAD of diets fed during lactation increased milk yield and dry matter intake (DMI). The focus of this paper is to provide an update regarding the most recently available information related to the application of DCAD in diets for both dry and lactating dairy cows.

DCAD During the Prepartum Period to Decrease Hypocalcemia

As indicated above, decreasing the DCAD $[(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{S}^{2-})]$ of the diet fed during the last several weeks before calving decreases hypocalcemia. Several mechanisms are likely responsible for this, including increased calcium flux related to increased urinary calcium excretion and increased sensitivity of tissues to parathyroid hormone for cows fed acidogenic diets (Wilkens et al., 2020). Research from our laboratory demonstrated that further decreasing the DCAD of a low K^+ control diet using anionic supplements linearly increased plasma calcium levels and linearly increased postpartum DMI and milk yield (Leno et al., 2017). Researchers at the University of Florida fed either positive or negative DCAD diets with two different sources of Vitamin D (cholecalciferol or calcidiol) during the prepartum period (Martinez et al., 2018; Rodney et al., 2018). Feeding negative DCAD prepartum increased postpartum circulating concentrations of total and ionized calcium, but did not affect postpartum DMI or milk yield; source of Vitamin D did not affect circulating calcium concentrations, but calcidiol supplementation prepartum increased postpartum milk yield.

Subsequent work by Lopera et al. (2018) sought to determine whether relationships existed between degree of acidification with anions and duration of feeding with outcomes. They fed diets with DCAD of -7 mEq/100 g DM (actual urine pH ~6.5) or -18 mEq/100 g DM (actual urine pH ~ 5.6) for either 21 or 42 d prepartum. Feeding the more negative DCAD diet decreased prepartum DMI, increased blood ionized Ca concentrations on the day of calving, and did not affect postpartum performance. Extending the duration of feeding did not affect blood calcium but decreased milk yield by 2.5 kg/d. These results differ from those of Weich et al. (2013) and Wu et al. (2015) who reported that extending the duration of feeding negative DCAD diets up to 42 d before

calving did not affect postpartum outcomes; however, in both of these studies the urine pH were more similar to the cows fed the -7 mEq/100 g DM diet in the Lopera et al. study.

Recently, two meta-analyses have been published that provide updates to previously conducted meta-analyses exploring the effects of DCAD and other macrominerals on hypocalcemia and performance. Santos et al. (2019) assembled a dataset including 42 experiments with 134 treatment means and 1,803 cows (including 5 experiments with 15 treatment means and 151 nulliparous cows) and evaluated relationships with outcomes. They developed a model to compare the estimated effects of decreasing the DCAD from +20 to -10 mEq/100 g of DM. Using this model, decreasing the DCAD resulted in a predicted decrease in DMI of 0.7 and 0.4 kg/d for nulliparous and parous cows, respectively. An interaction of parity and prepartum DCAD was present such that parous cows fed the negative DCAD produced 1.7 kg/d more milk postpartum; whereas milk production in nulliparous cows was not affected by prepartum DCAD. The more negative prepartum DCAD was predicted to increase postpartum blood Ca, decrease postpartum beta-hydroxybutyrate concentrations, and decrease incidence of milk fever, retained placenta, and metritis.

Lean et al. (2019) assembled a dataset including a maximum of 31 experiments, 58 comparisons, and a total of 1,571 cows with the objective of exploring the effects of reducing DCAD intake on outcomes. Treatments reflecting the lower DCAD intake had lower urine pH, lower DMI, increased postpartum DMI, and increased milk yield, although an interaction for parity existed for milk yield. Consistent with the Santos meta-analysis, treatments reflecting the lower DCAD intake decreased risk for clinical hypocalcemia and retained placenta and lowered the odds of metritis. Both meta-analyses highlighted the relative lack of data on the relationships of prepartum dietary DCAD on outcomes in nulliparous cows.

Dietary Calcium Supplementation with Low DCAD Prepartum Diets

Dietary calcium supplementation strategies in conjunction with low DCAD diets fed during the prepartum period continues to be an active area of discussion and debate in the industry. Moore et al. (2000) reported that concurrently decreasing the DCAD (+15, 0, -15 mEq/100 g) and increasing dietary calcium concentration (0.44, 0.97, 1.50% of DM) improved blood calcium status postpartum; however, the effects of DCAD and calcium supplementation cannot be separated in their experiment. Diehl et al. (2018) fed cows either moderate (~ -2.4 mEq/100 g) or low (~ - 21 mEq/100 g) DCAD diets with either 1.3% or 1.8% calcium during the prepartum period. Few differences in circulating Ca concentrations or performance were observed, except that cows fed 1.8% Ca had higher circulating Ca concentrations at d 1 postpartum and cows fed low DCAD made more milk after 45 DIM.

Recently, Glosson et al. (2020) fed cows either a non-acidogenic positive DCAD diet (+6 mEq/100 g; average urine pH ~ 8.1), or two negative DCAD diets (-24 mEq/100 g; average urine pH ~5.75) fed with either low dietary calcium (0.40% of DM) or high dietary calcium (2.0% of DM) for the last 28 d prior to calving. Feeding negative DCAD

slightly decreased prepartum DMI, increased both ionized and total calcium concentrations in blood directly after calving and 24 h after calving, and increased total calcium concentrations at 48 h postcalving. Postpartum DMI (% of body weight) tended to be increased for cows fed negative DCAD prepartum, but milk production was not affected by treatment.

Finally, Amundson et al. (2018) created an experimental model of hypocalcemia in nonlactating, nonpregnant cows and evaluated three different calcium feeding levels (0.45, 1.13, 2.02% of DM) in conjunction with low DCAD (-18 mEq/100 g DM). Hypocalcemia was induced by intravenous infusion of a Ca-specific chelator, EGTA. Cows fed the highest calcium concentration maintained higher circulating concentrations of ionized calcium during the challenge period, took longer to reach 60% of baseline circulating calcium concentrations, and required more EGTA to reach 60% of baseline concentrations, suggesting that cows fed higher calcium levels.

Increased Dietary DCAD for Lactating Cows

As described above, the meta-analysis conducted by Hu and Murphy (2004) was the first to summarize performance responses of lactating cows in the context of varying DCAD, and suggested curvilinear responses of both DMI and milk yield (along with fat-corrected milk) to increasing dietary DCAD. However, closer examination of most of the experiments included in this meta-analysis reveals that, in many cases, cations were added in the form of compounds with known rumen buffering capacity (e.g., sodium bicarbonate, potassium carbonate, potassium bicarbonate). Furthermore, for many of the low DCAD treatments included in the dataset, the anion concentrations were increased through addition of calcium chloride and ammonium chloride, and do not represent diets that would typically be fed to lactating dairy cows.

Harrison et al. (2012) reported that increasing dietary K from 1.3 to 2.1% of DM using a commercially available potassium carbonate sesquihydrate source increased milk fat percentage and tended to increase fat-corrected milk yield. Evidence in this study for a ruminal effect of treatment is the decreased content of *trans*-10 C18:1 in milk fat, which is correlated negatively with milk fat content (McCarthy et al., 2019). This would suggest a ruminal effect either related to potassium or to the increased buffering provided by the treatment.

Iwaniuk et al. (2015) conducted three experiments to evaluate the effects of cation addition and source of cations in diets for lactating cows. In experiment 1, they added 4, 9, and 13 mEq/100 g of DM from potassium carbonate to a basal diet containing +16 mEq/100 g of DM $[(Na^+ + K^+) - (Cl^- + S^{2-})]$. Neither milk yield nor DMI were affected by treatment; however, feeding increasing amounts of potassium carbonate linearly increased milk fat percentage and yield, and increased yields of 3.5% fat-corrected milk. In experiment 2, they added 11, 23, and 35 mEq/100 g of DM from potassium carbonate to a basal diet containing +19 mEq/100 g of DM. Dry matter intake was increased linearly by increasing amounts of potassium carbonate. Milk yield was not affected by treatment, but again potassium carbonate supplementation linearly increased milk fat percentage

and quadratically increased yields of milk fat and 3.5% fat-corrected milk. In experiment 3, they fed four diets with very similar DCAD, but varied the proportions of potassium and sodium by varying the proportions of potassium carbonate sesquihydrate and sodium sesquicarbonate in the diet. Neither DMI nor milk yield were affected by treatment, but as cows were fed more sodium sesquicarbonate, milk fat percentage and yield were increased linearly.

Catterton and Erdman (2016) fed lactating cows a basal diet of +20 mEq/100 g DM or diets supplemented with about 34 mEq/100 g of Na from NaCl, 34 mEq/100 g of K from KCl, 34 mEq/100 g from sodium bicarbonate, or 34 mEq/100 g from potassium carbonate, resulting in calculated DCAD for the supplemented diets of 20, 19, 54, and 54 mEq/100 g, respectively. There was a significant effect of DCAD on rumen pH such that cows fed sodium bicarbonate and potassium carbonate had higher rumen pH than the other three treatments, and an effect of anion in that cows fed KCl and NaCl had lower rumen pH than those fed bicarbonate or carbonate.

In summary, the original meta-analysis conducted by Hu and Murphy (2004) characterized relationships of DCAD with performance outcomes, it appears that relationships observed are likely to be the consequence of supplementation of sodium and potassium sources that also have a buffering role in moderating or increasing rumen pH, although work in continuous culture fermenters by Jenkins et al. (2014) suggests that there may be an effect on ruminal fermentation of potassium independent of ruminal pH. The studies with very low DCAD that are primarily responsible for the curvilinear response surfaces had high levels of anion supplementation that are not representative of diets fed to lactating dairy cattle.

Summary

Decreasing the DCAD of the prepartum diet fed to dairy cattle is effective at improving postpartum calcium status as well as milk yield in general. Recent work suggests that feeding lower DCAD levels that result in urine pH values around 5.5 may be detrimental if continued beyond the typical 21 to 28 day close-up period. Questions remain around how aggressive nutritionists should be in their DCAD and urine pH targets as well as appropriate dietary calcium supplementation levels. Recent work in model systems suggest that higher dietary calcium in the context of low DCAD diets may result in improved calcium status. Effects of sodium and potassium supplementation in lactating cow diets have long been rationalized in the context DCAD; however, the available information suggests that it is much more likely that the sources fed (i.e., carbonate, bicarbonate, or sesquicarbonate) are having effects as rumen buffers rather than a specific effect on postabsorptive acid-base balance, although there may also be a role for potassium in rumen microbial fermentation.

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