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 \((\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{S}^-)\) 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 \(\text{H}^+\) to high DCAD is very quick. It improves ruminal \(\text{NH}_3\) concentration, acetate while propionate and butyrate decrease which results in higher acetate to propionate ratio. Blood \(\text{pH}\), \(\text{HCO}_3^\text{–}\), serum cation anion balance (\(\text{Na}^+ + \text{K}^+ - \text{Cl}^- + \text{S}^-\)) also tended to increase with increasing DCAD. It increases urine \(\text{pH}\) which reflects alteration in blood \(\text{pH}\). 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 (\(\text{Na}^+\) and \(\text{K}^+\)) and anions (\(\text{Cl}^-\) and \(\text{S}^-\)), 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 \(\text{pH}\) (Stewart, 1981). Following equation is most commonly used for calculation of DCAD

\[
\text{(Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{S}^-) = \text{mEq/100 gm of DM}
\]

(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 \(\text{pH}\) and \(\text{HCO}_3^\text{–}\); e.g. high DCAD diet decreases blood \(\text{H}^+\) concentration and increases blood \(\text{HCO}_3^\text{–}\), blood and urine \(\text{pH}\) that indicate the improved blood buffering capacity (Roche et al., 2005; West et al., 1991). Low DCAD increases blood \(\text{H}^+\) 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, 327 meq/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 NaHCO₃ 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 NaHCO₃ supplementation improved the digestibility of NDF in both diets but did not significantly affect the digestion of other nutrients. In rumen NaHCO₃ is bifurcated into Na⁺ and HCO₃⁻ 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 cellulolytic microbial activity because it is directly related with rumen pH. The rumen pH is a principal factor for controlling microbial activity. 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 NaHCO₃ 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 NaHCO₃ 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 NaHCO₃ 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 6h, it dropped rapidly. Thus supplementation of buffer decreased ruminal fluid acidity.

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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 NH₃-N concentration increased with the addition of buffer and was highest at 0.7% NaHCO₃ and 0.28% MgO (Stokes et al., 1986). They further stated that the close relationship did not exist between ruminal NH₃-N and urinary N losses. Kennelly et al. (1999) pointed out that rumen NH₃-N concentration increased with buffer supplementation while concentrate feeding depressed it. They observed an interaction between dietary buffers and ruminal NH₃-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 NaHCO₃ 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% NaHCO₃ 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 HCO₃⁻ tended to increase as DCAD level was increased in early lactating cows (Roche et al., 2005). Increased blood pH and HCO₃⁻ 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 HCO₃⁻ 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 HCO₃⁻ 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 HCO₃⁻ 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 pCO₂ and H₂CO₃ (Hill, 1990). High DCAD diets decreased blood H⁺ and thus resulted in increased blood HCO₃⁻ (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 HCO₃⁻ 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 HCO₃⁻ and lowered blood H⁺ concentrations compared with low DCAD diets. Blood pH and HCO₃⁻ 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 HCO₃⁻ production and H⁺ excretion (Tucker et al., 1992). Huber (1976) reported that rumen pH declined rapidly with a decrease in blood HCO₃⁻ 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, HCO₃⁻ and feed intake to normal level. Blood HCO₃⁻ and pH returned to normal as CI 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 HCO₃⁻ and total CO₂ increased cubically with increasing DCAD. Blood pH and HCO₃⁻ contents declined in cows receiving low DCAD diet. The decreased blood HCO₃⁻ 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 HCO₃⁻ and were more alkaline. A cubic effect on blood base was observed with increasing DCAD, possibly due to dietary HCO₃⁻.

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 HCO₃⁻ 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 HCO₃⁻ (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. Krijgseld 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.

Urinary 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 urinary pH reflected increase in blood pH and kidneys played a vital role to
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minimize this change by making the urine pH alkaline, by excreting more H⁺ and conserving more HCO₃⁻ (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 HCO₃⁻ excretion increased by high DCAD diet and it decreased by low DCAD diet. Slightly more HCO₃⁻ 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 HCO₃⁻ and decreased serum CI⁻ 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 HCO₃⁻ 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 NaHCO₃ (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 NaHCO₃ (Klimmer 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 CO₂ 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 CO₂ 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, Bellbasakis and Triantos (1991) pointed out that Na₂CO₃ 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 NaHCO₃ 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., 1988; 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$_2$CO$_3$ 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$_2$CO$_3$ in cows resulted in increased milk fat percent and yield compared to those without Na$_2$CO$_3$ supplementation (Belibasakis and Triantos, 1991). An increase in fat FCM, SNF, or milk fat was also observed by Na$_2$CO$_3$ 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 percent has positive association with rumen pH (Allen, M.S. 1997) indicating adequacy of rumen pH and effective 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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