INFLUENCE OF VARYING LEVELS OF DIETARY CATION ANION DIFFERENCE ON RUMINAL CHARACTERISTICS, ACID BASE STATUS AND MILK YIELD OF EARLY LACTATING ANIMALS (A Review)

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Productive performance of the animal is directly related with feed intake. Among various nutritional tools used to improve feed intake, dietary cation anion difference (DCAD) has the most promising effect. The DCAD is the difference between cations and anions and ($Na^+ + K^+$) - ($Cl^- + S^{--}$) mEq/100 gm of DM is the most common equation used for DCAD calculation. High DCAD diet improves the pH of the rumen, required for optimum cellulytic microbial activity which increases dry matter intake. The rumen pH is a primary factor for controlling fiber digestion. There is a close relationship between rumen pH and forage digestion kinetics. Extent of cellulose digestion by microorganisms also depends upon ruminal pH and any change in pH from 6.8 to 6.5 increases lag time by 2h and on reducing below 6.0, lag time increased up to 8h due to loose attachment of bacteria to plant cell wall at low pH. The response of the ruminal H^+ to high DCAD is very quick. It improves ruminal NH_3-N concentration, acetate while propionate and butyrate decrease which results in higher acetate to propionate ratio. Blood pH, HCO_3^-, serum cation anion balance ($Na^+ + K^+ - Cl^- + S^{--}$) also tended to increase with increasing DCAD. It increases urine pH which reflects alteration in blood pH. Milk yield and its composition are also improved as a result of increased DCAD level in the diet.
Keywords: DCAD, feed intake, rumen pH, digestion kinetics, milk yield

INTRODUCTION

Significance of balance nutrition for productive and reproductive performance of dairy animals can't be denied. Provision of balance nutrition not only increases milk production but it also minimizes certain problems associated with reproduction (Osmanu, 1979; Bellows and Short, 1978). High yielding animals during early lactation usually experience negative energy balance due to decreased dry matter intake (DMI), which adversely affects animal productivity (Butler *et al.*, 1981; Stevenson and Britt, 1980).

There are various means and ways to improve the DMI to ensure sufficient nutrient supply to meet productive and reproductive needs of animal body. Dietary minerals are very important for biological functions of animal body. The difference between certain cation and anion imparts stronger effects on animal physiology than their individual effects. Among minerals, cations when excess of anions impart alkalogenic effect while an acidogenic effect is imparted by anions when in excess of cations. The difference between certain cations (Na^+ and K^+) and anions (Cl^- and S^{--}), in milliequivalents in the whole diet, is usually referred as dietary cation anion difference (DCAD).

The concept of DCAD is based upon the maintenance of desirable acid base status, placed third in homeostatic priorities after the need of oxygen and heat dissipation (Kronfeld, 1979). Dishington (1975) and Mongin (1981) were the first who used the concept of DCAD in livestock and poultry, respectively. Since then it has been used in prepartum and postpartum animals due to its beneficial effect. The strong cations and anions, which are used to calculate DCAD, determine the body fluid pH (Stewart, 1981).

Following equation is most commonly used for calculation of DCAD

($Na^+ + K^+$) - ($Cl^- + S^{--}$) = mEq/100 gm of DM

(Tucker *et al.,* 1992)

Physiologically, the DCAD affects acid base status and Ca metabolism of dairy cows (Sanchez, 2003). It influences the animal performance by affecting blood pH and HCO_3^-. e.g. high DCAD diet decreases blood H^+ concentration and increases blood HCO_3^-, blood and urine pH that indicate the improved blood buffering capacity (Roche *et al.*, 2005; West *et al.*, 1991). Low DCAD increases blood H^+ concentration that results in slight acidosis and Ca absorption increases (Fredeen *et al.*, 1988; Moore *et al.*, 2000). This paper reviews effect of DCAD on various parameters as under:

Nutrient Intake

The DCAD has favourable effect on DMI. Increasing DCAD in the diet of prepartum cows results in increased DMI (Moore *et al.*, 2000). They reported that DMI increased from 13 to 14.5 kg daily with increasing DCAD level in the diet. Wang and Beede (1992) also observed increased DMI as DCAD increased from -42 to +69 mEq/100g of DM in dry cows. They further stated that increased feed consumption in lactating cows with high DCAD diets might be due to positive effect of higher DCAD on rumen pH as low rumen H^+ concentration was reported in cows fed high DCAD diet (Tucker *et al.*, 1988). A high DCAD diet tends to make the rumen pH towards alkaline, required by variety of ruminal cellulolytic microbes for their optimum activity.

Cows receiving -7 mEq/100g DM DCAD had lower DMI compared to those fed +35 meq/100g DM DCAD prior to parturition (Joyce *et al.*, 1997). Anionic salts reduced DMI in prepartum cows (Tucker *et al.*, 1992). Similarly, Oetzel and Barmore (1993) observed increased DMI in cows when DCAD level was increased from –109 to +313 mEq/kg DM. A meta-analysis conducted by Hu and Murphy (2004) also reported that a linear relationship exists between DCAD and DMI. Their analysis revealed maximum feed intake at 40 mEq/100g DM DCAD. They used two DCAD levels (+69 and -42 mEq/100g DM) and recorded increased feed consumption in cows fed high DCAD compared to those fed low or negative DCAD level. The probable reason for decreased DMI in cows fed negative DCAD diet might be higher level of ammonium chloride and ammonium sulfate. These salts, being anionic in nature, were used to attain the desired reduced level of DCAD. The inclusion of these salts, being unpalatable, might have reduced the palatability of the diet. The reduced feed consumption due to poor palatability of diets with low or negative DCAD has also been reported by Goff *et al.* (1991). However, in contrast to present findings, Oetzel *et al.* (1988; 1991) reported that anionic salts supplementation had no effect on DMI. Similar results were also reported by Block (1984) in dry cows. The probable reason might be that anionic salts were delivered in total mixed ration in both trials. Anionic salts delivered in total mixed ration at doses 3.4 mEq/cow per day were palatable and nontoxic but these salts may be unpalatable and toxic when delivered in a grain mix.

Delaquis and Block (1995a) observed that DCAD (481, 327meq/kg of DM) had no effect on neutral detergent fiber (NDF) and acid detergent fiber (ADF) digestibilities in dry cows. In another study, Delaquis and Block (1995b) reported that during lactation period, dry matter digestibility increased in cows fed +55 compared to +375 mEq/kg DCAD diets, respectively. However, the difference remained non-significant. The NDF digestibility also remained unaltered by DCAD alteration. This might be attributed to the speculation that DCAD had no effect on ruminal fermentation pattern in lactating cows (Tucker *et al.*, 1991). Canale and Stokes (1988) reported the influence of $NaHCO_3$ supplementation on forage source and nutrient digestibility. It was observed that apparent dry matter digestibility of the corn silage based diet was greater than hay crop silage. The $NaHCO_3$ supplementation improved the digestibility of NDF in both diets but did not significantly affect the digestion of other nutrients. In rumen $NaHCO_3$ is bifurcated into Na^+ and HCO_3^- and they impart non-buffering and buffering effects, respectively (Schneider *et al.*, 1986).

Digestion Kinetics

The DCAD affects the digestion kinetics by affecting the pH of the rumen. Increasing DCAD level in the diet improves rumen pH, which enhances cellulytic microbial activity because it is directly related with rumen pH. The rumen pH is a principal factor for controlling fiber digestion. Grant and Weidner (1992) reported a close relationship between rumen pH and forage digestion kinetics. Slyter *et al.* (1970) pointed out that fiber digestion reduced at low ruminal pH, which might be attributed to decreased number of cellulolytic microbes. Extent of cellulose digestion by microorganisms was dependent on pH (Terry *et al.*, 1969). Low ruminal pH also hampered bacterial attachment to plant substrate (Cheng, *et al.*, 1984). Mould *et al.* (1984) also reported a moderate depression in fiber digestion when ruminal pH dropped from 6.8 to 6.0 and fiber digestion was severely reduced when ruminal pH was below 6. Groleau and Forsberg (1983) indicated that fibrolytic enzymes function efficiently at 6 to 6.8 pH. Grant and Weidner (1992) reported that decreasing pH below 6.2 depressed rate of NDF digestion. They further stated an inverse relationship between pH and lag time. Decreasing pH from 6.8 to 6.5 increased lag time by 2h and decreasing pH below 6.0 increased the lag time dramatically up to 8h. This might be associated with loose attachment of bacteria to plant cell wall at low pH (Cheng *et al.*, 1984).

Ruminal Characteristics

Infusion of $NaHCO_3$ exerts its effect on ruminal H^+ quickly (0.5 h of infusion). Hough *et al.* (1991) reported reduction in H^+ concentration in the rumen at 0-6 h infusion of $NaHCO_3$ and increased 7-8 h post feeding compared to cows fed control diet. Increase in rumen H^+ after 7-8 h post feeding might be due to feedback mechanism, which reduces salivary buffer flow in response to increased ruminal fluid pH in cows given $NaHCO_3$ infusion (Hough *et al.*, 1991). Ruminal fluid H^+ of control cows increased until 4-6 h post-feeding, which may be due to increased concentration of fermentation acids and after 6 h, it dropped rapidly. Thus supplementation of buffer decreased ruminal fluid acidity.

A positive linear relationship exists between DCAD and ruminal pH (Tucker *et al.* 1988). Ruminal pH tended to increase with increasing DCAD. Fredeen *et al.* (1988) pointed out that ruminal H^+ concentration was higher at 0.7 mEq/100 g DM DCAD and lower at 45.8 or 90 mEq/100 g DM DCAD in lactating or pregnant goats. Similarly, Ross *et al.* (1994) observed that ruminal pH tended to increase with increasing dietary electrolyte balance (DEB). Minimum (6.45) and maximum (6.76) pH was observed at –10 and +20 mEq/100 g DM DCAD diets, respectively. Ross *et al.* (1994) observed that ruminal pH tended to increase with increasing dietary electrolyte balance (DEB). Decreased ruminal pH at low DCAD diet might be due to acidogenic properties of the diet.

Ruminal NH_3-N concentration increased with the addition of buffer and was highest at 0.7% $NaHCO_3$ and 0.28% MgO (Stokes *et al.*, 1986). They further stated that the close relationship did not exist between ruminal NH_3-N and urinary N losses. Kennelly *et al.* (1999) pointed out that rumen NH_3-N concentration increased with buffer supplementation while concentrate feeding depressed it. They observed an interaction between dietary buffers and ruminal NH_3-N concentration. Other workers reported that ruminal ammonia concentration increased (Kilmer *et al.*, 1981), decreased (Stokes *et al.*, 1986) or remained unchanged (Kilmer *et al.*, 1980) by buffer supplementation.

Sodium sesquicarbonate supplementation increased acetate while propionate and butyrate decreased which resulted in higher acetate to propionate (A:P) ratio (Moore *et al.*, 1992). Addition of buffer increased rumen acetate, butyrate, isobutyrate, valerate, isovalerate, branched chain fatty acid, and A:P ratio while propionate concentration decreased (Kennelly *et al.*, 1999). Moreover, A to P ratio increased in cows fed concentrate but dramatic effect was observed by buffer supplementation (Kennelly *et al.*, 1999). Tucker *et al.* (1993) also reported an increase in acetate and propionate concentration with $NaHCO_3$ infusion while A to P ratio decreased at 11h post feeding. However, Stokes *et al.* (1986) pointed out that dietary buffer supplementation in cows resulted in decreased VFA concentration. This may be due to diet dilution by addition of minerals. It was recorded that A:P ratio was the lowest at 0.7% $NaHCO_3$ and decreased by other buffers while its reverse was true for propionate to butyrate ratio. Valerate decreased by buffer supplementation (Stokes and Bull, 1986). This revealed that fermentation was shifted from propionate and valerate towards acetate and butyrate (Kaufmann *et al.*, 1980; Klover and DeVeth, 2002). This may lead to more precursors for milk fat synthesis (Orskov, 1975). It was observed for buffer supplementation in corn silage based diets (Erdman *et al.*, 1982; Snyder *et al.*, 1983; Rogers *et al.*, 1982) but not with alfalfa hay diets (DePeters *et al.*, 1984; Rogers *et al.*, 1985). The probable reason might be its effect on ruminal microbial population and ruminal kinetics.

Blood Acid Base Status

Blood pH and HCO_3^- tended to increase as DCAD level was increased in early lactating cows (Roche *et al.*, 2005). Increased blood pH and HCO_3^- concentration was observed in cows fed 88 mEq/100 g DM DCAD compared to those fed 23 mEq/100 g DM DCAD. Similar findings were also observed by Jackson *et al.* (1992) in growing calves. They reported that blood pH and HCO_3^- were lower in calves fed 0 DCAD diet than those fed 21, 37 and 52 mEq/100 g of DM DCAD diets. They pointed out that a positive linear relationship existed between blood acid base status and DCAD. The main reason for reduced blood pH and HCO_3^- at low DCAD diets is due to its acidic properties. Calcium chloride is primarily used as an anionic salt to reduce the DCAD level which has more absorption and acidifying properties compared to all other anionic salts (Roche *et al.*, 2003). Chloride is absorbed from the posterior part of intestine in exchange of Na^+ and when it is in excess of Na^+ then with HCO_3^- resulting in reduced blood bicarbonate and increased H^+. It might have overcome the capacity of kidneys to excrete H^+ to maintain a constant blood pH, following slight metabolic acidosis. Moreover, when the body faces an acid load, it is compensated through respiratory rate that reduced pCO_2 and H_2CO_3 (Hill, 1990). High DCAD diets decreased blood H^+ and thus resulted in increased blood HCO^-_3 (Block, 1994). Other researchers (Roche *et al.*, 2003; Tucker *et al.*, 1988) also observed the effect of DCAD on blood acid base status. They reported that blood H^+ was greater at low DCAD than that of high DCAD diet during prepartum and postpartum. Blood pH tended to increase with increasing (98, 186 and 270 mEq/kg DM) DCAD level (Manna *et al.*, 1999). Blood HCO_3^- responses were inversely related to H^+ changes that reflected the metabolic nature of the acid challenge at low DCAD. These findings were consistent with those of Fredeen *et al.* (1988) who observed the effect of diets having low and high DCAD level (-38.3 vs + 33.1) in pregnant and lactating goats. They reported that high DCAD diet increased blood HCO_3^- and lowered blood H^+ concentrations compared with low DCAD diets.

Blood pH and HCO_3^- increased quadratically with increasing DCAD levels (-116.6 to 312.4 mEq/kg DM) and was lowest in cows receiving low DCAD diet (West *et al.*, 1991). This might be attributed to the fact that high DCAD diet resulted in increased pH due to more HCO_3^- production and H^+ excretion (Tucker *et al.,* 1992). Huber (1976) reported that rumen pH declined rapidly with a decrease in blood HCO_3^- and pH in calves offered high grain diet at once instead of alfalfa and increased after an adaptation period with the resumption in feed intake. Increasing level of DCAD in diets restored blood pH, HCO_3^- and feed intake to normal level. Blood HCO_3^- and pH returned to normal as Cl in the diet declined (West *et al.,* 1991). West *et al.* (1992) also observed the effect of increasing DCAD from 120.4 to 464.1 mEq/kg DM and reported that blood pH increased linearly while HCO_3^- and total CO_2 increased cubically with increasing DCAD. Blood pH and HCO_3^- contents declined in cows receiving low DCAD diet. The decreased blood HCO_3^- content might be due to high serum Cl^- content because both vary inversely to keep total anion concentration constant (Ganong, 1983); while the diets containing high DCAD levels contained increased Na^+, K^+ and HCO_3^- and were more alkaline. A cubic effect on blood base was observed with increasing DCAD, possibly due to dietary HCO_3.

Blood Minerals

The DCAD diet did not affect serum Na^+ or K^+ concentration but Cl^- concentration decreased with increased DCAD (West *et al.*, 1991; Tucker *et al.*, 1988). West *et al.* (1992) fed lactating cows diets containing varying DCAD levels (120.4 to 464.1 mEq/kg DM) and reported that serum Na^+ and K^+ were not altered by varying DCAD levels. Hu and Murphy (2004) reported that blood Na^+ and K^+ concentrations were not affected by DCAD and this may be due to their excess excretion through kidney. However, Fredeen *et al.* (1988) noticed that plasma Na^+ concentration increased as DCAD was increased from 45.8 to 90 mEq/100 g DM DCAD. Plasma K^+ concentration decreased by anionic diet in lactating goats while Cl^- concentration increased in both pregnant and lactating goats (Fredeen *et al.*, 1988). The possible explanation of this might be that high dietary Cl^- concentration decreased plasma K^+ concentration. Increased serum Cl^- concentration was observed in cows fed diet containing -79.4mEq/kg DCAD compared to those fed diets containing 47.2, 166.6 and 324.4 mEq/kg DM DCAD (West *et al.*, 1991). Increased plasma Cl^- concentration with decreasing DCAD reflected Cl^- content of the diet (Tucker *et al.*, 1991). Ganong (1983) observed that absorption of Cl^- took place in ileum and colon in exchange for HCO_3^- and change in DCAD affected the acid base chemistry of the animal. West *et al.* (1991) reported a linear increase in serum cation anion balance ($Na^+ + K^+ - Cl^-$) with increasing DCAD levels. The elevated serum $Na^+ + K^+ - Cl^-$ is closely associated with change in blood pH, which increases with increasing DCAD and thus greater blood buffering at higher DCAD. Serum ($Na^+ + K^+ - Cl^-$) increased linearly with increasing DCAD because it was closely related to increasing blood pH (Tucker *et al.*, 1988; West *et al.*, 1991) and enhanced blood HCO_3^- (Tucker *et al.*, 1988) in lactating dairy cows.

West *et al.* (1992) observed that DCAD had non-significant effect on blood serum Mg and P concentrations. Plasma Mg concentration remained unaltered by DCAD alteration in lactating cows (Tucker *et al.*, 1988). Feeding low DCAD prepartum enhanced plasma Mg (Oetzel *et al.*, 1988). Similar results were also reported by Gaynor *et al* (1989). However, a linear decrease in plasma Mg concentration was observed with increasing DCAD (Roche *et al.*, 2005; Fredeen *et al.*, 1988). Jackson *et al.* (1992) reported that with increasing DCAD from 0 to 52 mEq/100 g of DM, plasma Mg decreased linearly. Tucker *et al.* (1988) elucidated that DCAD had non-significant effect on plasma P in lactating cows (Oetzel *et al.*, 1988). Similar findings were observed by Gaynor *et al.* (1989) and Oetzel *et al* (1988) who reported that plasma P remained unaltered with varying DCAD level. However, in contrast to these findings, Roche *et al.* (2005) reported that plasma P concentration first increased with increasing DCAD level from 23 to 45 mEq/100 g DM and beyond that level, it decreased. The probable explanation of this might be that there is a threshold DCAD limit and above this level, there is little or non-significant effect of DCAD on P concentration.

Delaquis and Block (1995a) observed the effect of varying DCAD (481.8 to 327.2 mEq/kg) on plasma minerals in dry cows. They pointed out that DCAD had no effect on Mg^{++}, Na^+, K^+, or Cl^- concentrations in plasma at any sampling time. At 2-4 h post feeding, cows offered 327.2 DCAD had higher S^{--} concentration in plasma. Krijgsheld *et al.* (1979) reported that high dietary intakes of S^{--} affected its blood concentration in rats. Delaquis and Block (1995b) reported that concentration of macro minerals (except S^{--}) in plasma were non-significantly affected by DCAD. Plasma S^{--} concentration elevated at low DCAD during early, mid and late lactations. This is because S^{--} balance is regulated renally, not intestinally and increased intake increased absorption and concentration in plasma that resulted in increased urinary excretion.

Urine pH

A quadratic increase in urinary pH was observed as DCAD level was increased from 120.4 to 464.1 mEq/kg DM (West *et al.*, 1992). Alteration in urine pH reflected alteration in blood pH and kidneys played a vital role to minimize this change by making the urine pH alkaline, by excreting more H^+ and conserving more HCO_3^- (Roche *et al.*, 2003). A dramatic reduction in urine pH had also been reported with increased anion (Cl^- or S^{--}) or decreased cation (Na^+ or K^+) of the diet (Waterman *et al.*, 1991). It is well documented (Jackson and Hemken, 1994; Jackson *et al.*, 1992) that increased dietary anions decreased the urine pH. However, urine pH may decline as low as 4.5, due to low or negative DCAD level. The urine pH is generally used as an indicator of metabolic acid or alkali load (Sanchez, 2003).

Fredeen *et al.* (1988) investigated the effect of diets with varying DCAD from -38.3, –2.8 and 33.1 mEq/100 g DM on pregnant and lactating goats. They reported that urine pH increased with increasing DCAD level in the diet. Urinary HCO_3^- excretion increased by high DCAD diet and it decreased by low DCAD diet. Slightly more HCO_3^- was excreted in urine of lactating goats compared to pregnant ones. Similar effect was observed by West *et al.,* (1991) who reported quadratic increase in urine pH of the animals fed diets containing increasing DCAD levels (-79.4 to 324.4 mEq/kg). Increased urinary pH might be due to increased blood HCO_3^- and decreased serum Cl^- at high DCAD level. There was a linear increase in urinary DCAD ($Na^+ + K^+ - Cl^-$) with increasing DCAD. Urinary K^+ increased and Cl^- reduced quadratically with increasing DCAD. Excretion of each HCO_3^- carries Na^+ or other cations for renal rectification of alkalosis in order to ensure electrical neutrality of the body (Guyton, 2000).

Increase in urine pH was also observed in cows fed diet containing $NaHCO_3$ (1%) compared to those fed control diet (Ghorbani *et al.*, 1989). There was sharp decline in urine pH at 2 h post feeding and increased with time in buffered diets. Increased urinary pH was also observed with supplementation of sodium sesquicarbonate (Caddisa *et al.*, 1988) and $NaHCO_3$ (Kilmer *et al.*, 1981; Rogers *et al.*, 1982) in the diet. Under alkaline conditions, the kidney increases alkali excretion and suppresses H^+ excretion in order to maintain normal blood pH so urine pH increases (Cohen and Kassirer, 1982). Increased urine pH indicates that buffers may be useful to alleviate systemic acidosis.

Milk Yield and Composition

Milk production increased linearly with increasing DCAD level in the diet (Tucker *et al.,* 1988) which might be due to increased DMI. Delaquis and Block (1995b) also reported enhanced milk production in early and mid lactating cows fed diets containing high DCAD. Similar results were reported by Block (1994) who indicated that higher DCAD increased the milk production in lactating cows. Block (1994) further stated that lactating cows had higher metabolic rate that tended to make the cellular environment acidic due to more CO_2 production. A high DCAD, being alkalogenic in nature, reduces the extent of that acidity and thereby increases cellular glucose uptake whilst reverse is true for negative DCAD. An increased milk production due to high DCAD had also been reported by other workers (Hu and Murphy, 2004; West *et al.*, 1991; Delaquis and block, 1995). However, Roche *et al.* (2003) reported that increased DCAD did not affect milk yield significantly. The plausible explanation of their findings might be very high DCAD level (0 to 760 mEq/ kg) used.

A meta-analysis was conducted to determine the effect of varying DCAD on milk yield (Hu and Murphy, 2004). They presented a model, which pointed out that milk yield increased linearly with increasing DCAD. Block (1994) also reported that milk production increased with increasing DCAD in lactating cows. In these animals, intracellular environment becomes acidic due to excessive production of CO_2 and thus, high DCAD diet is used to make the cellular environment alkaline which is prerequisite for optimum cellular functions in lactating animals (Block, 1994).

Roche *et al.* (2003b) reported that milk production decreased with increasing DCAD level in cows. The probable explanation of this decreased milk yield might be a wider range (+21 to +127 mEq/100 g DM) of DCAD that decreased DMI by the animals. They further stated that a DCAD above +21mEq/100gDM would be responsible for reduction in milk yield. These results were also in agreement with proposal of Sanchez *et al.* (1994) for optimum DCAD level. Roche *et al.* (2005) fed diets containing various DCAD levels (from 23, 45, 70 and 88 mEq/100 g DM) and reported that milk production increased with increasing DCAD level although the difference was non-significant. The lack of DCAD effect on milk production might be attributed to very high levels of DCAD used in their study. Similarly, Belibasakis and Triantos (1991) pointed out that Na_2CO_3 supplementation in the diet resulted in non-significant increase in milk production. English *et al.* (1983) also reported non-significant effect on milk production by buffer addition in corn silage and hay crop silage based rations. Other researchers (Rogers *et al.*, 1985; Eichelberger *et al.*, 1985; Arambel *et al.*, 1988) reported that supplementation of $NaHCO_3$ in alfalfa hay and concentrate based diets did not affect milk yield significantly. Similar findings have also been observed by other investigators (Ghorbani *et al.*, 1989; Cassida *et al.*, 1986; Jordan *et al.*, 1985). The lack of effect of buffer supplementation might be due to high fiber content of the total ration (ADF 20%).

Milk protein percent, lactose and solid not fat (SNF) remained unaffected while total solids (TS) increased by Na_2CO_3 supplementation in the diet (Belibasakis and Triantos, 1991). A non-significant effect of DCAD on milk protein was observed in other studies (Tucker *et al.,* 1988; Delaquis and Block, 1995b) Similar findings were noticed by other workers (Arambel *et al.*, 1988; Eickelberger *et al.*, 1985; Rogers *et al.*, 1985) who reported that supplementation of $NaHCO_3$ in alfalfa hay had non-significant effect on milk protein percent and yield. However, Escobosa *et al.* (1984) and Rogers *et al.* (1982) reported that supplementation of buffer in the diet increased milk protein percent and yield.

Supplementation of Na_2CO_3 in cows resulted in increased milk fat percent and yield compared to those without Na_2CO_3 supplementation (Belibasakis and Triantos, 1991). An increase in fat FCM, SNF, or milk fat was also observed by Na_2CO_3 supplementation in coastal Bermuda grass (Loften and Mertens, 1979) and grass hay (Edwards and Poole, 1983). This might be attributed to the beneficial effect of buffer on milk fat (Canale and Stokes, 1988). Other investigators (Escobosa *et al.*, 1984; Edwards and Poole, 1983; Ghorbani *et al.*, 1989; Caddisa *et al.*, 1988) also noticed an increase in milk fat in cows offered diets supplemented with buffer. Contrary to these findings, Rogers *et al.* (1985) and Eickelberger *et al.* (1985) reported that milk fat percent remained unaltered by buffer supplementation. Arambel *et al.* (1988) also pointed out non-significant effect of $NaHCO_3$ supplementation on milk fat percent in early lactating cows.

Fat production was impervious by manipulation of DCAD during all stages of lactation (Delaquis and Block, 1995b). They observed reduction in milk fat percent during mid lactation with increasing DCAD, which might be associated with increased milk production. Tucker *et al.* (1988) observed non-significant effect of DCAD on milk fat percent. However, other workers (West *et al.*, 1991; Tucker *et al.*, 1991; Escobosa *et al.*, 1984) reported that DCAD had significant effect on milk fat percent. Milk fat percent has positive association with rumen pH (Allen, 1997) indicating adequacy of rumen pH and effective NDF (Klover and deVeth, 2002). Increased milk fat at high DCAD might be due to increased uptake of preformed fatty acids from the circulatory system as a result of increased DMI or increased de novo fatty acids synthesis which accounts for 60 percent bovine milk fatty acids (Bauman *et al.*, 1974). High DCAD diet resulted in greater VFA production due to increased DMI, which provided more substrate for synthesis of de novo fatty acids (Roche *et al.,* 2005). Moreover, high DCAD diets increase rumen pH, which shifts fermentation in favor of acetate and butyrate synthesis (Kaufmann *et al.*, 1980; Klover and de Veth, 2002). This shift in fermentation yields increased amount of acetate and butyrate and thus, enhanced *de novo* fatty acid synthesis that ultimately increases milk fat synthesis.

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