Nutritional Management of the Late Pregnant Dry Cow with Particular Reference to Dietary Cation-Anion Difference and Calcium Supplementation

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Introduction

All too often the dry pregnant cow is the most neglected creature on the dairy because she is not contributing to current cash flow and profit. It is commonly accepted that the milk yield peak the cow achieves in early lactation, to a large extent, influences her total lactational yield. Proper management of the late pregnant dry cow period should be portrayed to dairy producers as an investment in the future; an investment targeted towards achievement of maximal lactational and reproductive performance in the next lactation. Nutritional management strategies should be implemented during the last 3 to 4 weeks of pregnancy that can help ensure a high return on that investment postpartum. In recent years one such potential strategy, incorporation of the cation-anion difference concept into formulation of diets for cows in late gestation, has been reported and application is occurring in some commercial dairies.

Basics of Dietary Cation-Anion Difference

Dietary cation-anion difference (DCAD), also termed dietary electrolyte balance, cation-anion or anion-cation balance, alkali-alkalinity, or strong ion difference is a relatively new concept in dairy cattle nutrition and diet formulation. Certain physiological and productive functions of dairy cattle may be influenced by DCAD. In its most complete form DCAD is expressed in milliequivalents (meq) as: meq [(Na + K + Ca + Mg) - (Cl + SO_4 + H_2PO_4 + HPO_4)]/100g dry matter (DM). Sparse information about the bioavailability (absorbability) and physiological roles of Ca, Mg, SO_4, H_2PO_4 and HPO_4 have limited their inclusion in the functional expression. The most commonly used expressions are: meq [(Na + K) - Cl]/100g or meq (Na + K) - (Cl + S)/100g. Each of these mineral ions is known as a fixed ion, because it cannot be metabolized to a more rudimentary chemical level. To calculate DCAD in meq/100g of diet dry matter (DM) the equation is: [(%Na in diet DM / 0.023) + (%K in diet DM / 0.039)] - [(%Cl in diet DM / 0.035) + (%S in diet DM / 0.016)]

The major effect of DCAD is on the systemic acid-base status of the cow. When DCAD is a negative value (more meq of anions relative to cations) a mild metabolic acidosis may be produced, whereas with a positive DCAD value metabolic alkalosis may result. The mechanism for these effects is related to the physiological need to maintain electrical neutrality of the body. To maintain neutral ionic charges, consequent to the input of fixed anions, systemic hydrogen ion concentration [H^+] is increased. In contrast, if fixed cations are introduced an elevation in HCO_3^- results. Feeding a diet with a negative DCAD (anionic diet) in late pregnancy results in an inflow of negatively charged ions (Cl^-, $SO_4^=$) systemically. Consequently, positively charged ions [eg., H^+] are generated to neutralize the influx of anions. Acidosis ensues. Several compensatory mechanisms are triggered to annul alterations in blood pH. In chronic subclinical cases, bone tissue serves as an important reserve of $CO_3^=$ which buffers and corrects acidosis. In the process, bone Ca [and phosphorus] is mobilized and absorption of Ca from the gut is enhanced (Fredeen *et al.,* 1988). This sequence of events proceeds continuously and recurs as long as the cow continues to consume the anionic diet. Thus, higher blood Ca concentrations are maintained and the metabolic machinery to increase blood Ca is readily functional. This situation can be immensely helpful to the cow in the periparturient period when there is a sudden increased demand for Ca. Other related consequences on metabolic and production performance responses have been reported and suggested; these

will be presented subsequently in this paper.

Potential sources of supplemental anionic salts used to build a negative DCAD include: aluminum sulfate [$Al_2(SO_4)_3$], calcium chloride [$CaCl_2 \cdot 2H_2O$], calcium sulfate [$CaSO_4 \cdot 2H_2O$], magnesium chloride [$MgCl_2 \cdot 6H_2O$], magnesium sulfate [$MgSO_4 \cdot 7H_2O$], ammonium chloride [NH_4Cl], and ammonium sulfate [$(NH_4)_2SO_4$]. Generally, feed grade sources of $CaSO_4 \cdot 2H_2O$ (gypsum), $MgSO_4 \cdot 7H_2O$ (epsom salts), NH_4Cl, and $(NH_4)_2SO_4$ are more readily available in feed mills and less expensive; however, this may vary with location.

Association of Calcium Status and Other Periparturient Metabolic Problems

The most commonly recognized and definable metabolic disease associated with low blood Ca is clinical parturient paresis or milk fever. However, the possible relationships or associations of other metabolic disorders (eg., ruminal stasis, displaced abomasum, retained placenta, prolapsed uterus, early metritis and ketosis) with hypocalcemia (many times subclinical) have been suspected and evaluated by practitioners and researchers (Curtis *et al.,* 1983).

Organs (eg., uterus, rumen and abomasum) which normally have significant smooth muscle function have problems with hypocalcemia. Grohn *et al.* (1990) reported an epidemiological study of reproductive disorders of over 61,000 Finnish Ayrshire cows and the associations among host characteristics, disease and production. Clinical parturient paresis was found to be a significant risk factor for dystocia, prolapsed uterus, retained placenta and early metritis. Also, abomasal disorders were associated significantly with early metritis.

Risco *et al.* (1984) showed that cows with prolapsed uteri had a higher incidence of severe hypocalcemia (serum total Ca $\leq$ 4 mg/ dl) than herd mate controls which did not prolapse (19 vs. 2%). Additionally, older cows (44 second and greater parity cows) had significantly lower total serum Ca concentrations in samples taken within 24 h of calving if they had prolapsed uteri compared with herd mate controls (5.85 vs. 6.95 mg/ dl). There was no difference in serum Ca concentrations between 9 pairs of first parity cows which had prolapsed and their control counterparts.

The incidence of retained fetal membranes was reduced from 25% to 0% when multiparous cows were fed anionic mineral salts, increasing blood ionized Ca, compared with cows fed a more traditional dry cow ration without the anionic salts (Oetzel *et al.,* 1988). Hypocalcemia is also thought to be associated with slowed uterine involution postpartum.

Hypocalcemia has been implicated in association with displaced abomasum and stasis of the reticulo-rumen. Hull and Wass (1973) reported that of 90 cows sampled in three Iowa herds, eight cows had abomasal displacement and seven of these had abnormally low blood Ca concentrations preceding displacement. Abomasal displacement also occurred in seven of 26 cows with low blood Ca concentrations postpartum. There was only one abomasal displacement among the 64 cows which had normal blood Ca concentrations after calving.

The study of Huber *et al.* (1981) provided direct evidence of the effect of hypocalcemia on the motility of the ruminant stomach. Sheep were surgically-fitted with pressure transducers in the rumen and abomasum. The potent Ca chelator sodium EDTA was infused intravenously over time, binding up the diffusible blood Ca. As the infusion of EDTA progressed the amplitude of ruminal contractions decreased dramatically. Major conclusions of the study were that: 1) ruminal contractions ceased long before signs of hypocalcemia were observed, 2) ruminal dysfunction may occur substantially before the clinical signs of hypocalcemia, and 3) abomasal contractions were somewhat reduced at the serum diffusible Ca concentrations causing ruminal stasis and clinical signs of hypocalcemia. This study demonstrated a true cause and effect relationship between hypocalcemia and normal smooth muscle contractility of the ruminant stomachs.

This brief review of the effects of hypocalcemia on organs possessing smooth muscles is by no means exhaustive. However, it is apparent that associations (and cause and effect relationships) among hypocalcemia and various disorders of these organs can exist. It is suggested that these disorders may have major consequences on the health and productivity of the postpartum cow. It may be that they are associated with "droopy cow" syndrome sometimes observed in the early postpartum period; even with cows which did not show clinical signs of milk fever. This syndrome is often characterized by lethargy and inappetence; factors which can cause poor feed intake and milk production in the early postpartum period. Potentially, this may have greater negative impact on productivity and on economic consequences to the dairyman than a normally low, but real, incidence of clinical parturient paresis.

Prepartal Dietary Calcium Restriction and Milk Fever

The most practiced method of controlling the incidence of milk fever is to restrict intake of Ca during late gestation. The NRC (1989) suggests Ca intake of 33 to 42 g/d for mature dry cows (1200 to 1500 lb body weight) during the last 2 mo of gestation.

Review of some of the literature suggests that restriction of Ca intake in the late prepartum period was not efficacious always for preventing milk fever (Josson *et al.,* 1978; Kendall *et al.,* 1970). Generally, only when Ca intake was restricted to less than 20 g/d could prevention be certain (Boda and Cole, 1954; Boda, 1956; Wiggers, 1975).

Often times it is very difficult to formulate diets from available feedstuffs which contain low Ca (about 0.39%, according to NRC, 1989) to achieve the prescribed rate of

Ca intake; many forages typically are quite high in Ca content. Additionally, when not enough attention is given to amount of feed offered and number of cows being fed in a group, total ration and Ca intakes may be greater than prescribed; this may predispose cows to problems.

Recently, Oetzel (1991) reported results of a meta-analysis which evaluated possible nutritional risk factors for milk fever. Data from a large number of trials from the literature were evaluated. In the analysis dietary Ca content was found to be related to the incidence of milk fever. This relationship was curvilinear, with a low (less than 2%) incidence of milk fever being predicted with low Ca (less than 0.5% dietary Ca) or very high Ca (greater than 2.0% dietary Ca). However, greater incidence rates were predicted with dietary Ca between these two bounds; up to 10% at about 1.2% dietary Ca. Interestingly, dietary sulfur (S) content, ranging from 0.10 to almost 0.55%, had a much more profound effect on the incidence of milk fever. With low dietary S the incidence was about 60% whereas with 0.55% S the incidence was very near 0%. Sulfur, of course, is a dietary anion which may affect the cow's acid-base balance and Ca metabolism to reduce the incidence of milk fever and hypocalcemia, as previously suggested.

Previous Research on Negative DCAD in Late Pregnancy

In some management systems it might be of great practical advantage to reduce DCAD during late gestation to assist in the prevention of milk fever and other related problems.

Norwegian studies (Ender *et al.* 1962; 1971) first showed that grass silage preserved with mineral acids (H_2SO_4 and HCl) prevented milk fever, whereas sugar beets with high Na and K contents caused milk fever. Later Dishington (1975) showed that diets with negative DCAD [from supplementation of a mixture of $CaCl_2 \cdot H_2O$, $MgSO_4 \cdot 7H_2O$, and $Al_2(SO_4)3$] also prevented milk fever. This was confirmed by (Dishington and Bjornstad, 1982).

Block (1984) tested this approach using 19 cows each receiving a basal diet with DCAD's of +33 meq (cationic diet) or -13 meq/100g (anionic diet) in a 2 yr switchback design. None of the cows consuming the anionic diet for 5 wk prepartum had milk fever, whereas an incidence of 47.4% (9 of 19) was observed for cows consuming the cationic diet. Subsequently, cows fed the anionic diet prepartum had higher (7% increase) 305-d milk yields than cows fed the cationic diet. Also, within the cationic diet group, cows which did not have clinical milk fever had 16% higher milk yields than those that had milk fever. Blood plasma Ca and hydroxyproline concentrations, an indicator of bone tissue mobilization, were greater for cows fed the anionic than cationic diet, suggesting that the dietary manipulation enhanced mobilization of Ca from bone, thus making it available at the time of high metabolic demand around parturition.

More recently, Oetzel *et al.* (1988) reported results of a trial in which 12 cows each were fed dietary treatments containing -7.5 or +18.9 meq/100g with either 53 or 105g Ca/d for 3 wk prepartum. The anionic diet was made by supplementing 100g each of NH_4Cl and NH_4SO_4 /cow/d. The incidence of milk fever was 4% in cows fed the anionic diet, but 17% without the addition of the ammonium salts. Cows fed the anionic diet had higher total and ionized Ca (iCa) in blood at parturition. Using risk analysis, it was found that when Ca intake was high, supplementation with ammonium salts reduced the risk of hypocalcemia ten-fold.

With 18 mature Jersey cows fed a diet containing over 90% alfalfa haylage, Gaynor *et al.* (1989) used additions of chloride salts of Mg, NH_4, and Ca to make a DCAD of +22 meq/100g diet and compared it with +66 and +126 meq/100g diets having added alkalogenic salts (eg., $NaHCO_3$) for 6 wk prepartum. Though still having a positive DCAD, the +22 meq diet tended to result in less clinical milk fever and higher blood total Ca within 36 h of parturition. Plasma concentrations of 1, 25 di-OH vitamin D were increased with +22 meq/100g compared with the other two treatments.

Recent Experiment with Negative DCAD and High Dietary Calcium

In each of the previous studies, under controlled experimental conditions, the number of cows per treatment was relatively small. Additionally, in some studies the cationic treatments were made with very high positive DCAD, seemingly to increase chances of detecting differences in response compared with the negative DCAD.

In 1990 a large field experiment was undertaken in a commercial dairy to compare the effects of feeding an anionic (acidogenic) diet compared with a control diet formulated as close as possible to NRC (1989) recommendations, for dry cows during the last 2 mo of pregnancy. The anionic diet was -25 meq/100g DM by supplementation of 108g of NH_4Cl, 53g of $(NH_4)_2SO_4$ and 34g of $MgSO_4 \cdot 7H_2O$/cow/d (Treatment). The Treatment and Control (+5 meq/100g) diets were composed of the same basal ingredients, but the Treatment diet contained more Ca (1.81 vs 0.92%), higher Cl (1.06 vs 0.55%) and higher S (0.44 vs 0.30%) from the mineral supplement; Na and K concentrations were similar. From December 24, 1989 through April 30, 1990, 510 dry cows were group-fed their respective treatments in late pregnancy (3 to 5 wk prepartum). Blood samples were drawn within 18 h of calving and prior to treatment of all clinical milk fever cases.

Concentrations of Blood Serum Ca and P, and Incidences of Milk Fever and Hypocalcemia.

Results of this experiment showed that blood serum concentrations of iCa (4.31 vs 3.80 mg/dl) and total serum Ca (7.94 vs 7.10 mg/dl) were higher for cows fed the Treatment (anionic diet) than Control diet (Table 1, $P < .01$). Concentrations of serum phosphorus also were higher for

Table 1. Experimental results from a recent experiment in which Holstein cows were fed diets with negative (Treatment) or positive (Control) cation-anion difference.[a]

Measurement	Treatment - 25 meq/100g	Control + 5 meq/100	P <
Clinical milk fever incidence, %			
≤ 2 lactations	0	0	NS[b]
≥ 3 lactations	5	12	.01
All cows	4	9	.01
Subclinical hypocalcemia incidence, %[c]			
≤ 2 lactations	2	16	.01
≥ 3 lactations	28	66	.01
All cows	19	50	.01
	------- All Cows -------		
Serum iCa, mg/dl	4.31	3.80	.01
Serum Ca, mg/dl	7.94	7.10	.01
Serum P mg/dl	4.44	3.64	.01
Milk yield, lbs [305-d ME]	20,627	19,908	.01
Pregnancy rate, %			
@ 100 d postpartum	35	28	.26
@ 150 d postpartum	55	42	.03
@ 200 d postpartum	71	54	.01
@ 250 d postpartum	77	66	.06
Services/Pregnancy	3.0	3.4	.16
Ave. days to first heat	68	72	.22
Ave. days open (for pregnant cows)	124	138	.10

[a] Cows calving from December 24, 1989 through April 30, 1990 were divided randomly into two groups (260 cows on Treatment and 250 cow on Control) and were fed the experimental diets for 3 to 5 wk prepartum. After calving cows were co-mingled and fed the same lactation ration. Lactational and reproductive performance data presented are through December, 1990.

[b] NS = Not a significant effect of dietary treatment.

[c] Blood serum ionized calcium (iCa) concentrations ≤ 4.0 mg/dl defined a cow as being hypocalcemic.

Treatment than Control cows (4.44 vs 3.64 mg/dl, P < .01). In addition, the incidence of clinical milk fever (cows diagnosed and treated for milk fever) by treatment group and parity were: Treatment diet, ≤ 2 parities = 0%, and ≥ 3 parities = 5%; Control diet, ≤2 parities = 0%, and ≥ 3 parities = 12% (treatment and parity effects, P < .01). The incidence of hypocalcemia (iCa ≤ 4 mg/dl) was: Treatment diet, ≤2 parities = 2%, and ≥3 parities = 28%; Control diet, ≤ 2 parities = 16%, and ≥ 3 parities = 66% (treatment and parity effects, P < .01). There were no differences between treatment groups in body condition or umbilical-udder edema scores taken 1 to 2 wk prepartum. The incidences of retained placenta, dystocia, displaced abomasum, metritis (at the first postpartum evaluation) or clinical ketosis were not affected by treatments, based on evaluation of farm health records. However, only 10 of the 510 cows in the study were diagnosed as having displaced abomasum. When data from both treatments were pooled to test for the likelihood of a cow having a displaced abomasum if she was hypocalcemic, cows with serum iCa concentrations of less than or equal to 4 mg/dl) were 3.49 times more likely to have a displacement than cows with iCa greater than 4 mg/dl (P < .02). If serum total Ca of less than or equal to 8 mg/ dl was used to define hypocalcemia, cows which were hypocalcemic were 7.35 times more likely to have abomasal displacement than cows with serum total Ca greater than 8 mg/dl (P < .004).

Lactational and Reproductive Performance.

Milk production data were from the regular DHI tests (10 test months) and data for reproductive performance were from the dairy's regular herd health records. Milk yield during the next lactation (305-d ME) was 3.61% greater (20,627 vs 19,908 lbs, P < .01) for cows fed the Treatment (anionic diet) during the late prepartum period compared with cows fed Control. All cows were co-mingled and fed the same diet during their lactation.

Reproductive performance after calving also was improved for cows fed the Treatment compared with Control diet (Table 1). Pregnancy rates at 150, 200, and 250 d after calving were 11 to 17 percentage units higher for cows receiving the Treatment diet compared with the Control. Number of services per pregnancy (P < .16) and average days from calving to confirmed pregnancy (P < .10) tended to be reduced for cows fed the Treatment compared with the Control diet. There was also a dietary treatment by parity of cow interaction on pregnancy rate at 200 d postpartum (P < .05). As parity increased, pregnancy rates of cows fed the Treatment remained over 70%, whereas pregnancy rates declined linearly from about 65% for first parity cows to about 35% for cows of fifth parity or greater when fed the Control treatment.

Summary and Discussion

Results of this experiment indicated that incorporating anionic (acidogenic) salts into the ration of cows during the last 3 to 5 wks before calving made it much easier for the cow to cope with the high metabolic demand for Ca during early lactation. Reduced incidences of clinical milk fever and subclinical hypocalcemia occurred with the feeding of the acidogenic diet. This occurred even though the diet of cows fed the negative DCAD contained 1.81% Ca, dry basis (compared with 0.92% Ca in Control). This works out to an estimated Ca intake of 181 g/cow/day if dry matter intake is estimated at 22 lbs/day for a mature (1400 lb BW) Holstein cow. Although this high dietary concentration and rate of Ca is not typically recommended for late pregnant dry cows, it was used in this experiment to test and challenge the effectiveness of the negative DCAD formulation. Observations and conversations from the field have indicated no difficulties of feeding 150 to 200 g Ca/cow/day if the anionic (acidogenic) agents also are supplemented properly.

Positive effects of the negative DCAD treatment on subsequent lactational and reproductive performance were economically important. For example, the additional milk income realized during the full lactation amounted to about $52 per cow (719 lbs extra milk x $0.145 per lb milk) x 0.5 [if 50% of the value of the extra milk was expended as feed cost]. The cost of the additional anionic salts for one cow during the month before freshening will be about $5. The improvement in milk yield alone potentially can return about $10 for each $1 invested. Of course, this does not

include any estimate of the potential economic advantage because the incidence of milk fever was less and reproductive performance was significantly improved.

It appears that we now have a reasonably practical way, through manipulation of the dietary cation-anion difference in the late prepartum ration, to reduce the incidence of hypocalcemic-related problems in the early postpartum cow, and improve reproductive and lactational performance. Readers are urged to refer to the detailed description presented by Byers (1991) in the Practice Tips section of these proceedings on how to formulate anionic dry cow rations.

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Abstract

Transmission of salmonellae among calves penned individually

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Veterinary Record (1991) **129**, 327-329

An analysis of the spatial and temporal patterns of excretion of salmonellae by calves penned individually showed that non-contagious routes were more important than contagious routes in disease spread. The avoidance of aerosol production, and the effective cleaning and disinfection of utensils between feeds and of buildings between batches, are likely to be more important than pen design in the control and prevention of calf salmonellosis.