DIETARY ELECTROLYTE BALANCE AND ANIONIC DIETS FOR DAIRY COWS IN LATE GESTATION

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INTRODUCTION

The time around parturition is particularly crucial in the productive cycle of the dairy cow because of dynamic physiological changes which happen. The occurrence of metabolic diseases (milk fever, hypocalcemia, retained placenta, uterine prolapse, ketosis, displaced abomasum, udder edema, etc.) may be problematic. These disease episodes may impact negatively on subsequent reproductive and lactational performance. Recently there has been considerable interest in altering dietary electrolyte balance (DEB) [or fixed ion balance] to possibly affect metabolism, health and production.

Dietary electrolyte balance (DEB) is a relatively new concept in dairy cattle nutrition and diet formulation in the United States. Although limited, some research suggests that formulating rations for cows in late gestation (heavy springers) which have a negative DEB (diets which are anionic or acidogenic) may decrease the incidence of milk fever and hypocalcemia around the time of parturition and improve subsequent lactational performance and health. This paper will review some of the studies evaluating calcium (Ca) restriction in the springer ration and information on the basic principles of DEB and acid-base status, effects of electrolytes and mineral salts on acid-base status, equations for calculating DEB, and recent research investigating use of negative DEB in springer rations for dairy cows in late gestation.

PREPARTUM CALCIUM INTAKE AND INCIDENCE OF MILK FEVER

The most widely practiced method for controlling the incidence of milk fever is to restrict the intake of Ca during late gestation. The NRC (1989) suggests Ca intake of 36 to 42 g per day for mature dry cows (1200 to 1300 lb body weight) during the last 2 months of gestation. However, often times it is very difficult to formulate diets from available feedstuffs which will result in Ca intake this low; particularly when often it is desired to have cows consume as much bulky feed as possible during late gestation. Many of these bulky feeds are relatively high in Ca content.

A review of some of the literature suggests that restriction of Ca intake in the late prepartum period was not always as efficacious as desired. Figure 1 (following page) shows the incidence of milk fever of cows consuming varying quantities of Ca. Data from seven different experiments are represented in the plot and Ca intake ranged from about 10 to over 200 g per cow per day. Literature

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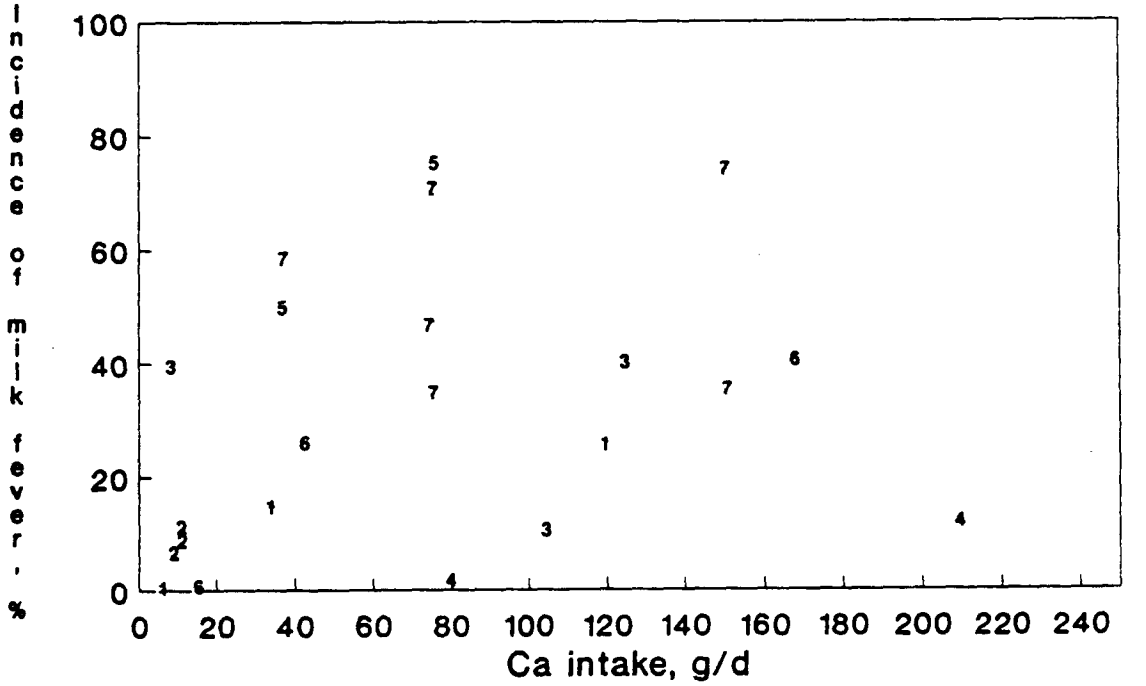
Figure 1. Effect of dietary calcium intake perpartum on incidence of milk fever in dairy cows. Data points are signified by numbers according the literature source: 1 = Boda and Cole, 1954; 2 = Boda, 1956; 3 = Kendall et al., 1970; 4 = Gardner and Park, 1973; 5 = Beitz et al., 1974; 6 = Wiggers et al., 1975; and 7 = Josson et al., 1980.



citations from which the data were obtained are listed in the legend of the figure and Literature Cited. Quite high Ca intake increased the incidence of milk fever compared with a lower intake rate in the experiments of Gardner and Park (1973) and Wiggers (1975). Reducing Ca intake from 75 - 150 g to 37 g/cow/day did not reduce the incidence of milk fever in Sweden (Josson et al., 1978). Kendall et al., (1970) found no benefit by restricting Ca intake to 10 g/day compared with 103 or 122 g/d. In three other studies, limiting Ca intake to less than 20 g/day reduced the incidence of milk fever (Boda and Cole, 1954; Boda, 1956; Wiggers, 1975). However, generally it is impractical or impossible to limit Ca intake to such low rates. It also has been suggested that severely limiting Ca intake prepartum may reduce milk production during the following lactation.

Therefore, it would be a great practical advantage to have another means of reducing or preventing the incidence of milk fever and hypocalcemia. The prospect of instituting some sort of dietary manipulation of the springer ration could offer a relatively easy, and perhaps inexpensive approach, if effective. A potential manipulation was proposed nearly 30 years ago in Norway (Ender et al., 1962), but has been researched only recently in North America (Block, 1983; Oetzel et al., 1988; Gaynor et al., 1989).

BASIC PRINCIPLES AND DEFINITIONS OF DEB

The next four sections deal with the basic principles about DEB, and its influence on acid-base status, Ca metabolism and the calculation of DEB.

Electrolyte balance involves numerous chemical and physical properties. A basic understanding of these is essential for understanding the complex interactions of electrolytes, acid-base status, and production.

Atoms cannot be weighed so the quantitative unit of atomic weight was established. This was determined to be 6.02×10^{23} atoms (Avogadro's number; equal to one mole of atoms). Because ions react according to valence (or charge) the unit of expression is moles of charge (i.e., milliequivalents) rather than moles of atoms. For monovalent ions, concentrations in equivalents are the same as concentrations in moles. For n-valent ions, concentrations in equivalents are just n times the concentration in moles. The importance of this is seen in the reaction of one mole (1 g) of hydrogen ions (H^+) with one mole (35.5 g) of chlorine ions (Cl^-). The reactants have the same number of milliequivalents (mEq), but differ drastically in weight. In using DEB in feed formulation it will be necessary to convert concentrations to mEq, which is simply an adjustment for ionic charge; see section on calculations later in this paper. Many substances, when dissolved in solution, dissociate into ions and due to their ability to conduct electricity (or carry a charge), are called electrolytes. Strong electrolytes are by definition completely dissociated, whereas weak electrolytes change their degree of dissociation. Ions derived from strong electrolytes are strong ions. The most common strong ions in biological solutions are Na^+, K^+, Cl^-, Mg^{2+}, SO_4^{2-}, Ca^{2+}, and a few organic anions, notably lactate$^-$ (Stewart, 1981). In all biological fluids the sum of positive charges must equal the sum of negative charges (electrical neutrality). Dietary electrolytes contribute positive and negative charges to the body and therefore can affect electrical balance, subsequent acid-base status, and animal performance.

ACID-BASE STATUS

Prior to our discussion on specific macromineral salts and DEB we shall first review some of the pertinent information on acid-base status as it involves electrolytes. For this discussion an acid will be defined as any substance that when added to a solution increases the hydrogen ion concentration, $[H^+]$. A base decreases $[H^+]$.

Kronfeld (1979) placed maintenance of physiological acid-base status high on the list of homeostatic priorities, third behind O_2 need and heat dissipation, just ahead of CO_2 elimination and water retention. The respiratory and renal systems maintain long term acid-base status. A series of buffer systems (particularly the

bicarbonate-carbonic acid buffer pair) respond to short-term perturbations.

Acid-base status is a function of $[H^+]$. Concentrations of H^+ generally are measured in extracellular fluid (ECF) in very small quantities - one tenth to one hundred millionth of an Eq/L (approximately 40 nEq/L; pH 7.4). Although minuscule, $[H^+]$ is critical to living systems for several key reasons. Because H^+ atoms are so small they have a large charge density and electric field gradient per gram-atomic weight; hydrogen bonds are important in determining molecular structure and configuration; enzyme activities are very sensitive to $[H^+]$; and finally, H^+ turnover exceeds that of any other single metabolite (Stewart, 1981; Morris, 1986).

Blood acid-base status parameters in cattle including pH (normal range of 7.35 to 7.5), pCO_2 (35 to 44 mm Hg), HCO_3^- (20 to 30 mmol/L), and serum electrolytes may be good measures of physiological responses to dietary and environmental stimuli (Duncan and Prasse, 1986), but these are not available commonly for assessment of dairy cattle nutrition programs. The rumen is a source of large quantities of H^+ via ruminal fermentation. When ruminal acid production cannot be regulated adequately, changes in blood acid-base status occur. In conditions of blood acid excess, HCO_3^- is quickly moved into the blood. Serum HCO_3^- decreases as it combines with H^+ to form carbonic acid (H_2CO_3). Also, H^+ may shift into the cell in exchange for K^+. The kidney tries to compensate by exchanging more K^+ and H^+ for Na^+ in the tubules and by forming more HCO_3^- (Collins, 1988). In contrast to Na, K is not conserved well by the kidney. In acidosis, cellular K can be depleted through these exchange mechanisms. Degradation of body tissues, as in cases of negative energy balance, also may deplete K. Sodium and HCO_3^- also can be lost in urine under conditions of acidosis (as summarized by Morris, 1986). Although acute clinical acidosis is relatively uncommon in dairy cattle, milder chronic cases and resulting altered metabolism are more prevalent (Kronfeld, 1976).

EFFECTS OF ELECTROLYTES AND MINERAL SALTS ON ACID-BASE STATUS

Nutrient metabolism results in a large H^+ flux from degradation of carbonaceous compounds. In normal dairy cattle diets, dietary inorganic cations exceed dietary inorganic anions by 20 to 25 mEq/100g diet DM/day. Assuming the diet is electrically neutral some other source of anions must be present in the diet. These "other" anions generally are organic compounds such as bicarbonate, citrate, lactate and fatty acids (Austic, 1988). This results in an excess of organic anions which can be metabolized to HCO_3^- and thus represents potential base to buffer $[H^+]$. Stewart (1981) suggested that three variables were responsible for $[H^+]$ in the body. The total weak acid, pCO_2 and the strong cations minus strong anions.

There are three primary reasons why mineral salts change acid-base status. The mineral salt is solubilized; upon dissociation it is

absorbed; and after absorption, any non-mineral portion is further metabolized such that its end-products do not change [H^+].

To illustrate mineral salts that meet all of the criteria we shall use the example of sodium bicarbonate ($NaHCO_3$) an alkalogenic agent and ammonium chloride (NH_4Cl) an acidogenic agent. Other examples can be found in the review by (Morris, 1986). To understand the exact mechanism of how these mineral salts affect acid-base status their chemical reactions upon absorption should be understood. When $NaHCO_3$ reaches the gastrointestinal tract it combines with water and dissociates as follows:

$$NaHCO_3 + H_2O \text{ <-----> } Na^+ + \underline{OH^-} +$$

$$HCO_3^- + H^+ \text{ <-----> } H_2O + CO_2(gas)$$

The net result is an increase in OH^- or base. The metabolism of HCO_3^- is the key reason that $NaHCO_3$ is base-forming.

Reaction of NH_4Cl has an opposite effect on acid-base status. The dissociation products are NH_4^+ and Cl^-, and NH_4^+ is further metabolized to NH_3 and H^+. Two NH_3 combine with CO_2 to form urea. The overall reaction is:

$$2\ NH_4Cl \text{ <------> } 2\ Cl^- +$$

$$2\ NH_4^+ \text{ <----> } \underline{2\ H^+} +$$

$$2\ NH_3 + CO_2 \text{ <----> urea} + H_2O$$

The acidogenic nature of NH_4Cl is due to the remaining two H^+. Although $NaHCO_3$ and NH_4Cl affect acid-base status differently, the mechanisms are similar because the non-mineral portions of the salts are further metabolized.

Calcium chloride ($CaCl_2$), like NH_4Cl is acidogenic, but for different reasons. Here only a portion of the mineral salt is absorbed. Calcium absorption in dairy cattle is about 38% (as calculated by NRC, 1989) with the remainder excreted in the feces. To maintain neutrality in the digestive tract Ca^{2+} reacts with HCO_3^- to form the precipitate $CaCO_3$.

$$CaCl_2 + HCO_3^- \text{ <-----> } 2\ Cl^- + \underline{H^+} + CaCO_3(ppt.)$$

$CaCl_2$ is acidogenic because it increases H^+ (due to Ca drawing base out of solution).

Note that mineral salts such as NaCl contain "fixed" ions (i.e., there is no non-mineral portion to metabolize). These yield no change in acid-base status (discounting any indirect effect they may have on ruminal osmolality). Sodium chloride is a neutral salt because both Na and Cl are solubilized and absorbed, but neither are metabolized.

CALCULATING DIETARY ELECTROLYTE BALANCE

Dietary electrolyte balance is the difference between total dietary cations and anions. Differential absorption and metabolism make true quantification and characterization difficult. In addition, several equations exist for calculating DEB. The term DEB would more appropriately be called electrolyte difference but to prevent adding more terms to the literature we shall continue to use the term DEB. A true DEB would include all ions solubilized and absorbed, but not metabolized extensively.

Leach (1979) reviewed different expressions of electrolyte interrelationships. The most common procedure used experimentally was a ratio or difference between two or more of the ions. Researchers intuitively knew it was difficult to study one ion without considering others. Common equations describing DEB are as follows:

$$DEB = mEq\ (Na + K - Cl)$$
$$DEB = mEq\ [(Na + K) - (Cl + S)]$$
$$DEB = mEq\ [(Na + K + Ca + Mg) - (Cl + S + P)]$$
$$DEB = mEq\ [(Na + K + Ca + Mg) - (Cl + SO_4 + H_2PO_4 + HPO_4)]$$

The problem with including Ca, Mg, P, and S is due to inadequate information on their physiologically functional state and bioavailability compared with Na, K and Cl (NRC, 1989; Aitken, 1976). As an example, a valence for P of 1.8 instead of 2 has been used (Dwyer et al., 1985) because P exists in the blood as a buffer and only 80% exists as the divalent anion, [(.8)(2)+(.2)(1)] = 1.8.

Most publications on DEB have used the simple Na+K-Cl (in mEq/100g or kg) to calculate electrolyte balance. This expression, while useful, ignores any contribution of other minerals on acid-base status and may need future modification if these other minerals have major influence. To calculate DEB, one must first convert concentrations to milliequivalents with the following equation:

$$\text{mEq/100g of diet DM} = \frac{[(\text{milligrams})(\text{valence})]}{[(\text{atomic weight})]}$$

As an example, calculations of DEB for a lactation diet with .18% Na, .9% K and .25% Cl (NRC, 1989 recommendations) are as follows. In 100g of diet DM there are 180mg Na (.18% = 180mg/100g), 900mg K and 250mg Cl. Therefore, this diet contains:

$$\text{mEq Na} = \frac{[(180\text{mg})(+1\text{ valence})]}{[(23\text{g atomic weight})]} = 7.8\text{ mEq Na/100g DM}$$

$$\text{mEq K} = \frac{[(900\text{mg})(+1\text{ valence})]}{[(39.1\text{g atomic weight})]} = 23.1\text{ mEq K/100g DM}$$

$$\text{mEq Cl} = \frac{[(250\text{mg})(+1\text{ valence})]}{[(35.5\text{g atomic weight})]}. = 7.0\text{ mEq Cl/100g DM}$$

The net milliequivalents =

$$mEq\ (Na + K - Cl) = 7.8 + 23.1 - 7.0 = \underline{+ 23.9/100g\ DM.}$$

Prior to using DEB in ration formulation it will be necessary to correct any dietary deficiencies or toxicities of minerals in question. Note that because NaCl is a neutral salt, rations differing in NaCl content may have the same DEB, but may have profoundly different influences on animal performance. Work at Cornell (Fettman et al., 1984) with low Cl diets produced a primary hypochloremic, secondary hypokalemic metabolic alkalosis. Altered DEB would not improve performance or correct these symptoms unless additional Cl was added.

RESEARCH WITH DAIRY CATTLE ON DIETARY ELECTROLYTE BALANCE

Most research on DEB with dairy cattle has been with dry cows in late gestation. Overall objective of this research was to determine if dietary manipulation of DEB prepartum affects Ca metabolism around the time of calving and incidence of milk fever and hypocalcemia.

DEB In Dry Cow Nutrition

Ender et al. (1962; 1971) and Dishington (1975) in Norway have had longstanding interests in prevention of milk fever, and were among the first to consider the idea that "alkali alkalinity of the diet (ie., AA = mEq [(K + Na) - (S + Cl)] daily)" might be of overriding importance in determination of Ca availability. They also suggested that prevention of milk fever might be possible with a prepartum diet with negative AA. They suggested that dietary manipulation of electrolyte contents might be more efficacious than restricting Ca intake prepartum or vitamin D administration. In an early study 12 of 14 cows fed a basic (cationic) diet supplemented with Na_2CO_3 and $NaHCO_3$ (AA = +20 to +39 mEq/100g diet DM) suffered milk fever, whereas only 1 of 13 cows receiving the same basal diet but supplemented with $CaCl_2$, $Al_2(SO_4)_3$ and $MgSO_4$ (AA = -3 to -13 mEq/100g) in place of the sodium salts showed mild paretic signs. It was possible to induce and prevent milk fever at successive parturitions in the same cow by altering the dietary conditions. The dietary manipulation, formulating an acidic (anionic) or negative DEB diet, appeared to be a convenient prophylactic approach.

Later studies (Dishington and Bjornstad, 1982; Block, 1984; Oetzel et al., 1988; Fredeen et al., 1988) have confirmed the early work and have served to characterize some of the physiological and metabolic responses to anionic diets as well as addressing some practical aspects. In Block's (1984) study, 20 cows were used in a 2-year switchback design to test the effects of making a basal diet positive with excess cations (mEq [(Na + K) - (Cl + S)] = +45/100g) or negative with excess anions (mEq = -17/100g). Cows consuming the anionic diet did not have milk fever, whereas 47%

incidence of milk fever was observed for cows consuming the cationic diet. Subsequently, cows that did not experience milk fever had higher milk yields. Blood plasma of cows fed the anionic diet contained normal Ca and P concentrations at the time around parturition, whereas the cows fed the cationic diet had decreased concentrations of these minerals. Plasma hydroxyproline concentrations of healthy cows not exhibiting milk fever, were higher than for cows afflicted with milk fever, suggesting that the dietary manipulation enhanced mobilization of bone tissue prepartum, thus making more Ca available at this time of high metabolic demand.

To further characterize the effects of DEB on Ca and P balance and metabolism, and acid-base status, Fredeen et al.(1988) used pregnant nonlactating and lactating dairy goats. The DEB was calculated as: mEq (Na + K - Cl)/100g diet DM. Diets with +40 to +50 mEq DEB resulted in normal acid-base status. When DEB was less than +10 mEq/100g subclinical hyperchloremic, hyponatremic metabolic acidosis produced hypercalciuria, enhanced Ca and P absorption and apparently facilitated Ca resorption from bone tissue. These data suggest that lower DEB is effecting physiological mechanisms of the prepartum doe and cow to help mediate necessary Ca metabolism around the time of parturition.

Undoubtedly the most comprehensive experiment to date with dry cows has been that of Oetzel et al. (1988). Forty-eight multiparous Holstein cows with no previous history of milk fever were fed one of four diets starting 21 d prepartum in a 2 X 2 factorial arrangement of treatments. Factors were: daily Ca intake of 53 or 105 g and a DEB [mEq (Na + K) -(Cl + S)/100g] of -7.5 or +18.9. To make the anionic diet, 100g each of NH_4Cl and $(NH_4)_2SO_4$/cow/day were used. This study also addressed practical aspects of formulation of an anionic dry cow ration because it considered the cost of the acidogenic salts. Feed intake was not affected by the treatments fed in a total mixed ration consisting of 62% corn silage, 14% oat hay, 9% alfalfa hay plus concentrates. The incidence of milk fever was 4% with the anionic diet, but 17% without the added ammonium salts. Cows fed the anionic diet also had higher total and ionized serum Ca concentrations at parturition. When Ca intake was low, the lower DEB had no effect on parturient hypocalcemia (ionized blood Ca below 4.0 mg/dl). However, based on risk analysis, when Ca intake was high, supplementation with ammonium salts decreased the risk of parturient hypocalcemia 10-fold. Serum concentration of Mg, P, Na, K, and Cl at calving were not affected by dietary treatments.

From a practical standpoint the ammonium salts were attractive because they were relatively less expensive than other potential acidogenic salts. Ammonium chloride and ammonium sulfate cost only $.045 and .015/Eq, respectively, compared with $.196, .057, .091, and .080/Eq for aluminum sulfate, magnesium sulfate, granular calcium chloride and pelleted calcium chloride, respectively (Oetzel et al., 1988). The authors warned that potential toxicity of the ammonium salts exists. They concluded that feeding 100g of

each daily in a total mixed ration was not a problem as long as consumption was spread out over the day. Feeding large quantities of these ammonium salts as part of a grain mix separate from the forage, however, could lead to palatability problems. Also, feed mixing errors could be dangerous. In some situations using a portion of other commercially available salts (ie., magnesium sulfate) may be preferred and safer.

Recently Tennessee researchers used additions of the chloride salts of magnesium, ammonium and calcium to make a low positive DEB diet (+22 mEq/100g DM), and compared it with intermediate (+59.9 mEq) and high positive (+125.8 mEq) DEB diets (Gaynor et al., 1989). Eighteen multiparous Jersey cows were fed their respective diets for 6 weeks prior to calving; one cow was dropped from the study due to physical injury. Number of clinical cases of milk fever were 0 of 5, 2 of 6 and 1 of 6 for cows fed the low, intermediate and high positive DEB diets, respectively. Plasma Ca concentrations within 36 h after calving were 7.0, 6.5 and 6.3 mg/dl for the low, intermediate and high positive DEB treatments. Perhaps the most useful new information from the study was the suggestion that the low DEB diet may have enhanced the action of vitamin D in Ca metabolism. Plasma concentrations of 1,25 dihydroxy vitamin D were increased in cows fed the low positive DEB diet compared with the other two groups. Authors suggested that the intestinal absorption of Ca may have been activated to a higher degree in these cows. Similar data need to be collected from cows consuming a truly anionic (negative) diet to verify this observation and possible mode of action.

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