

Review

Dietary Cation Anion Balance in the Ruminants II- Effects During Mid Lactation and Dry Period

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ABSTRACT

Acid-base manipulation by dietary cation-anion balance (DCAB) during the dry period is a factor in the induction and prevention of milk fever. The apparent reason that cows do not become paretic with the anionic diet is the maintained serum calcium (Ca^{+2}) and phosphorus levels during Ca^{+2} stress. Prior to parturition, when cows fed the anionic diet, plasma hydroxyproline (index of bone resorption) increases, responding to parathyroid hormone, whereas the reverse is the case with cationic diet just before parturition. In mid-lactation, increasing DCAB can increase dry matter intake (DMI), suggesting a beneficial effect from feeding cationic diet; raise blood pH (7.45) and; increases the blood bicarbonate (29.8 mEq/L). During peak, milk production respond positively (8.6% higher) to a higher DCAB (+20 vs. -10 mEq/kg) diets. Milk fat percentage and milk protein increases with increase in DCAB:Na+K but cation source and amount of cation has no effect on milk fat yield and milk protein percentage. The DCAB effect on H^{+} concentration in milk is more marked during mid-lactation than in early or late-lactation. During dry period, there is lack of effect of DCAB on DMI but tends to consume and absorb more free water, when offer diet having higher DCAB (+481 vs. +327 mEq/kg). There is evidence that cationic (sodium and potassium) diets induce milk fever but those high in anions (chloride and sulfur) can prevent it. The effects of DCAB on Ca^{+2} metabolism may be mediated by changes in the acid-base status and renal function. Urinary concentration of H^{+} increases and total daily excretion of HCO_3^{-} decreases with the decrease in DCAB by manipulating dietary Cl⁻ and as a consequence glucocorticoids increases.

Key Words: Dietary cation-anion balance; Mid-lactation; Dry period; Milk fever; Milk production

INTRODUCTION

The concept of lowering dietary cation-anion balance (DCAB) to improve the calcium (Ca^{+2}) status of lactating dairy cows became popular during the early 70's (Ender *et al.*, 1971). They showed that acid-base manipulation was a factor in the induction and prevention of milk fever. DCAB manipulation during the dry period to prevent periparturient hypocalcaemia is well documented (Gaynor *et al.*, 1989; Goff *et al.*, 1991), because DCAB affects both blood pH and bicarbonate (HCO_3^{-}) concentration (Fredeen *et al.*, 1988; Tucker *et al.*, 1991a; West *et al.*, 1991; Wang & Beede, 1992b). Prophylactic measures for milk fever are thought to be mediated by modifications of the acid-base status of the cows (Shrestha *et al.*, 1996). Block (1984) used the equation milliequivalents (mEq) of [{sodium (Na^{+}) + potassium (K^{+}) } - {chloride (Cl^{-}) + sulfur (S^{-2}) }] / 100g dietary dry matter (DM) to calculate DCAB and reported that feeding a negative DCAB (anionic) during the dry period eliminated the incidence of milk fever completely compared with 47.7% incidence for cows fed a positive DCAB (cationic) diet. The apparent reason that cows did not become paretic with the anionic diet was the maintained serum Ca^{+2} and phosphorus (P) levels during Ca^{+2} stress. These results were supported by Tucker *et al.* (1988) and Gaynor *et al.* (1989), who reported an increase in plasma and urinary Ca^{+2} when cows were fed diets supplying lower level of DCAB.

Increased plasma hydroxyproline (an index of bone resorption) was reported when cows were fed the anionic diet, suggesting the osteoclastic bone resorption, capable of mobilizing larger amounts of Ca^{+2} enhanced prior to parturition (Block, 1984, Gaynor *et al.*, 1989; Goff *et al.*, 1991). The anionic diet may increase activity of preexisting osteoclasts or stimulate proliferation of new osteoclasts. If more osteoclasts are present prior to parturition, then there will be more osteoclasts available to respond to parathyroid hormone (PTH) as lactation begins, improving the pace of adaptation to the Ca^{+2} demand. Interestingly, plasma hydroxyproline tended to decrease just before parturition in cows fed the cationic diet, aside increasing PTH stimulation at that time. This suggests that osteoclasts of cows that are in metabolic alkalosis are refractory to PTH stimulation at a time when bone Ca^{+2} resorption is critical to Ca^{+2} homeostasis (Goff *et al.*, 1991).

Mid Lactation

A comparison was studied by Staples and Lough (1989) between Alkaten (unrefined sodium sesquicarbonate (NaHCO_3 and Na_2CO_3) having 6% inert materials) and NaHCO_3 during mid-lactation, when corn silage (CS) was the main forage and claimed the effectiveness of Alkaten in elevating per cent milk fat but a higher level of Alkaten was required to increase 4% FCM yield above the NaHCO_3 . No increase in yield of 4% FCM was observed in cows consuming either S-Carb (refined Na-sesquicarbonate) or NaHCO_3 supplemented diets (Muller & Sweeney, 1985).

Sodium bicarbonate improved milk fat test 0.25% units, Rumen Mate (KCl and $\text{MgCO}_3 \cdot \text{Na}_2\text{CO}_3 \cdot \text{NaCl}$) 0.46% units at 1% and 0.69% units at 3% level, when supplemented to CS-wheat based diets (Staples *et al.*, 1986).

a) Dry matter intake. The higher intake was observed in cows consuming the DCAB:Na+K diets than DCAB:magnesium (Mg) diets (Waterman *et al.*, 1991). Thomas *et al.* (1984) using same concentrations of NaHCO_3 and magnesium oxide (MgO) have reported that cows consumed more feed when 1% NaHCO_3 was added than 0.5% MgO. Increased dietary dry matter intake (DMI) was noted with increasing DCAB, suggesting a beneficial effect from feeding cationic diet during mid-lactation (Tucker *et al.*, 1988; Tucker *et al.*, 1991a; West *et al.*, 1991; Delaquis & Block, 1995).

b) Blood acid-base status. A Higher blood hydrogen (H^+) was observed with the lower DCAB diets compared with the higher DCAB diets (Waterman *et al.*, 1991). This difference was attributed to the decrease in blood H^+ as the level of cation increased in the $\text{Na}^+ + \text{K}^+$ diets. This is in agreement with others (Fredeen *et al.*, 1988; Tucker *et al.*, 1991a), who reported higher blood H^+ when diets were supplemented with S^{2-} and Cl^- , thus lowering the DCAB.

The normal range of Blood pH was 7.2-7.5. Higher DCAB produced a higher blood pH (7.45) than the lower DCAB (Thomas *et al.*, 1984; Waterman *et al.*, 1991). This difference was attributed primarily to the high $\text{Na}^+ + \text{K}^+$ diet and corresponds with the H^+ recorded (Waterman *et al.*, 1991). But no significant differences were observed from 10-20 mEq (Teh *et al.*, 1985) or from 0-15mEq (Tucker *et al.*, 1988). Blood partial pressure of carbon dioxide (pCO_2) was unaffected by either source or amount of cation (Waterman *et al.*, 1991; Tucker *et al.*, 1991).

Blood HCO_3^- was higher in cows fed the DCAB:Na+K diets compared with the DCAB:Mg diets. As DCAB increased for both sources of cation, blood HCO_3^- was also increased (Tucker *et al.*, 1988; Waterman *et al.*, 1991). Tucker *et al.* (1991a) also reported lower blood HCO_3^- as the DCAB was reduced by addition of supplemented Cl^- or S^{2-} .

c) Milk and milk components. Milk production responded positively to a higher DCAB in mid-lactation (Tucker *et al.*, 1988, 1991a; Waterman *et al.*, 1991; West *et al.*, 1991; Delaquis & Block, 1995). Tucker *et al.* (1988) noticed 8.6% higher milk yield in cows consuming +20 vs. -10mEq/kg diets. Similarly, in another study, Tucker *et al.* (1991a) found a decrease in actual milk yield with the reduction in DCAB from +15 to 0 mEq/kg diet. Milk fat percentage was increased with increase in DCAB:Na+K when compared to DCAB:Mg (Waterman *et al.* 1991) and was in agreement with the previous work (Erdman *et al.*, 1982; Teh *et al.*, 1985) in which similar levels of NaHCO_3 and MgO showed increased milk fat test. Waterman *et al.* (1991) further noticed that cows receiving the DCAB:Mg diets produced a higher but non-significant milk fat test than DCAB:Na+K diets, whereas cation source and amount of cation had no

significant effect on milk fat yield. However, Delaquis and Block (1995b) noticed that the production of fat was unaffected by DCAB at all stages of lactation.

No effect was found for milk protein percentage for either cation source or level. However, cows produced more milk protein when consuming DCAB:Na+K diets. The difference is assumed to be primarily due to the milk production (Waterman *et al.* 1991). The use of Mg^{+2} to manipulate DCAB:Mg, as the cation source, had no beneficial effects on any of the production variables. Waterman *et al.* (1991) postulated this as a result of lower absorption of Mg^{+2} in the gastrointestinal tract (GIT) than for DCAB:Na+K, thus altering the balance of minerals absorbed from that which had been fed. Na^+ and K^+ are more readily absorbed than Mg^{+2} .

The effect of DCAB on H^+ concentration in milk and total secretion was more marked in mid-lactation than in early or late-lactation (Delaquis & Block, 1995). This difference could be related to a higher secretion of glucocorticoids in mid-lactation that increased the activity of a Na^+ and H^+ exchanger in mammary epithelial cells, as they do in other epithelial cells (Freiberg *et al.*, 1982). The presence of glucocorticoids in the mammary glands has been well documented, but the presence of the ion exchanger and its regulation remain to be studied.

d) Urinary ion balance. No differences due to DCAB occurred for blood pH or HCO_3^- concentration in the mid-lactation (Delaquis & Block, 1995). These results appeared to contradict those of Tucker *et al.* (1991a) and West *et al.* (1991), who demonstrated significant effect of increasing DCAB on blood pH and HCO_3^- concentration. However, as mentioned earlier, in the study of West *et al.* (1991), the range of DCAB used was lower than that of Delaquis and Block (1995a). The cows on the lowest DCAB had lower blood pH than cows consuming higher DCAB (Delaquis & Block, 1995b). As level of cation increased, urine H^+ decreased (Waterman *et al.*, 1991). Tucker *et al.* (1988) and Fredeen *et al.* (1988) also reported lower urine H^+ when Cl^- was removed from the diet, with increased DCAB. Urine was more acidic for the diets having DCAB:Mg compared with DCAB:Na+K. The higher urine H^+ for the Mg^{+2} diets may be explained if the DCAB was calculated using the equation DCAB:S or DCAB:Na. These equations would lower the DCAB:Mg for the diets and would explain the higher urine H^+ , which is in agreement with Tucker *et al.* (1991a). The urine H^+ declined more dramatically for DCAB:Mg diets, when the cation-anion balance (CAB) was increased. This may be due to the alkalinizing capabilities of MgO. The kidney increases alkali excretion in order to maintain normal blood pH. This also may explain the increase in HCO_3^- with the higher DCAB:Mg. As the amount of cation increased, urine pH increased for both the DCAB:Na+K and DCAB:Mg diets. Similarly, Tucker *et al.* (1988) also reported that urine pH increased linearly with increasing DCAB when Na^+ , K^+ and Cl^- were ionized to calculate the DCAB.

Dry Period

With the increase in gestation, the bovine fetal placental mass and its demand for energy, protein and minerals increase dramatically. Especially during the last trimester of gestation, daily development of fetus requires about 0.82 Mcal of energy, 117 g of protein, 10.3 g of Ca^{+2} , 5.4 g of P and 0.2 g of Mg^{+2} (Bell *et al.*, 1995).

a) Dry matter intake. The lack of effect on DMI of dry cows with DCAB (-75 to +190 & -228 to 978 mEq/Kg) has been observed by Oetzel *et al.* (1988) and Goff *et al.* (1991), respectively. The study of Delaquis and Block (1995a) also supported the previous results for dry cows (Leclerc & Block, 1989) and sheep (Takagi & Block, 1991abc) in which DCAB from -27 to +343 mEq/Kg had no effect on DMI. Intakes of Na^+ were similar between diets because of similar dietary DMI and mineral concentrations in the diets (Delaquis & Block, 1995). There was no effect of varying DCAB for pregnant does on total concentration and molar percentages of ruminal VFA (Fredeen *et al.*, 1988).

b) Water metabolism. Cows tended to consume and absorb more free water with high DCAB (+481 mEq/kg) than low (+327 mEq/kg) (Delaquis & Block, 1995). Reports by Escobosa *et al.* (1984) for lactating cows and Fredeen *et al.* (1988) for pregnant does also showed increased water consumption with an increase in DCAB. It could have been related to both the increases in Na^+ intake and blood Na^+ concentration as the DCAB of the ration was increased by manipulating the dietary concentration of Na^+ . In blood, Na^+ plays a role in the control of thirst (Delaquis & Block, 1995). Increased water consumption with higher DCAB cannot be attributed to the intake of Na^+ , but higher K^+ intake may have played a role, causing a favourable osmotic gradient for water absorption in the GIT. The increased water consumption could also have resulted from the increased excretion of HCO_3^- creating again a favourable gradient for water excretion in the urine of cows consuming a ration having DCAB value of +481 mEq/kg. Interestingly, Fredeen *et al.* (1988) observed reduced water intake and urine volume due to increase in DCAB by reduction of Cl^- and S^{2-} and argued it as the changes, caused by DCAB, not by a specific mineral. This can be attributed to the presence of anion antiport in renal epithelial cells that can transport Cl^- or S^- in exchange for HCO_3^- . Tucker *et al.* (1992) linked DCAB to udder edema during the periparturient period and was supported by the observation that water metabolism was affected by DCAB.

Changes in blood or extracellular fluid volumes were other regulators of water balance and vasopressin secretion (Robertson & Berl, 1991) and may have mediated the effects of DCAB on urine volume. If DCAB stimulated thirst directly, the increased water consumption could increase blood volume, reducing vasopressin secretion and renal water reabsorption. Consequently, urine volume would increase. The hormonal regulation of fluid volumes and osmolarities may also be modulated or affected by

perturbation of the acid-base equilibrium.

c) Metabolism. Dietary CAB of the prepartum diets can also influence the incidence of milk fever (Oetzel *et al.*, 1988; Gaynor *et al.*, 1989). Diets high in cation especially Na^+ and K^+ tended to induce milk fever but those high in anions primarily Cl^- and S^{2-} can prevent milk fever. Addition of anions to the prepartal diet is thought to induce a metabolic acidosis in the cow, which facilitate bone Ca^{+2} resorption (Block, 1984) and intestinal Ca^{+2} absorption (Ferdeen *et al.*, 1988b). Latter on Tucker *et al.* (1991a) demonstrated that the effect of S^{2-} on systematic acid-base status in lactating dairy cows is similar to the effect of Cl^- . In the GIT, a large fraction of S^{2-} is absorbed by Co-transport with Na^+ (Conway *et al.*, 1989). The high amount of S^{2-} absorbed with diet having DCAB value +327 vs. +481 mEq/kg could have limited the amount of Na^+ available for Cl^- absorption; Cl^- is also absorbed in a Na^+ dependent manner in some areas of the intestine (Powell, 1987). Because intestinal absorption of Cl^- did not differ between the 2 diets, some other positively charged element must have been absorbed, or some negatively charged element must have been excreted, to maintain electrical neutrality and Cl^- absorption. K^+ is not a likely candidate because its GIT absorption or urinary excretion did not differ between diets (Delaquis & Block, 1995). However, an increased activity of the $\text{Cl}^-/\text{HCO}_3^-$ or $\text{SO}_4^{2-}/\text{HCO}_3^-$ exchangers present in the intestinal and renal epithelium could have played a role in the maintenance of Cl^- absorption and increase in S^{2-} absorption (Powell, 1987).

The acid stress caused by dietary manipulation was evident from urine analysis and was insufficient to overload the renal compensatory mechanisms, which would explain why the acid-base parameters in blood were not different post-feeding. Urinary concentration of H^+ increased, and total daily excretion of HCO_3^- decreased as DCAB was decreased by manipulating dietary Cl^- not S^{2-} in pregnant goats (Ferdeen *et al.*, 1988a; Delaquis & Block, 1995a).

The heavier acid load caused by elevated dietary S^{2-} may have increased the glucocorticoids as normally occurs in acidosis (England *et al.*, 1991). These hormones inhibit S^{2-} reabsorption by the kidneys (Renfro *et al.*, 1989), which would explain the increased urinary concentration of S when DCAB having +327 mEq/kg was fed. However, whether plasma S^{2-} alone can affect the $\text{Na}^+/\text{SO}_4^{2-}$ transport system or the $\text{HCO}_3^-/\text{SO}_4^{2-}$ exchanger (Renfro *et al.*, 1989) in the renal tubules is not known.

Milk fever (parturient paresis) is a hypocalcemic disorder of dairy cows associated with the onset of lactation. On the day of parturition, dairy cows commonly produce 10 litre or more of colostrum, >23 g of Ca^{+2} , approximately 6X as much Ca^{+2} as the extracellular Ca^{+2} pool contains (Goff *et al.*, 1987). Most cows adapt to this Ca^{+2} stress by rapidly increasing intestinal Ca^{+2} absorption and bone Ca^{+2} resorption, regulated by PTH and 1,25 dihydroxyvitamin D_3 [$1,25\text{-(OH)}_2\text{D}_3$] (Reinhardt *et al.*, 1988). However, in some cows, the Ca^{+2} homeostatic

mechanisms of the body fail to replace adequately the Ca⁺² lost from the extracellular pool. These animals become severely hypocalcemic, resulting in recumbency and paresis, which is clinically referred as “milk fever”. Manipulation of DCAB for dry cows can be as effective as vitamin D therapy before calving and in most cases, more practical than a low Ca⁺² diet fed prepartum (Gaynor *et al.*, 1989).

The effects of DCAB on Ca⁺² metabolism may be mediated by changes in the acid-base status and renal function. Oetzel *et al.* (1988) reported that feeding a low DCAB prepartum increased both ionized and total Ca⁺² in serum, although this response was most evident during the peri-parturient period. However, Gaynor *et al.* (1989) did not found increase plasma Ca⁺² prepartum with lowest DCAB (+26.5). Plasma Ca⁺² was higher for low DCAB (-3.41) than for high DCAB (+9.35) at parturition only. Similarly plasma Ca⁺² was consistently higher for heifers than for mature cows (Tucker *et al.*, 1992). Feeding a low DCAB increases the flow of Ca⁺² through the readily exchangeable Ca⁺² pool (Takagi & Block, 1988) and

increases the concentration of ionized calcium (iCa) in the blood (Oetzel *et al.*, 1988). Each of these responses would increase the availability of Ca⁺² for metabolic functions. In addition, this increases the amount of Ca⁺² presented to the kidneys for filtration, so urinary Ca⁺² excretion increases. Tucker *et al.* (1992) found that urinary Ca⁺² concentration increased with increases in plasma Ca⁺², blood H⁺, and urine H⁺ and argued that increases in urinary Ca⁺² excretion typically represent increased availability of Ca⁺² for metabolic functions and may be useful to evaluate Ca⁺² status. Fredeen *et al.* (1988) and Wang and Beede (1992b) demonstrated that reduced DCAB caused a reduction in HCO₃⁻ concentration in blood/urine, a higher excretion of Ca⁺² and a lower excretion of Mg⁺² in dry cows and goats. Cows offered DCAB (+481 mEq/kg) may have had a greater true absorption of Na⁺, creating an increased gradient for water absorption from the gut. The duodenum is a leaky epithelium and absorbs fluid isotonically (Powell, 1987). This increased fluid volume would, increase atrial natriuretic peptide secretion. which responded to volume

Table I. List of the DCAB's used commonly with their formulae, elements manipulated, forage to concentrate ratio, and physiological stage

Treatment	Forage: Concentrate	Formula used mEq/kg of DM	DCAB	Elements Manipulated	Stage	Days ± parturition	Author
Cation	39%CA:57%CS	(Na+K)-(Cl+S)	+33.05		DP	+45	Block (1984)
Anion			-12.85				
0% CaCl ₂	64%CS:36%C	Na+K-Cl	20.2	CaCl ₂	DP		Tucker <i>et al.</i> (1991b)
0.5% CaCl ₂			14.9				
1.0% CaCl ₂			8.1				
1.5% CaCl ₂			-11.7				
Cation (L.CaLP)	TMR	Na+K-Cl	23.0		DP	-120	Romo <i>et al.</i> (1991)
(HcaLP)			22.0				
(LcaHP)			23.0				
(HcaHP)			22.0				
Anion (L.CaLP)			-8.0				
(HcaLP)			-8.0				
(LcaHP)			-7.0				
(HcaHP)			-7.0				
Anionic	AH	(Na+K)-(Cl+S)	-228.0	Cl	DP	-42	Goff <i>et al.</i> (1991)
Cationic			+978.0	Na & Cl			
Hi-Bal (+limestone)	10%SS:56AH:34C	(Na+K)-(Cl+S)	+9.35		DP	-21	Tucker <i>et al.</i> (1992a)
Lo-Bal (+CaCl ₂)			-3.41				
Fresh	AH:SS:WC:C		+5.99		EL		
Mg (Normal)	68%CS:32%C	(NA+K)-(Cl+S)	-302.0	(NH ₄) ₂ SO ₄ and MgSO ₄	DP		Wang and Beede (1992a)
Mg (High)			-289.0				
Control	68%CS:32%C	(NA+K)-(Cl+S)	+69.0	(NH ₄) ₂ SO ₄ and NH ₄ Cl	DP		Wang and Beede (1992b)
Treatment			-428.0				
D+481	AH based	(Na+K)-(Cl+S)	+481.8	K ₂ CO ₃	DP	-144 to -250	Delaquis and Block (1995a)
D+273			+327.2	MgSO ₄			
AH	AH based	(Na+K)-(Cl+S)	+440.2		L	+14	Delaquis and Block (1995c)
CS	CS based		+412.0				
Na+K (Low)	35%CS:65%C	Na+K-Cl	+13.61		ML		Waterman <i>et al.</i> (1991)
Na+K (High)			+26.28				
Mg (Low)		(Na+K+Mg)-Cl	+34.69				
Mg (High)			+45.95				
EA	AH based (51%)	(Na+K)-(Cl+S)	+258.1		EL	+25 to +50	Delaquis and Block (1995b)
EB			+55.5				
MA	AH based (49%)		+372.7		ML	+107 to +137	
MB			+140.2				
LA	AH based (56%)		+374.6		DP	+162 to +234	
LB			+199.8				

NOTE: C= concentrate; CS= corn silage; SS= sorghum silage; AH= alfalfa haylage; WC= whole cottonseed; Lo-Bic= low bicarbonate; Hi-Bic= high bicarbonate; Med-Bic= Medium bicarbonate; Ctrl= control; LCALP= low calcium and low phosphorus; HCaLP= High calcium and low phosphorus; LCaHP= Low calcium and high phosphorus; HCaHP= high calcium and high phosphorus; EA= early lactation with high DCAB; EB= early lactation with low DCAB; MA= mid-lactation with high DCAB; MB= mid-lactation with low DCAB; LA= late-lactation with high DCAB; LB= late-lactation with low DCAB; EL= Early lactation; ML= mid-lactation; L= lactation; DP= dry period

expansion in goats (Olsson *et al.*, 1991). The increased secretion of atrial natriuretic peptides would then increase natriuresis and diuresis, as observed by Delaquis and Block (1995a). The trend toward higher glomerular filtration rate was observed as DCAB was increased and could explain some of the increase in urine volume. Urine volume was increased by higher DCAB (Fredeen *et al.*, 1988; Delaquis & Block, 1995). Interestingly, Fredeen *et al.* (1988) modified the DCAB by addition of Cl⁻ to reduce the balance, instead of S²⁻, therefore the effects on water consumption and urine volume appear to be caused by altered DCAB rather than by individual minerals in goats.

d) Urinary ion balance. The effect of DCAB on urinary Ca⁺² (Delaquis & Block, 1995) is little, in contrast to results of previous studies (Fredeen *et al.*, 1988; Gaynor *et al.*, 1989; Takagi & Block, 1991b) and probably because of the smaller difference (154mEq/kg) in DCAB (Delaquis & Block, 1995) vs the greater difference (379 mEq/kg) altering Ca⁺² excretion in the study by Gaynor *et al.* (1989). The same reasoning can be applied to the lack of effect of DCAB on urinary Mg⁺² excretion in the study of Delaquis and Block (1995a) compared with the results of Gaynor *et al.* (1989), in which urinary excretion of Mg⁺² increased as DCAB was increased from 220-500mEq/kg diet. Concentration and total excretion of HCO₃⁻ via the urine were reduced by a reduction in DCAB which indicated a definite effect of diet on acid-base status. Cows fed DCAB (+327 mEq/kg) excreted 43% less HCO₃⁻ daily and had urinary concentration 34% lower than cows fed DCAB (+481 mEq/kg), demonstrating the greater acid load caused by DCAB (+327 mEq/kg). Modulation of renal reabsorption of HCO₃⁻ is a classical renal compensatory response to perturbation of acid-base balance (Delaquis & Block, 1995). The kidneys seem to respond to a decline in blood pH, which stimulates the Na⁺/H⁺ counter transport system in the renal tubules (Sabatini & Kurtzman, 1989). However, Delaquis and Block (1995a) could not establish whether Na⁺/H⁺ transport system was affected alone or whether a modification of the activity of the H⁺ ATPase (Alpern *et al.*, 1991) also contributed to the renal response to a modified acid-base status. Excretion of free H⁺ also increased in cows offered diets supplying DCAB (+327 mEq/kg).

In summary, the research done on the DCAB, along with the values used, ions to be manipulated, as well as ratio of forage to concentrate, along with the physiological state of the animal is shown in table I.

CONCLUSIONS

During mid-lactation DMI, milk production and milk fat percentage increases with increasing DCAB, i.e. by cationic diets but milk protein remains unchanged for either cation source or level. Anionic diets increases blood H⁺ and decreases blood HCO₃⁻, as opposed to the cationic diets. Increasing DCAB reduced the urine H⁺ loss, i.e. the normal renal compensatory mechanism to check the acid stress. In

dry cows and sheep, no effects of DCAB has been seen on DMI. Cows having high DCAB consume more water during dry period due to increase in Na intake. High DCAB increases urinary Ca excretion, whereas low DCAB decrease HCO₃⁻ loss via urine indicating greater acid load.

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