Practical Management of Prepartum Anionic Diets for Dairy Cattle

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Feeding anionic diets to prepartum dairy cows has become a common strategy to prevent parturient paresis. However, implementing anionic diets in the field is challenging. Feeding anionic salts will help prevent parturient paresis (Block 1984, Dishington 1975, Oetzel *et al* 1988) and perhaps improve calcium homeostasis by increasing ionized calcium levels at parturition (Oetzel *et al*, 1988, Beede *et al,* 1992), but the mechanism of action of the anionic salts has not been clearly defined. Since the mechanism of action is not defined a number of questions arise during manipulation of the dietary cation-anion difference (DCAD).

What determines acid base balance in the cow?

Acid-base balance in the body is determined by the regulation of $pC0_2$, and the concentration of strong ions and weak acids by the lungs, liver, kidney and gastrointestinal tract (Stewart, 1981). However, the major factor affecting acid-base balance interactions between biological fluids are movements of strong ions across membranes separating them (Stewart, 1981). Strong ions are electrolytes that dissociate completely into ions in solution. Changes in H^+ and $HCO3^-$ concentration between body fluids result from exchanges of strong ions and not from adding H^+ or $HCO3^-$. The most common strong ions are Na^+, K^+, Cl^-, Mg^{++}, SO4 and Ca^{++} (Stewart, 1981).

The strong ion difference (SID), also referred to as the dietary cation difference (DCAD), is defined as the sum of all the strong base cations minus the sum of all the strong acid anions and is expressed in milliequivalents (meq)/kg of dry matter. Equation 1 is the most common equation used in the literature and by most computer ration balancing programs to calculate the DCAD.

Equation 1

$$[(Na^+ + K^+) - (Cl^- + S^{--})]$$

Goff (1992) suggests that equation 2, which takes into account the relative absorption rates of the different ions, more accurately describes the relationship of DCAD to acid-base balance.

Equation 2

$$[(Na^+ + K^+ + .3Mg^{++} + .38Ca^{++}) - (Cl^- - .6S^{--} + .5P^{---})]$$

From equation 2, it is apparent that monovalent ions are absorbed better than ions of higher valence. Cl^- is probably a better acidifying ion than S^{--} because it is absorbed better. K^+ is absorbed with high efficiency compared to Ca^{++} and Mg^{++}. The commonly used DCAD equation (equation 1) has mostly monovalent ions except for S^{--}. The trend toward higher soil fertility on dairy farms resulting from excess manure application to corn and grass hay would favor an excess of K^+ absorbed by the cow. Since Na^+ is generally low in feeds, the major ions affecting acid base balance in the cow are K^+ and Cl^-.

How does the DCAD affect the acid base balance?

To maintain electrical neutrality, the sum of the positively charged ions must equal the sum of the negatively charged ions (Stewart, 1981). To maintain electrical neutrality, when an ion moves across a membrane, it must either be exchanged with an ion of the same charge or move together with an ion of the opposite charge (Stewart, 1981). A neutral solution exists when the DCAD=0. When strong acid anions exceed strong base cations by say the addition of anionic salts, the DCAD is negative and the $[H^+]$ will be larger than $[OH^-]$ and the solution will be acidic. When the strong base cations exceed the strong acid cations, the DCAD is positive and the $[OH^-]$ will be greater than $[H^+]$ and the solution will be alkaline (Stewart, 1983). Common anionic salts such as $CaCl_2$ cause acidification because the Cl^- is absorbed more efficiently than the Ca^{++} resulting in a negative DCAD. To maintain electrical neutrality when the Cl^- is absorbed, either an H^+ must accompany it, or an OH^-, $HCO3^-$ or $CO3^{--}$ must move in the opposite direction. As a result, the $[H^+]$ will be larger than the $[OH^-]$ and the solution will be acidic.

How is the DCAD commonly calculated?

Equation 3 can be used to calculate the DCAD in meq/kg of dry matter.

Equation 3

[(%Na/0.0023) + (%K/0.0039)] - [(%Cl/0.00355) + (%S/0.0016)]

What determines calcium homeostasis?

The daily requirement of calcium prior to parturition is approximately 30 grams. There is a massive demand for calcium at parturition when the cow loses 30 grams or more of calcium to the milk. Calcium homeostasis is controlled by parathyroid hormone (PTH) and vitamin 1,25 dihydroxyvitamin D_3 (1,25-$(OH)_2D_3$), the active metabolite of Vitamin D. The increased demand for calcium is satisfied by increased resorption of calcium by the kidneys, increased absorption of calcium from the gastrointestinal tract, and increased mobilization of calcium reserves from bone.

When plasma calcium levels decrease, PTH is secreted by the parathyroid glands and immediately causes the kidneys to conserve calcium. If plasma calcium levels are not sufficiently restored, PTH will continue to be secreted. The PTH stimulates bone reabsorption of calcium and the conversion of 25-hydroxyvitamin D_3 to 1,25-$(OH)_2D_3$ in the kidney. 1,25-$(OH)_2D_3$ stimulates bone reabsorption of calcium and active transport of calcium from the intestine. During the dry period, calcium demands can be entirely satisfied by passive absorption of calcium by the gastrointestinal tract, so calcium homeostatic mechanisms would be inactive.

Feeding diets low in calcium (20-40 grams/day) during the dry period prevents parturient paresis because the cow is in a negative calcium balance prior to parturition, which stimulates secretion of PTH and production of 1,25-$(OH)_2D_3$. At parturition, the cow is able to efficiently absorb calcium from the intestine and mobilize calcium from the bone.

How does feeding anionic salts affect calcium homeostasis?

The mechanism by which anionic salts affect calcium homeostasis is not known. Diets high in anionic salts cause metabolic acidosis (Freeden, *et al*, 1988) by mechanisms previously described or at least reduce the alkalinity of the diet. Because blood pH is maintained within narrow limits by the body, the impact of the DCAD might not be reflected in measurements of blood pH and would be expected to occur at the cellular level (Block, 1994).

Cows fed anionic diets consistently have greater calcium loss in the urine compared to control cows (Gaynor *et al*, 1989, Freeden *et al*, 1988, Oetzel *et al* 1991, Phillipo *et al* 1994, Van Mosel *et al*, 1993). The origin of the increased calcium loss in the urine is not known. The hypercalciuria could result from reduced renal calcium reabsorption or an increase in the filtered load of calcium either via increased mobilization of calcium from bone or increased absorption of calcium from the intestine. Stacy *et al*, (1970) in studies with sheep showed that the hypercalcuria was due to a direct effect of acidosis on renal calcium reabsorption. The increased excretion of calcium could be sufficient to stimulate the calcium homeostatic mechanisms increasing PTH and 1,25-$(OH)_2D_3$ before parturition which would in turn activate bone reabsorption and intestinal absorption of calcium. Gaynor *et al* (1989) and Goff (1991) suggest that during metabolic alkalosis the kidneys and bone tissue might be refractory to stimulation by PTH, and that anionic diets could work to increase target tissue responsiveness to PTH.

How do you monitor anionic diets?

Anionic salts can decrease dry matter intake (DMI) when greater than 300 meq anion/kg are added to the diet (Goff, 1992). Decreases in DMI before calving should be avoided and are associated with a greater occurrence of postpartum diseases (Zamet *et al*, 1979) and high plasma non-esterified fatty acid concentrations (Grummer, 1995). DMI can drop 30-35 percent before parturition and there is a strong positive correlation between DMI before and after calving (Grummer, 1995). Therefore, DMI should be monitored in the close-up dry cows and maintained at a minimum of 22 lbs DM (Byers, 1994) and the incidence of postpartum diseases should be monitored.

Jardon (1995) suggests using urinary pH to monitor anionic diets. The major mechanism by which the kidneys affect plasma [H+] is by adjusting the [SID] from the plasma to the urine (Stewart, 1981). The kidneys lower the $[H^+]$ of plasma by excreting more Cl^- than Na^+, thereby raising the plasma [SID]. When Cl^- is excreted into the urine, either H^+ must accompany it, or OH^-, $HCO3^-$, or $CO3^{--}$ must move in the opposite direction. As Cl^- is excreted into the urine, the [SID] of the urine decreases and this causes the urine $[H^+]$ to rise, resulting in a low urine pH. Manipulation of Cl^- is the major mechanism by which the kidneys affect [SID] because Na^+ is also involved in extracellular fluid volume regulation (Stewart, 1981).

The kidneys excrete a wide variety of substances that have acid-base properties that are only important in the urine so caution should be taken to avoid over

interpretation of urine pH (Jardon 1995). However, failure to acidify the urine might suggest that sufficient acidosis has not occurred and this could potentially increase the risk of parturient paresis depending on the level of calcium in the diet. Jardon (1995) suggests titrating the rations with anionic salts to a pH value of 6-7 to prevent parturient paresis. Oetzel *et al* (1991) evaluated six anionic salts ($MgSO_4$, $MgCl_2$, $CaCl_2$, $CaSO_4$, NH_4Cl, $(NH_4)_2SO_4$) for their effects on acid-base status and urinary calcium excretion. All six anionic salts significantly increased urinary calcium excretion compared to the control diet. However, the salts had varying effects on urinary pH. The correlation between sufficient systemic acidosis and urinary pH levels has not been determined.

How do you formulate prepartum anionic diets?

The following guidelines are suggested in the literature for formulating prepartum diets using anionic salts:

1. Analyze feed samples for chloride and sulfur in addition to the standard macrominerals calcium, magnesium, phosphorous, and potassium. The effects of water on the DCAD have not been evaluated.
2. Remove cations such as sodium bicarbonate and use feeds low in potassium. Diets requiring the addition of greater than 300 meq anion/kg could negatively affect feed intake (Goff, 1992). The effect of calcium carbonate on DCAD has not been studied. Calcium carbonate could have an alkalizing effect suppressing calcium homeostasis because Ca^{++} is a strong ion and its accompanying anion is weak.
3. Oetzel *et al* (1991) recommends using a mixture of anionic salts ($MgSO_4$, $MgCl_2$, $CaCl_2$, $CaSO_4$, NH_4Cl, $(NH_4)_2SO_4$) to reduce toxicity due to excessive NPN, SO_4, or Mg. Sniffen *et al* (1996) recommends balancing sulfur and magnesium at .36-.4% of DM and phosphorous at 30-60 grams.
4. Bio-chlor[1] can be used to formulate anionic diets. Bio-chlor is a nutrient enriched source of anionic salts high in chloride (8.6% Cl) that has been shown to stimulate rumen function and, potentially, dry matter intake. The recommended inclusion rate is 2 - 2.5 lbs/day. Field experience suggests that Bio-chlor is palatable and safe.
5. The suggested target DCAD level is -100 meq/kg DM (Goff, 1992) in prepartum close-up dry cow rations, but more research is needed to establish the optimal DCAD in prepartum dairy rations.
6. The optimum level of calcium to feed with anionic diets has not been established. Jardon *et al* (1994) suggests the optimum level of calcium may be greater than 150 grams per day. Most trials reported in the literature used high calcium levels.
7. Byers (1994) recommends feeding a total mixed ration to minimize feed selection by cows and to maintain a DMI of at least 22 lbs in the close up dry cows.
8. Cows should be fed anionic salts for at least 2-3 weeks prior to parturition (Gaynor *et al*, 1989).

[1] *Biovance*

Summary

The literature supports the concept that the DCAD should be routinely calculated during formulation of dry cow prepartum rations. A potential benefit of incorporating anionic salts into the diet would be the ability to feed high calcium feedstuffs such as alfalfa to the prepartum dry cow if so desired. However, feeding anionic diets to prepartum dairy cows in the field is challenging because the mechanism of action have not been clearly defined. Until further clarification, dry matter intake and urinary pH should be routinely monitored on prepartum dry cows consuming anionic diets.

References

Beede DK, Risco CA, Donovan GA, Wang C, Archbald LF, Sanchez WK. 1992. Nutritional management of the late pregnant dry cow with particular reference to dietary cation anion difference and calcium supplementation. *Proceedings* of the 24th annual convention of the AABP, pg 51. Block E. 1984. Manipulating dietary anions and cations for prepartum cows to reduce incidence of milk fever. *J. Dairy Sci.* 67:2939. Byers D. 1994. Management considerations for successful use of anionic salts in dry-cow diets. *Compend. on Cont. Edu.* February:237. Dishington IW. 1975. Prevention of milk fever by dietary salt supplementation. *Acta. Vet. Scand.* 16:503. Freeden AH, DePeters EJ, Baldwin RL. 1988. Characterization of acid-base disturbances and effects on calcium and phosphorus balances of dietary fixed ions in pregnant or lactating does. *J. Anim. Sci.* 66:159. Gaynor PJ, Mueller FJ, Miller JK, Ramsey N, Goff JP, Horst RL. 1989. Parturient hypocalcemia in Jersey cows fed alfalfa haylage-based diets with different cation to anion ratios. *J Dairy Sci.* 72:2525. Goff JP. 1992. Cation-anion difference of diets and its influence on milk level and subsequent lactation: The good and the bad news. *Proc Cornell Nutr Conf*:35. Goff JP, Horst RL, Mueller FJ, Miller JK, Kiess GA, Dowlen HH. 1991. Addition of Chloride to a prepartal diet high in cations increases 1,25-Dihydroxyvitamin D response to hypocalcemia preventing milk fever. *J Dairy Sci* 74:3863. Grummer R. 1995. Impact of changes in organic nutrient metabolism on feeding the transition dairy cow. *J. Animal Sci.* 73:2820. Jardon PW, 1995. Using Urine pH to monitor anionic salt programs. *Compend.* on Cont. Edu. Vol 17(6):860. Jardon PW, Weaver LD, Holmber CA. 1994. Effect of calcium level on hypocalcemia and associated conditions in dairy cattle fed acidic diets in the late dry period. *J. Dairy Sci.* vol 77, Suppl. 1:133. Oetzel GR, Fettman MJ, Hamar DW, Olson JD. 1991. Screening of anionic salts for palatability, effects on acid-base status and urinary calcium excretion in dairy cows. *J Dairy Sci* 74:965. Oetzel GR, Olson JD, Curtis CR, Fettman MJ. 1988. Ammonium chloride and ammonium sulfate for prevention of parturient paresis in dairy cows. *J Dairy Sci* 71:3302. Phillipo M, Reid GW, Nevison IM. 1994. Parturient hypocalcemia in dairy cows: Effects of dietary acidity on

plasma mineral and calciotropic hormones. *Res Vet Sci* 56:303. Sniffen CJ, Thomas ED. 1996. Feeding and managing the transition cows: Metabolic basis of the cation anion difference concept. *Proceedings of the Penn Annual Conference.* Stacy BD, Wilson BW. 1970. Acidosis and hypercalciuria: Renal mechanisms affecting calcium, magnesium and sodium excretion in the sheep. *J. Physiol.* 210:549. Stewart, PA. 1981. How to understand acid-base balance. A quantitative acid-base primer for biology and medicine. New York, Elsevier. Stewart, PA. 1983. Modern quantitative acid-base chemistry. *Can. J. Physiol. Pharmacol.* 61:1444. Van Mosel M, Van't Klooser A, Van Mosel F, Van Der Kuilen J. 1993. Effects of reducing dietary [($Na^+ + K^+$)-($Cl^- + SO4^{--}$)] on the rate of calcium mobilization by dairy cows at parturition. Research in *Veterinary Science* 54:1. Zamet CN, Colenbrander VF, Callahan CJ, Chew BP, Erb RE, Moeller NJ. 1979. Variables associated with peripartum traits in dairy cows. Effect of dietary forages and disorders on voluntary intake of feed, body weight and milk yield. *Theriogenology* 11:229.

Abstract

The dispersal of *Culicoides brevitarsis* in eastern New South Wales and associations with the occurrences of arbovirus infections in cattle

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Distributions of the vector *Culicoides brevitarsis* Kieffer (Diptera: Ceratopogonidae) (determined from light trap data) and 2 arboviruses (determined from seroconversions in sentinel cattle) were studied in eastern New South Wales in 1993-94. *C brevitarsis* was recorded progressively from endemic areas on the north coast, to Nowra on the south coast, and westward to Scone, in the Hunter Valley. *C brevitarsis* also survived through winter at Paterson, in the Hunter Valley. Its apparently focal reappearance in this marginal area had no obvious effect on the broad pattern of its progression or the dispersal of Akabane and bluetongue viruses. These viruses were first recorded from foci near Coffs Harbour, on the mid-north coast. Their first occurrences at different locations were associated with those of *C brevitarsis*, but not with each other. The viruses were found only within the recorded limits of the vector's distribution. Delays between the initial occurrence of *C brevitarsis* and first evidence of virus transmissions at locations ranged from 2 to 7 months. The delays decreased away from the points of focus and were negatively associated with the time of initial occurrence of the vector. Seroconversions to the virus were related to the presence of *C brevitarsis*. However, the densities of *C brevitarsis* had no apparent effect on the initial numbers of cattle seroconverting to either viruses. The results support the conclusion that the progressions of *C brevitarsis* and Akabane and bluetongue viruses were the result of gradual movements by the vector.