

## A REVIEW ON ACID BASE STATUS IN DAIRY COWS: IMPLICATIONS OF DIETARY CATION-ANION BALANCE

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### ABSTRACT

The acid base status of a dairy cow is maintained within a narrow range. The key mechanisms involving blood, cells and lungs, perform this function. Although other minerals have an impact on acid base metabolism, the minerals used in dietary cation-anion balance (DCAB) namely sodium (Na), potassium (K) and chloride (Cl) have the greatest effect. Hence, acid base status implicates other biological functions of dairy cows. Low DCAB prepartum reduces the incidence of milk fever and increases the productivity by simmering down the severity of hypocalcaemia. High DCAB diets have proved to increase dry mater and water intake and production and to mitigate the effects of heat stress.

**Key words:** Dietary cation-anion balance, acid base balance, milk fever, heat stress, dairy cow.

### INTRODUCTION

Regulation of hydrogen ions balance is similar as the regulation of other ions in the body. To achieve homeostasis, there must be a balance between the intake and the removal of hydrogen ions from the body. A net influx of any mineral cation or anion into the body of animal changes its acid base status. The difference between the amounts of absorbable anions and cations in a diet determines the acid base status of a cow (Pehrson *et al.*, 1999). A cow becomes acidotic when it absorbs more anions than cations while she becomes alkalotic when it absorbs more cations than anions (Tucker *et al.*, 1991).

The kidneys play major role in regulating the acid base balance. However, precise control of extra cellular fluid hydrogen ion concentration involves much more than simple elimination of hydrogen ions from the kidney. There are also multiple acid base buffering mechanisms involving cells, blood and lungs that are essential in maintaining normal hydrogen ion concentration both in extra and intra cellular fluids (Guyton, 1986).

The term "fixed ions" refers to bio-available ions that are not metabolized, namely,  $\text{Na}^+$ ,  $\text{K}^+$  and  $\text{Cl}^-$ . The fixed ions balance plays a major role in determining acid-base balance in biological fluids (Stewart, 1978). The importance of these minerals in ruminant metabolism lies in their indirect participation in osmotic balance, acid base balance and integrity, and pumping mechanisms of cell membranes.

This paper reviews the acid base status in dairy cows with special reference to the implications of changing dietary anion and cation balance.

#### Sodium potassium pumping mechanism

The sodium-potassium pumping mechanism of cells actively maintains high levels of  $\text{K}^+$  and low levels of  $\text{Na}^+$  intracellularly and requires energy in the form of ATP (Block, 1994). Because glucose is the main source of cellular energy, a slowdown of the sodium-potassium pump would not allow cells to operate at full potential, especially for the active mammary gland for which high quantities of glucose are used for lactose synthesis. Obviously, excess of one cation in relation to other cation causes the pump to slow (causing the cell to use more energy in pumping but obtain less glucose) or to speed up beyond an optimal level (creating high energy requirements to maintain the cell).

In the posterior segment of the intestine,  $\text{Cl}^-$  is absorbed, when it is in excess of  $\text{Na}^+$ , in exchange for a bicarbonate ( $\text{HCO}_3^-$ ) to maintain electrical neutrality. If insufficient  $\text{Na}^+$  is present to allow the absorption of sodium chloride (NaCl), an excess drain of blood  $\text{HCO}_3^-$  can lead to an acidotic condition. Alternatively, there is potential for intestinal exchange of ingested  $\text{Na}^+$  with  $\text{H}^+$  in blood when  $\text{Na}^+$  in excess of  $\text{Cl}^-$  leads to metabolic alkalosis. The  $\text{Na}^+$  exhibited the alkalogenic effect, as suggested by Leach (1979).

### Buffering mechanisms

There are three primary systems that regulate the hydrogen ion concentration in the fluids to prevent acidosis or alkalosis: (1) the chemical acid-base buffer system of the body fluids, which immediately combines with acid or base to prevent excessive changes in hydrogen ion concentration, (2) the respiratory centers, which regulate the removal of  $\text{CO}_2$  (and therefore,  $\text{H}_2\text{CO}_3^-$ ) from the extra cellular fluid and (3) the kidneys, which can excrete either acid or alkaline urine, thereby reducing the extra cellular fluid hydrogen ion concentration toward normal during acidosis or alkalosis (Guyton, 1986).

Buffer systems play an important role in maintaining blood pH; the bicarbonate/carbonic acid system is a buffer relationship; the body can control by altering partial pressure of carbon dioxide ( $\text{pCO}_2$ ) by respiratory means or by metabolic control of  $\text{HCO}_3^-$  concentration in blood (Leach, 1979) by diffusing through cell membrane. This diffusion of the elements of the bicarbonate buffer system causes the pH in intracellular fluids to change when there are changes in extra cellular pH.

### Chloride shift

Principle protein buffer in blood is potassium salt of oxy-hemoglobin ( $\text{KHbO}_2$ ) in erythrocytes. As tissue carbon dioxide diffuses from the tissue cells into the capillaries, it combines with water in the presence of carbonic anhydrase to form carbonic acid in red blood cell (Georgievskii, 1981). Some of the carbonic acid enters the plasma and rest reacts with  $\text{KHbO}_2$  to form  $\text{HCO}_3^-$ , thus liberating the oxygen for respiration and  $\text{K}^+$  from  $\text{KHbO}_2$ . The  $\text{H}^+$  is bound by the hemoglobin buffer system. The  $\text{HCO}_3^-$  enters the plasma in exchange with  $\text{Cl}^-$ . Sodium bicarbonate is formed in the plasma and the  $\text{Cl}^-$  in erythrocyte is neutralized by  $\text{K}^+$  released in exchange of  $\text{HCO}_3^-$  with  $\text{Cl}^-$ . The reaction is reversible in lungs where  $\text{Cl}^-$  is transferred back to the plasma neutralizes the  $\text{Na}^+$  released when  $\text{HCO}_3^-$  reenters the erythrocyte for removal of  $\text{CO}_2$  on respiration. This exchange is called chloride shift (Georgievskii, 1981). If these ions are not balanced, the production of alkalosis or acidosis is possible via insufficient exchange of  $\text{H}^+$  and  $\text{HCO}_3^-$ .

### Renal control of acid base balance

The kidneys control acid base balance by excreting either acidic or basic urine. Excreting acidic urine reduces the amount of acid in extra cellular fluid. Whereas, excreting basic urine removes the base from extra cellular fluid (Tucker *et al.*, 1988). The kidneys must also prevent the loss of bicarbonate in the urine. The kidneys regulate the extra cellular fluid  $\text{H}^+$  concentration through three basic mechanisms i.e.

secretion of hydrogen ion, reabsorption of filtered bicarbonate ions and production of new bicarbonate ions.

The mobilization of  $\text{H}^+$  in the proximal tubules of the kidney, the secretion of  $\text{H}^+$  and the production of ammonia in the distal tubules of the kidney all depend on reabsorption of  $\text{Na}^+$  to neutralize electrically the absorption of  $\text{HCO}_3^-$  from the tubular cell to the blood (Block, 1994). If excess  $\text{Cl}^-$  is present in the glomerular filtrate,  $\text{Cl}^-$  in the filtrate and  $\text{HCO}_3^-$  in the cell may exchange, resulting in  $\text{NaCl}$  reabsorption and reduction in  $\text{HCO}_3^-$  absorption. When the animal is under the stress of mild acidosis in extracellular fluids, the kidneys may conserve  $\text{HCO}_3^-$  ions by reabsorption; the reverse is true for alkalosis (Gaynor *et al.*, 1989). A reciprocal ion of same charge is needed to maintain neutrality and  $\text{Cl}^-$  is used for this purpose because it is the anion in greatest concentration in extra cellular fluid (Coppock, 1986).

### Cation-anion relationship

The increasing use of sodium bicarbonate and electrolytes has stimulated interest among ruminant nutritionists in this topic. Leach (1979), a poultry nutritionist, used the term "cation-anion balance, to refer to interrelationship among sodium, potassium and chloride. He has stated that main adverse effect comes from the acidogenic action of excessive  $\text{Cl}^-$  and this effect could be reversed by the alkalogenic nature of sodium and potassium.

Dietary cation-anion balance (DCAB) can be defined quantitatively as milliequivalents of  $\{(\text{Na}^+ + \text{K}^+) - \text{Cl}^-\}$ , and it affects the acid base status in adult ruminants (Fredeen *et al.*, 1988), lactation performance (Tucker *et al.*, 1988), Ca metabolism in prepartum dairy cow (Block, 1984; Gaynor *et al.*, 1989, Oetzel *et al.*, 1991) and P metabolism in young calves (Beighle *et al.*, 1988). As  $\text{Na}^+$  or  $\text{K}^+$  in this equation is increased, the DCAB will increase if Cl content remains constant (Jackson *et al.*, 1992). Absorption of  $\text{Na}^+$  or  $\text{K}^+$  is linked to the generation of systemic  $\text{HCO}_3^-$  whereas  $\text{Cl}^-$  absorption increases systemic free proton ( $\text{H}^+$ ) generation (Takagi and Block, 1991). As a result of DCAB increase, blood pH tends to rise, whereas a reduction in DCAB increases systemic  $\text{H}^+$ . Cows in heat stress often experience a respiratory alkalosis, resulting from hyperventilation (West *et al.*, 1991). Compensation results in urinary bicarbonate loss in an attempt to balance the ratio of carbonic acid to bicarbonate in the blood (Tucker and Hogue, 1990).

Gastrointestinal tract (GIT) absorption of mono-valent mineral ions from the diet influences systemic acid base status. Absorption of cations occurs in exchange for the secretion of  $\text{H}^+$  ions into the GIT lumen, whereas anion absorption is accompanied by the

secretion of  $\text{HCO}_3^-$  (Lomba *et al.*, 1978). The net result is that cation absorption increases the systemic base generation and anion absorption increases systemic acid generation. Therefore, DCAB will influence the blood acid base status (Tucker and Hogue, 1990).

A negative DCAB has excessive amounts of anions in relation to cations in the diet and is considered acidogenic in nature. The highly acidogenic  $\text{Cl}^-$  diet overwhelmed the capacity of the kidneys to excrete  $\text{H}^+$  ions and maintains blood pH at normal level (West *et al.*, 1992). Acid base balance, as affected by acidogenic diets, alters Ca metabolism (Gupta *et al.*, 1970; Horst and Jorgensen, 1973; Fredeen *et al.*, 1988; Wang and Beede, 1992) via resorption (Escobosa *et al.*, 1984; Takagi and Block, 1991; Goff *et al.*, 1991), intestinal Ca absorption (Block, 1994) and renal handling of Ca (Jackson *et al.*, 2000).

### Heat stress

Cows in heat stress often experience a respiratory alkalosis, resulting from hyper-ventilation (West *et al.*, 1991). Compensation results in urinary bicarbonate loss in an attempt to balance the ratio of carbonic acid to bicarbonate in the blood. Thus, acid base status of the heat stressed cows is altered from that of normothermic cows. West *et al.* (1992) demonstrated that high DCAB increases the dry matter intake in heat stressed cows. They hypothesized that the increase in blood buffering capacity with higher DCAB was responsible for the increase in dry matter intake. Water intake also increases with increase in dry matter intake but milk fat and protein percentages are not altered by higher DCAB. Delaquis and Block (1991) showed that there were increases in water intake, absorption and urinary volume with elevated DCAB.

### Milk fever

Initiation of lactation places one of the greatest stresses on Ca homeostasis and is associated with hypocalcemic parturient paresis among high producing dairy cows (Horst and Reinhardt, 1983). On the day of parturition, dairy cows commonly produce ten liter or more colostrum containing 23 g or more of Ca that is six times as much as the extra cellular pool contains (Goff *et al.*, 1987). Most cows adapt to this Ca stress by rapidly increasing intestinal absorption and bone Ca resorption, activities regulated by parathyroid hormone and 1-25 dihydroxy-vitamin D (Goff *et al.*, 1991).

Dietary factors can greatly influence the incidence of milk fever in dairy cows. Feeding a low calcium diet prepartum stimulates parathyroid hormone secretion and 1-25 dihydroxy-vitamin D production prior to parturition, activating calcium transport mechanism in bone and intestine that would be needed to adapt to

lactational Ca demand (Takagi and Block, 1991). A negative DCAB diet (1) may increase the intestinal absorption of Ca by reducing pH in the GIT, causing an increase in the more soluble forms of Ca (ionized form) present, (2) may cause an alteration in acid base balance of animal, resulting in an increase of Ca availability from exchangeable Ca pool and (3) may cause the reduction in the Ca absorption by interference created by the presence of excess of cation mineral elements such as aluminum and magnesium (Tucker *et al.*, 1991). This idea is supported by the work of Block (1984), who reported that feeding a diet low in DCAB during dry period completely prevented parturient paresis.

### CONCLUSION

Acid base status of a dairy cow involves certain biological functions to follow its suit. The optimal DCAB will not be same for all productive functions. Some biological functions respond better when the balance is positive, but others do so when the balance is negative. More research is needed to glimmer this obscure and gloomy area of ruminant nutrition.

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