Altering Dietary Cation-Anion Difference in Lactating Dairy Cows to Reduce Phosphorus Excretion to the Environment

S. I. Borucki Castro,¹ L. E. Phillip,¹ V. Girard,² and A. Tremblay²

¹Department of Animal Science,
McGill University, Ste-Anne-de-Bellevue, Quebec, Canada H9X 3V9

²Département de Biomédecine Vétérinaire, Université de Montréal,
St-Hyacinthe, Quebec, Canada, J2S 7C6

ABSTRACT

Four early-lactating dairy cows were randomly allocated to 4 diets with dietary cation-anion difference [DCAD; $(\text{Na} + \text{K}) - (\text{Cl}^- + \text{S}^{2-})$ mEq/100 g dry matter] values of +14, +18, +24, and +45. Diets were formulated to be isoenergetic and isonitrogenous, and supplied similar levels of P (0.46%) and Ca (0.77%). The salts, MgCl_2 , MgSO_4 , K_2CO_3 , and NaHCO_3 were used to alter DCAD. The main objective of the study was to ascertain whether a decrease in DCAD would reduce fecal P excretion in lactating dairy cattle. The experiment was conducted as a 4×4 Latin square design with 21-d periods. During the last 5 d, diets were offered at a restricted level and samples of blood, milk, feces, and urine were collected. Measures of acid-base status of the cows were linearly related to DCAD, but the animals did not experience metabolic acid stress. Neither fecal P nor urinary P was affected by DCAD, and there was no change in overall P balance. Plasma P tended to increase and blood concentrations of ionized Ca were enhanced as DCAD decreased; P excretion in milk showed a quadratic response to DCAD. Milk yield and milk composition were unaffected by changes in DCAD. Although DCAD may have influenced P homeostasis in lactating cows, there was no evidence that, within the range of + 14 to + 45 mEq/100 g dry matter, DCAD could be used as a nutritional strategy to reduce manure P from dairy cattle.

(Key words: phosphorus excretion, cation-anion difference, dairy cattle, acid-base balance)

Abbreviation key: DCAD = dietary cation-anion difference, MP = metabolizable protein.

INTRODUCTION

In recent years, over-fertilization and manure application to agricultural soils have led to phosphorus (P) be-

coming a significant source of environmental pollution in North America. Due to surface runoff, erosion of soil particles (Simard et al., 2000), and leaching from saturated soils (Jamieson et al., 2003), loss of P to surface water bodies has been a major contributor to eutrophication of lakes and estuaries (Carpenter et al., 1998; Correll, 1998). The problem of eutrophication is particularly acute in areas of intensive livestock production. In Canada, for example, the provinces of Quebec and Ontario have the highest density of farm animals and account for two-thirds of the watersheds with the greatest concentrations of manure P (Statistics Canada, 2003). Within Quebec, dairy farming accounts for about 30% of the total manure P output from livestock (Statistics Canada, 2001), and this has led to the need for strategies to reduce manure P output from dairy cattle.

Feces represent the major route of P excretion in ruminants (Scott, 1988; Challa et al., 1989), and, for this reason, most nutritional strategies to reduce manure P from dairy cattle focus on reductions in fecal P (Van Horn et al., 1996; Tamminga, 1996; Valk et al., 2000). Feces contain both particulate and dissolved P, but urine, the other component of manure, contains only dissolved P, a form that is readily available for eutrophication (Nurnberg and Peters, 1984). Therefore, in any investigation of feeding strategies to reduce environmental pollution due to manure P, the excretion of urinary P should be monitored (Braithwaite, 1984; Scott and Buchan, 1988; Bravo et al., 1998).

Prior investigations have shown that DCAD has a major impact on Ca homeostasis in lactating cows (Delaquis and Block, 1995; Horst et al., 1997), but there is a scarcity of research on the impact of DCAD on P homeostasis and P excretion in dairy cattle. The aims of this research were to ascertain whether a decrease in DCAD would reduce fecal P excretion by lactating dairy cows and to evaluate the impact of changes in DCAD on the partition of P between feces and urine.

MATERIALS AND METHODS

Animals, Experimental Diets, and Design

The study was conducted at the Centre de Recherche en Science Animales de Deschambault (CRSAD) of the

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Corresponding author: L. E. Phillip; e-mail: Leroy.Phillip@McGill.ca.

Institut de Recherche et de Développement en Agroenvironnement (IRDA) in Deschambault, Quebec. Animal care procedures followed the guidelines of the Canadian Council on Animal Care (CCAC, 1993), and the protocol was approved by the Animal Care Committee of the CRSAD-IRDA. The experiment involved the use of 4 multiparous, nonpregnant Holstein dairy cows (average BW 630 ± 50 kg) in early lactation (60 ± 10 d from calving). The study was conducted as a 4×4 Latin square with four 21-d periods, each consisting of 16 d for adaptation and 5 d for excreta collection and sampling. During the first 16 d the animals were fed ad libitum. They were then restricted to 85% of ad libitum intake to ensure the total consumption of the respective DCAD during the collection period.

The cows were randomly assigned to 4 dietary treatments with the following levels of DCAD, +14, +18, +24, and +45 mEq/100 g DM. The diets were provided as a TMR consisting of corn silage, grass silage, chopped hay, cracked corn grain, rolled barley grain, soybean meal, a commercial protein supplement, and a commercial vitamin-mineral mixture. The salts, K_2CO_3 , $NaHCO_3$, $MgCl_2(6H_2O)$, and $MgSO_4(7H_2O)$ were added to the vitamin-mineral mixture to achieve the appropriate DCAD values. The MgO was added to +24 and +45 DCAD treatments to meet Mg requirements. The purity of the added salts was as follows: K_2CO_3 , 99% (Fisher Scientific, Fairlawn, NJ); $NaHCO_3$, 100% (Church & Dwight Co. Inc., Princeton, NJ); $MgCl_2(6H_2O)$, 96% anhydrous basis (Peters Chemical Co., Hawthorne, NJ); and $MgSO_4(7H_2O)$, 99% anhydrous basis (PQ Corp., Etobicoke, ON). Diet concentrations of NE_L , MP, NDF, calcium (Ca), and P were adjusted for milk production (NRC, 2001). The animals were housed individually in tie stalls and fed twice daily, in equal portions, at 0700 and 1800 h; all cows had free access to water.

Sampling Procedure and Measurements

The amounts of feed offered and refused were recorded. Samples of the diet and refusals (if any) were collected, then dried, and pooled for each day to calculate daily DMI. Total output of feces from each cow was weighed and thoroughly mixed. Fecal samples were obtained 2 times daily, pooled for each day, and frozen for subsequent analysis. Apparent digestibility of P was calculated as: P intake (g/d) – fecal P (g/d) divided by intake P (g/d), and expressed as g/100 g.

Two days before the sampling period, urethral catheters (24 Fr. 0113L24 75cc Bardex Lubricath, C. R. Bard Inc., Covington, GA) were inserted into the bladder of each cow. To collect urine, the catheter was connected, using polyvinylchloride tubing, to a polyethylene bottle (25-L capacity). Thymol (1 g/20 L of urine) was added as

a preservative, and mineral oil (20 mL/20 L of urine) was added to the bottle to prevent loss of urinary gasses (Halperin et al., 1974). Urine volume was recorded 2 times daily, and samples were taken from beneath the layer of mineral oil. Urine pH was recorded immediately with a portable pH meter, and urine samples were pooled for each day, then frozen (-20°C) for subsequent analysis.

To obtain blood samples, 2 catheters (30.5-cm 16 G Intercath Becton Dickinson Vascular Dickinson and Co, Franklin Lakes, NJ) were inserted in the jugular vein of each cow, 2 d before the sampling period. The catheters were filled with heparinized saline solution and secured to the neck of the cow with elastoplast bandage. On d 17, 12 mL of blood was collected into each of 2 evacuated tubes at 30 min before the morning feeding and at 2 and 4 h thereafter. One blood sample was kept in a refrigerator (4°C) and analyzed, within 5 h, with a blood gas analyzer (Stat profile Nova Biochemical, Watham, MA), for pH, pCO_2 , HCO_3 , and ionized calcium; the other blood sample was centrifuged at $3000 \times g$ (4°C) for 20 min and frozen (-20°C) for subsequent analysis of plasma phosphate using a biochemical auto analyzer (Synchron CX5, Beckman Coulter, Brea, CA).

Milk volume was recorded daily during the collection period using Tru-test meters (SURGE, Mississauga, ON). Samples from the morning and afternoon milking were collected in proportion to milk yield. The samples were collected in plastic bottles containing bronopol as a preservative; the samples were pooled for each day, and then analyzed for milk constituents.

Analytical Methods

Samples of the TMR were analyzed for DM using a forced air oven maintained at 60°C for 72 h. Total N in feed samples was determined using the LECO N auto analyzer (LECO Corp., St. Joseph, MI); N in milk, wet feces, and urine was determined using a N auto analyzer (BD Digest Industrial Methods no. 329-74 W/A, Technicon Instruments Corporation, Tarrytown, NY) according to the methods of AOAC (1995) and Isaac and Johnson (1976). Analyses of NDF and ADF were performed according to the methods of Van Soest et al. (1991) using the Ankom System (ANKOM 200, Fiber Analyzer, Fairport, NY) with heat stable alpha-amylase and without sodium sulfite. Ash content was determined according to AOAC (1995) using a muffle furnace (Thermolyne, Sybron Co., Dubuque, IA). Minerals (P, K, Ca, Mg, and Na) in samples of diets, milk, feces, and urine were analyzed using direct current plasma emission spectroscopy (Isaac and Johnson, 1985). Sulfur was determined according to AOAC (1995) using a Perkin Elmer ICP 3000 (Perkin Elmer Corp., Norwalk, CT); chloride was determined according to the AOAC (1995) procedure

using a chloride-specific ion Orion electrode (Orion Research, Beverly, MA).

Urinary bicarbonate and titrable acidity were analyzed using the methodologies of Lin and Chan (1973) and Chan (1972), respectively. Milk concentrations of fat, protein, and lactose were analyzed at the Dairy Herd Analysis Service of PATLQ (Programme d'analyse des troupeaux laitiers du Québec) with an infrared system using an electric Milk-O-Scan 4000 (Foss-Food Technology, Hillerød, Denmark). The instrument was calibrated with reference standards determined by Mojonniér and Kjeldahl methods (AOAC, 1995).

Statistical Analysis

Data were analyzed using the mixed model procedure of the SAS System (version 8.2, Cary, NC) according to Littell et al. (1996). A Latin square classification model was used to obtain least square means for treatments; linear and quadratic effects were tested by fitting DCAD as a regression variable. The following statistical models were adopted:

$$Y_{jk} = \mu + \text{Treatment}_{jk} + \text{Per}_j + A_k + \varepsilon_{jk};$$

$$Y_{jk} = \mu + b_1 \text{DCAD}_{jk} + b_2 \text{DCAD}_{jk}^2 + \text{Per}_j + A_k + \varepsilon_{jk};$$

$$Y_{jk} = \text{variable studied during the } j\text{th period (1, 2, 3, and 4) on the } k\text{th animal, (1, 2, 3, and 4) receiving the treatment DCAD } jk$$

$$\mu = \text{overall mean;}$$

$$\text{Treatment}_{jk} = \text{the effect of the } jk\text{th treatment (} jk = 1, 2, 3, \text{ and } 4\text{);}$$

$$\text{DCAD } jk = \text{DCAD value for the } k\text{th cow during the } j\text{th period;}$$

$$b_1 = \text{linear regression effect;}$$

$$b_2 = \text{quadratic effect;}$$

$$\text{Per}_j = \text{effect of the } j\text{th period;}$$

$$A_k = \text{effect of the } k\text{th cow; and}$$

$$\varepsilon_{jk} = \text{random error on the } jk\text{th measure}$$

The mixed model analysis estimation method was restricted maximum likelihood (REML). A repeated measures procedure was applied only to blood parameters, and time of sampling was added to the model. There were no interactions between DCAD and sampling time for any of the blood parameters. The criterion for declaring an effect to be statistically significant was predetermined at the 5% level; values at 10% level of probability were considered as expressing a tendency.

RESULTS

Table 1 reveals the ingredient and chemical composition of the experimental diets as well as the DCAD levels

Table 1. Ingredient and nutrient composition of diets (% of DM).

	DCAD ¹ (mEq/100 g DM)			
	+14	+18	+24	+45
Ingredient composition				
Corn silage	25.0	25.0	25.4	24.3
Grass silage	26.1	26.1	25.9	25.8
Chopped grass hay	7.1	7.1	7.1	7.0
Rolled barley grain	11.4	11.5	11.5	11.6
Cracked corn grain	14.9	15.0	15.0	15.2
Soy bean meal, 49% CP	9.6	9.6	9.7	9.7
Protein supplement ²	4.2	4.2	4.2	4.2
Vitamin-mineral premix ³	1.6	1.4	1.1	2.3
Salts ⁴				
Magnesium chloride	0.08	0.05	—	—
Magnesium sulfate	0.08	0.05	—	—
Potassium carbonate	—	—	0.02	0.12
Sodium bicarbonate	—	—	0.02	0.14
Nutrient composition:				
CP	17.7	17.3	18.1	17.8
NDF	36.2	35.9	36.6	38.2
ADF	21.3	21.4	21.9	22.2
Ash	7.3	7.3	7.1	7.9
P	0.46	0.46	0.46	0.46
Ca	0.77	0.75	0.77	0.77
Mg	0.32	0.27	0.28	0.28
Na	0.19	0.19	0.22	0.29
K	1.68	1.68	1.71	2.14
Cl	0.74	0.65	0.58	0.32
S	0.26	0.24	0.21	0.20

¹DCAD = Dietary cation-anion difference (Na + K) - (Cl⁻ + S²⁻) mEq/100 g DM.

²Commercial protein supplement (BRETON, St.-Marc-des-Carrières, QC).

³(of total DM): 0.66% Ca, 0.40% P, 0.34% Mg, 0.18% Na, 300 mg/kg of Fe, 60 mg/kg of Mn, 69 mg/kg of Zn, 14 mg/kg of Cu, 0.68 mg/kg of I, 0.48 mg/kg of Co, 17,978 IU/kg of vitamin A, 4,364 IU/kg of vitamin D, and 36 IU/kg of vitamin E.

⁴Amounts of salts provided daily: DCAD 14: 270 g MgCl₂·6H₂O (2660 mEq Cl), 270 g MgSO₄·7H₂O (2195 mEq S); DCAD 18: 75 g of MgCl₂·6H₂O (739 mEq Cl), 75 g of MgSO₄·7H₂O (610 mEq S); DCAD 24: 28 g of K₂CO₃ (406 mEq K), 34 g of NaHCO₃ (405 mEq Na), 39 g of MgO; DCAD 45: 300 g of K₂CO₃ (4348 mEq K), 250 g of NaHCO₃ (2976 mEq Na), and 39 g of MgO.

achieved. The actual DCAD values (+14 ± 1.4; +18 ± 2.6; +24 ± 1.5; +45 ± 4.1 mEq/100 g DM) were higher than those planned (+5, +10, +20, and +40 mEq/100 g DM); this was due mainly to a higher contribution of K from the feed ingredients and lower than required quantities of the salt that supplied S. Measures of blood and urinary acid-base status responded to changes in DCAD (Table 2). There were linear decreases ($P < 0.01$) in blood pH, HCO₃, and base excess as DCAD decreased. The linear reductions ($P < 0.01$) in urinary pH, HCO₃ and titrable acidity were consistent with responses in blood acid-base status. Plasma P concentrations tended ($P < 0.10$) to increase as DCAD decreased (Table 2), and there was an inverse relationship ($P < 0.05$) between blood concentrations of ionized-Ca and DCAD. Table 3 shows regression equations describing the relationships between DCAD and acid-base status, and between DCAD and P

Table 2. Effects of DCAD¹ on blood and urinary acid-base parameters.

	DCAD ¹				SE	n	P =	
	+14	+18	+24	+45			1	q
Blood								
pH	7.428	7.420	7.434	7.452	0.0058	47	<0.001	0.789
pCO ₂ , mm Hg	38.63	37.04	38.00	38.95	0.980	47	0.562	0.872
HCO ₃ , mM	25.75	24.30	25.74	27.46	0.821	47	0.014	0.729
Base excess, mM	2.02	0.66	2.14	3.98	0.778	47	<0.01	0.713
Ionized- Ca, mM	1.11	1.09	1.10	1.04	0.02	47	<0.01	0.742
Plasma phosphate, mM	1.69	1.46	1.38	1.47	0.095	47	0.092	0.190
Urine								
pH	7.73	8.09	8.28	8.36	0.183	16	0.043	0.14
HCO ₃ , mM	70	105	146	258	19.3	16	<0.001	0.23
Titrate acidity, mM	-81	-122	-164	-272	25.5	16	<0.001	0.26

¹DCAD = Dietary cation-anion difference (Na + K) - (Cl⁻ + S²⁻) mEq/100 g DM.

and Ca in blood; graphic representations of the relationship between DCAD and measures of acid-base status are shown in Figures 1 and 2.

The results of P excretion and balance are presented in Table 4 and graphically represented in Figure 3. No differences in P digestibility were detected ($P > 0.10$). As expected, fecal P was the main route of P excretion, followed by milk and urine. There were no effects ($P > 0.10$) of DCAD on fecal P, urinary P, nor P balance; however, P excreted in milk showed a quadratic response ($P = 0.05$) to DCAD. Calcium in urine tended ($P = 0.10$) to increase at lower values of DCAD, but Ca balance was unaffected ($P > 0.10$) by changes in DCAD (Table 4). Milk production and milk composition were not affected ($P > 0.10$) by DCAD (Table 5).

DISCUSSION

The results clearly indicate that DCAD altered the acid-base status of the cows, in that most of the acid-base measurements were inversely related to DCAD. There was no indication that the cows experienced metabolic acidosis, and the linear increase in urinary HCO₃ as DCAD increased is an indication that the kidneys excreted excess HCO₃ thereby preventing metabolic al-

kalosis (Halperin and Goldstein, 1994). The effects of DCAD on blood and urinary acid-base measurements are consistent with studies by Tucker et al. (1988) and West et al. (1991, 1992). Compared with the present study, Tucker et al. (1988) obtained a more pronounced relationship between DCAD and blood HCO₃, but their experiment was conducted over a wider and more anionic range of DCAD than that reported here.

The concept underlying the study was that a reduction in DCAD would induce acidogenic conditions within the animal that would favor P absorption and reduce its excretion. Despite inducing changes in acid-base status of the animals, the main goal of reducing fecal P excretion by reducing DCAD was not achieved. If, as suggested by the apparent increase in plasma P, saliva recycling and fecal excretion of absorbed P did occur (Challa et al., 1989), this may explain why DCAD had no effect on total fecal P. Working with oxen, Beighle et al. (1997) reported a reduction in fecal P excretion by reducing DCAD from +26 to +17 mEq/100 g DM. In studies with dairy cows, however, Delaquis and Block (1995) observed no significant effect on fecal P excretion when DCAD was reduced from +25.8 to +5.5 mEq/100 g DM. In the present study, a DCAD as low as +5 mEq/100 g DM was not achieved, limiting the range of DCAD under investigation. This

Table 3. Regression equations describing the effect of DCAD¹ in lactating dairy cows.

		R ²	$\sigma^2_{e^2}$	P =
Blood pH	y = 7.41 (0.009) + 0.0001 (0.00003) x	0.77	2×10^{-4}	<0.001
Blood HCO ₃ , mM	y = 23.89 (1.23) + 0.008 (0.003) x	0.57	5.338	0.014
Blood base excess, mM	y = 0.08 (1.13) + 0.008 (0.003) x	0.58	4.378	0.002
Blood ionized-Ca, mM	y = 1.14 (0.03) - 0.0028 (0.0001) x	0.80	0.0027	0.002
Plasma phosphate, mM	y = 1.80 (0.13) - 0.0005 (0.003) x	0.51	5×10^{-5}	0.092
Urinary pH	y = 7.7 (0.27) + 0.002 (0.0006) x	0.76	0.109	0.043
Urinary HCO ₃ , mM	y = -3.2 (24.5) + 0.6 (0.06) x	0.94	130.9	<0.001
Urinary titrate acidity, mM	y = -13.5 (33.48) - 0.6 (0.08) x	0.90	1480.8	<0.001

¹x = Dietary cation-anion difference (DCAD): (Na + K) - (Cl⁻ + S²⁻) mEq/ 100 g DM.

² $\sigma^2_{e^2}$ = Residual variance.

Table 4. Least square means of effects of DCAD¹ on P and Ca excretion and balance.

	DCAD ¹				SE (n = 16)	P =	
	+14	+18	+24	+45		l	q
P intake, g/d	83.5	89.1	88.0	84.6	4.12	0.60	0.26
Fecal P, g/d	60.4	65.4	61.6	60.5	4.00	0.48	0.99
Apparent digestibility of P, g /100 g P intake	28.6	26.6	29.8	28.5	2.90	0.83	0.45
Urinary P, g/d	3.2	0.8	0.7	2.4	1.58	0.76	0.25
Milk P, g/d	25.7	29.1	28.6	27.6	1.50	0.05	0.05
P balance, g/d	-5.8	-6.2	-2.9	-5.9	1.43	0.81	0.11
Ca intake, g/d	137.5	146.8	146.7	143.4	6.83	0.85	0.15
Fecal Ca, g/d	105.5	116.6	108.5	109.1	7.99	0.98	0.76
Urinary Ca, g/d	2.8	1.2	0.99	0.64	0.69	0.10	0.17
Milk Ca, g/d	32.8	35.6	34.9	33.4	1.98	0.59	0.09
Ca balance, g/d	-3.7	-6.6	1.3	0.3	6.16	0.55	0.17

¹DCAD = Dietary cation-anion difference (Na + K) - (Cl⁻ + S²⁻) mEq/100 g DM.

may have contributed to our inability to establish a relationship between fecal P and DCAD over a wide range of values.

An effect of DCAD on urinary P excretion was also undetected; this is consistent with the results of Delaquis and Block (1995) with lactating cows and those of Van Mosel et al. (1993) with dry cows. In contrast, West et al. (1991) has shown a cubic response in urinary P to reductions in DCAD (from + 31.2 to -10 to mEq/100 g DM) in lactating cows, while Vagnoni and Oetzel (1998) reported reduced urinary P in dry cows receiving DCAD of -4 and -6 mEq/100 g DM compared with +20 mEq/100 g DM. Such variable responses in urinary P to changes in DCAD suggest that this relationship needs to be further explored.

The apparent increase in plasma P as DCAD decreased suggests that P absorption may have been enhanced. Support for this concept could be adduced from the work of Beighle et al. (1997). In a recent study with dairy cattle, Roche et al. (2003) reported that a decrease in DCAD from +69 to -12 mEq/100 g DM led to an increase in Mg absorption, but no measurements were made of P

absorption or excretion. An alternative explanation for the apparent increase in plasma P is the possibility that, at the low levels of DCAD, there was increased bone turnover through mechanisms involved in P and Ca homeostasis (Abu Damir and Phillip, 1993; Phillip and Reid, 1994; Lobaugh, 1996). In fact, at low values of DCAD blood Ca increased and urinary Ca tended to increase. However, in studies with transition cows, Van Mosel et al. (1993, 1994) and Roche et al. (2003) showed that at low DCAD, blood Ca and Mg did increase without evidence of bone mobilization or changes in plasma P (Van Mosel et al., 1993, 1994). There is a need, therefore, for further research to improve understanding of the relationship between DCAD and P homeostasis in dairy cattle.

Within the range investigated in this study (+14 to +45 mEq/100 g DM), DCAD had no effect on milk production; this is consistent with other reports in which lactation performance was studied over a DCAD range of +25 to +40 mEq/100 g DM (Escobosa et al., 1984; West et al., 1992; Sanchez et al., 1997; Ward and McCormick, 2000). Tucker et al. (1988) reported a positive response in milk

Table 5. Least square means of effects of DCAD¹ on lactation performance of dairy cows.

	DCAD ¹				SE (n = 16)	P =	
	+14	+18	+24	+45		l	q
DMI, kg/d	20.1	21.1	20.6	20.1	0.73	0.43	0.43
Milk yield, kg/d	29.2	32.2	31.3	30.3	1.98	0.89	0.14
FCM, ² 4%	28.9	31.6	30.6	30.1	1.37	0.95	0.28
Milk composition							
Fat, %	3.96	3.88	3.90	4.01	0.234	0.36	0.16
Protein, %	2.91	2.88	2.87	2.86	0.064	0.45	0.54
Lactose, %	4.59	4.63	4.65	4.63	0.053	0.42	0.42
P, mg/L	883	905	911	909	18.2	0.62	0.87
Ca, mg/L	1133	1107	1111	1100	17.4	0.17	0.34
Mg, mg/L	104	104	102	102	2.5	0.17	0.33

¹DCAD = Dietary cation-anion difference (Na + K) - (Cl⁻ + S²⁻) mEq/100 g DM.

²Fat-corrected milk at 4%.

Figure 1. Relationship between blood pH and dietary cation-anion difference. Blood pH (Δ); predicted response (blood pH) (—).

production when DCAD was increased from -10 to $+20$ mEq/100 g DM, and West et al. (1991) observed a positive linear response in milk production when DCAD was increased from -10 to $+31$ mEq/100 g DM. Based on analysis of a large data set covering a DCAD range of $+5$ to

Figure 2. Relationship between urinary HCO_3^- and dietary cation-anion difference. Urinary HCO_3^- ($\blacksquare$); predicted response (urinary HCO_3^-) (—).

Figure 3. Fecal and urinary P excretion and dietary cation-anion difference. Fecal P excretion ($\blacksquare$); Urinary P excretion (Δ).

$+61$ mEq/100 g DM, Sanchez et al. (1994) concluded that milk production was optimized at a DCAD value of $+38$ mEq/100 g DM. In the present study, anionic diets were not utilized nor were very low DCAD values achieved; a relatively narrow DCAD range, a small number of cows, and a short experimental period may have further limited our ability to detect a response in milk production or milk composition.

CONCLUSIONS

Over the range of $+14$ to $+45$ mEq/100 g DM, there was an inverse relationship between DCAD and measures of acid-base status of lactating dairy cows. Despite a tendency for higher concentrations of plasma P at low levels of DCAD, there was no effect of reducing DCAD on P excretion in feces and urine. Therefore, the potential to influence P output in dairy cattle manure by manipulating DCAD was not realized. Neither milk yield nor milk composition was altered by changes in DCAD.

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