J. Dairy Sci. 101:1–15
https://doi.org/10.3168/jds.2018-14748
© American Dairy Science Association®, 2018.

Effect of dietary cation-anion difference on acid-base status and dry matter intake in dry pregnant cows

R. Zimpel,*† M. B. Poindexter,* A. Vieira-Neto,*† E. Block,‡ C. D. Nelson,* C. R. Staples,* W. W. Thatcher,*† and J. E. P. Santos*†[1]
*Department of Animal Sciences, and
†DH Barron Reproductive and Perinatal Biology Research Program, University of Florida, Gainesville 32611
‡Arm and Hammer Animal Nutrition, Princeton, NJ 08540

ABSTRACT

The objective was to determine if the reduction in dry matter (DM) intake of acidogenic diets is mediated by inclusion of acidogenic products, content of salts containing Cl, or changes in acid-base status. The hypothesis was that a decrease in intake is mediated by metabolic acidosis. Ten primigravid Holstein cows at 148 ± 8 d of gestation were used in a duplicated 5 × 5 Latin square design. The dietary cation-anion difference (DCAD) of diets and acid-base status of cows were manipulated by incorporating an acidogenic product or by adding salts containing Cl, Na, and K to the diets. Treatments were a base diet (T1; 1.42% K, 0.04% Na, 0.26% Cl; DCAD = 196 mEq/kg); the base diet with added 1% NaCl and 1% KCl (T2; 1.83% K, 0.42% Na, 1.23% Cl; DCAD = 194 mEq/kg); the base diet with added 7.5% acidogenic product, 1.5% $NaHCO_3$, and 1% K_2CO_3 (T3; 1.71% K, 0.54% Na, 0.89% Cl; DCAD = 192 mEq/kg); the base diet with added 7.5% acidogenic product (T4; 1.29% K, 0.13% Na, 0.91% Cl; DCAD = −114 mEq/kg); and the base diet with 7.5% acidogenic product, 1% NaCl, and 1% KCl (T5; 1.78% K, 0.53% Na, 2.03% Cl; DCAD = −113 mEq/kg). Periods lasted 14 d with the last 7 d used for data collection. Feeding behavior was evaluated for 12 h in the last 2 d of each period. Reducing the DCAD by feeding an acidogenic product reduced blood pH (T1 = 7.450 vs. T2 = 7.436 vs. T3 = 7.435 vs. T4 = 7.420 vs. T5 = 7.416) and induced a compensated metabolic acidosis with a reduction in bicarbonate, base excess, and partial pressure of CO_2 in blood, and reduced pH and strong ion difference in urine. Reducing the DCAD reduced DM intake 0.6 kg/d (T1 = 10.3 vs. T4 = 9.7 kg/d), which was caused by the change in acid-base status (T2 + T3 = 10.2 vs. T4 + T5 = 9.6 kg/d) because counteracting the acidifying action of the acidogenic product by adding salts with strong cations to the diet prevented the decline in intake. The decline in intake caused by metabolic acidosis also was observed when adjusted for body weight (T2 + T3 = 1.75 vs. T4 + T5 = 1.66% BW). Altering the acid-base status with acidogenic diets reduced eating (T2 + T3 = 6.7 vs. T4 + T5 = 5.9 bouts/12 h) and chewing (T2 + T3 = 14.6 vs. T4 + T5 = 13.5 bouts/12 h) bouts, and extended meal duration (T2 + T3 = 19.8 vs. T4 + T5 = 22.0 min/meal) and intermeal interval (T2 + T3 = 92.0 vs. T4 + T5 = 107.7 min). Results indicate that reducing the DCAD induced a compensated metabolic acidosis and reduced DM intake, but correcting the metabolic acidosis prevented the decline in DM intake in dry cows. The decrease in DM intake in diets with negative DCAD was mediated by metabolic acidosis and not by addition of acidogenic product or salts containing Cl.

Key words: acid-base status, dietary cation-anion difference, dry cow, dry matter intake

INTRODUCTION

Hypocalcemia is an important metabolic disease that affects dairy cattle around parturition because many cows are unable to compensate the irreversible loss of Ca required for synthesis of colostrum and milk at the onset of lactation. Goff (2008) stated that clinical and subclinical hypocalcemia are considered gateway diseases that impair health and reduce productivity in the ensuing lactation. In fact, cows that develop clinical and subclinical hypocalcemia have increased risk of numerous other diseases in early lactation (Curtis et al., 1983; Martinez et al., 2012). Therefore, prevention of hypocalcemia should be a major goal of transition cow feeding programs.

A dietary strategy to minimize the risk of clinical and subclinical hypocalcemia is the manipulation of the mineral content of prepartum diets to induce a compensated metabolic acidosis by feeding acidogenic diets.

Received March 14, 2018.
Accepted May 17, 2018.
[1]Corresponding author: jepsantos@ufl.edu

ARTICLE IN PRESS

Prepartum diets with negative DCAD reduce clinical and subclinical hypocalcemia in dairy cows (Ender et al., 1971; Block, 1984; Martinez et al., 2018b), and the prevention of hypocalcemia by acidogenic diets also reduces the risk of retained placenta and metritis and improves lactation performance in parous cows (Santos et al., 2018).

According to the USDA (2016), at least 28% of the dairy farms in the United States feed prepartum acidogenic diets to prevent hypocalcemia. The recommended range of DCAD in diets for prepartum cows has been suggested as −50 to −150 mEq/kg (NRC, 2001), although the ideal DCAD has not been established to optimize health and performance. One issue with acidogenic diets is the depression in DMI (Charbonneau et al., 2006). Numerous experiments have shown that reducing the DCAD of prepartum diets depresses DMI in dairy cows (Joyce et al., 1997; Lopera et al., 2018; Martinez et al., 2018a). Charbonneau et al. (2006) calculated the DCAD assuming that K, Na, and Cl are equally absorbed, but absorption of S is only 60% of those of K, Na, and Cl. According to their calculations, a reduction in DCAD of 300 mEq/kg (i.e., from 200 to −100 mEq/kg of DM) would result in a depression in DMI of 1.3 kg/d. The same diets calculated with the DCAD equation considering S with equal bioavailability to the other elements in the equation would result in a greater difference in DCAD. Theories proposed to explain the depression in DMI when diets with negative DCAD are fed include the unpalatable effect of the salt sources (Oetzel et al., 1988; Oetzel and Barmore, 1993) or a response to the metabolic acidosis induced by the strong anions incorporated into the diet (Vagnoni and Oetzel, 1998). Nonetheless, neither of those theories has been evaluated specifically.

Recently, we showed that the reduction in DMI in prepartum cows fed diets with acidogenic products was observed regardless of the product fed, either an acidogenic salt or a commercial product (Santos et al., 2018), suggesting that metabolic acidosis and not palatability might be the reason for the reduced intake. We hypothesized that depression in DMI induced by feeding diets with negative DCAD is mediated by changes in acid-base status and not because of inclusion of acidogenic product or salts containing Cl. Thus, the objective was to determine if the decrease in DMI in cows fed acidogenic diets is mediated by the addition of acidogenic product, inclusion of salts containing Cl, or induced by changes in acid-base balance.

MATERIALS AND METHODS

The experiment was conducted at University of Florida Dairy Unit (Hague, FL) from January to April of 2017 and all procedures with cows were approved by the Institutional Animal Care and Use committee of the University of Florida, protocol number 201709707. Throughout the manuscript, the terms salts containing Cl or Cl salts refer to the addition of NaCl and KCl, which increased dietary Cl content but were expected not to alter the acid-base status of cows.

Cows, Housing, and Feeding

Ten primigravid Holstein cows were used. Cows averaged (±SD) 562 ± 32 d of age, 148 ± 8 d pregnant, and 523 ± 29 kg of BW at enrollment. At completion of the 70-d experiment, cows averaged 632 d of age, 218 d pregnant, and 624 ± 31 kg of BW.

Cows were housed in a cross-ventilated tiestall barn with individual feed bins and water troughs equipped with flow meters. Cows were moved to the experimental facilities 2 wk before the beginning of the experiment to acclimate to the new environment and daily handling. Stalls had air mattresses bedded with sand, and bedding material was cleaned 4 times a day and replaced thrice a week. Cows were fed twice daily at 0800 and 1800 h, and the amounts of feed offered to individual cows were adjusted once daily to ensure at least 5% orts, which were weighed before the morning feeding. Twice weekly, dietary ingredients were sampled and dried at 55°C for adjustment of amounts of DM to be offered daily.

Experimental Design and Treatments

The experiment was a replicated 5 × 5 Latin square design with treatment sequence balanced for carry-over. The 10 cows were stratified based on BW at enrollment and the 5 heaviest cows were assigned to one square, whereas the remaining 5 cows to the second square. The experiment consisted of periods of 14 d, in which d 1 to 7 were for adaptation to dietary treatments and d 8 to 14 were used for data collection.

The diets were offered as TMR and formulated to meet or exceed the metabolizable energy and protein requirements of a 550 kg primigravid Holstein cow to gain at least 1.2 kg/d including conceptus when DMI averages 10 kg/d (NRC, 2001). The DCAD (mEq/kg of DM) was calculated assuming exactly the same absorption for the strong ions with the following formula: DCAD = [(mEq of K^+ + mEq of Na^+) − (mEq of Cl^- + mEq of S^{2-})]. Ingredients were analyzed a priori to design diets to contain either 200 or −100 mEq/kg, a difference of 300 mEq/kg of DM. After diets were prepared and analyzed, there were minor deviations from the expected DCAD and mineral concentrations. Concentrates and forages were the same in all diets,

Download English Version:

https://daneshyari.com/en/article/8956501

Download Persian Version:

https://daneshyari.com/article/8956501

Daneshyari.com