Improving Reproductive Performance in Dairy Cattle Via Milk Fever Prevention

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Relationship of Milk Fever to Reproduction

Milk fever (parturient paresis) is often not considered a major determinant of reproductive performance in dairy herds. Dairy producers in particular may not see the connection between a metabolic disease problem that occurs around calving time and impaired fertility that occurs several months later. However, both direct and indirect links between episodes of milk fever and impaired reproductive performance in later lactation have been established.

The first evidence for a role of milk fever in determining reproductive performance comes from epidemiological evidence which shows increased risk of other periparturient disorders in cows with clinical milk fever. A large study of 7761 lactation records from 34 commercial dairy herds[1] showed that cows with clinical milk fever had 2.6 times greater risk of dystocia, 2.4 times greater risk of ketosis, and 2.3 times greater risk of left-displaced abomasum. Dystocia was then linked to 2.2 times greater risk of retained placenta and 2.1 times greater risk of metritis. If retained placenta was present, then the risk of metritis increased 6.0 times. An earlier analysis of a subset of this data[2] showed that clinical milk fever directly increased the risk of retained fetal membranes 3.2 times and the risk of metritis 1.7 times.

Milk fever has also been associated with uterine prolapse; 53 cows with uterine prolapse had significantly lower serum calcium concentrations than 53 matched cows.[3] Multiparous cows with uterine prolapse were more likely to be hypocalcemic than primiparous cows with uterine prolapse. Delayed cervical and uterine involution during hypocalcemia may explain why hypocalcemia is associated with uterine prolapse.[3,4]

Milk fever has also been qualitatively linked with increased incidence of cystic ovarian disease.[5] No direct mechanism for this link has been established. Cystic ovarian disease may be associated with any episode of periparturient disease.

The effects of periparturient diseases on reproductive performance have been reviewed.[6] Dystocia, retained placenta, cystic ovaries, metritis, and any periparturient health problem were found to either directly or indirectly decrease reproductive performance. Because milk fever increases the relative risk of these important periparturient diseases, it indirectly, but predictably, decreases fertility.

The theoretical link between milk fever and decreased reproductive performance, which is based on data from retrospective studies, has been substantiated in a large, prospective field study (Table 1).[7] Cows fed anionic salts in this study had less clinical milk fever and subclinical hypocalcemia than cows not fed anionic salts. Cows fed the anionic salts also had higher pregnancy rates, lower services per pregnancy, reduced days to first heat, and reduced days open for pregnant cows. The positive results seen in this study are likely the net effect of the prevention of periparturient diseases that are associated with both clinical milk fever and reduced reproductive performance. An additional mechanism for explaining this effect could be increased dry matter intake in early lactation in the cows that did not get either clinical or subclinical milk fever. This mechanism has not been evaluated in a research study, but there is indirect evidence to support it. Subclinical hypocalcemia was present in 50% of the control cows in this study, but only 19% of the cows receiving the anionic salts were classified as having subclinical hypocalcemia. Calcium is necessary for smooth muscle contractions in the body, including the gastro-intestinal tract.[8] Impaired motility of the gastro-intestinal tract in early lactation may impair dry matter intake during this critical period. One indication that postpartum dry matter intake depression may occur with hypocalcemia

is the observed overall higher milk production in cows that receive anionic salts prior to calving.[7,9] If hypocalcemia does impair dry matter intake in early lactation, then negative energy balance will be exacerbated and reproductive performance will be correspondingly impaired.

Table 1. Effect of feeding anionic salts on the incidence of clinical milk fever, subclinical hypocalcemia, and reproductive performance in Holstein cows. Adapted from Beede *et al.*[7]

Measurement	Treatment Diet (with anionic salts)	Control Diet (without anionic salts)	$P <$
Clinical milk fever incidence, %			
≤ 2 lactations	0	0	NS
≥ 3 lactations	5	12	.01
All cows	4	9	.01
Subclinical hypocalcemia incidence, %			
≤ 2 lactations	2	16	.01
≥ 3 lactations	28	66	.01
All cows	19	50	.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
Milk yield, lbs (305-d ME)	20,627	19,908	.01

Methods of Preventing Milk Fever

Prevention, rather than treatment, of milk fever is imperative in dairy herds. Not only do cows that suffer from clinical milk fever have impaired reproductive performance, but they also produce about 14% less milk in the subsequent lactation.[9] Most cows with clinical milk fever respond well to a single treatment with an intravenous solution containing a calcium salt; however, about 25% of cases will relapse and require additional treatment.[10] More alarmingly, 15% of all cows affected with clinical milk fever and presented in sternal recumbency will either die or require disposal.[11]

Dietary Calcium Restriction During the Dry Period

The traditional method of preventing milk fever has been to restrict calcium intake during the dry period.[12] If extremely low calcium diets (< 20 grams of daily calcium) are fed before parturition and high-calcium diets are fed after parturition, the incidence of milk fever can be drastically reduced.[13] Low calcium diets prior to calving apparently prevent the cow's active intestinal calcium absorption and bone calcium resorption mechanisms from becoming quiescent and unable to respond to the sudden calcium outflow that occurs at parturition.[14] Calcium intake during the dry period is usually restricted by replacing some or all of the alfalfa in a dry cow diet with grass hay and using additional corn silage and concentrates. This approach can work in some dairy herds. If milk fever (clinical and subclinical) is not a problem in a herd and this feeding program is being used, then it probably should not be changed.

Switching feed ingredients in the dry period diet with the exclusive goal of lowering calcium intake does not work well in some dairy herds and can have several drawbacks.[15] Feeding larger amounts of corn silage and/or concentrates to dry cows may be expensive and may predispose cows to abomasal displacements if fed in excess.[16] Eliminating alfalfa from the dry period diet simply because of its high calcium content can be an expensive decision. On many dairies, alfalfa is the most readily available forage and is also an inexpensive source of dietary protein. In these herds, alfalfa is likely to be the primary forage in the lactating cow diets, and there are advantages to maintaining similar forage type throughout the peripartum period.[17]

Dietary Acidification in the Dry Period

Introduction to Dietary Acidification

Dietary acidity or alkalinity is more important in controlling milk fever than calcium intake.[7,9,18,19] Alkalogenic diets fed prior to calving tend to cause milk fever, while acidogenic diets tend to prevent it. A large meta-analysis of previously published milk fever studies showed that dietary calcium influences the incidence of milk fever; however, it does so in a limited and non-linear fashion (see Figure 1). Both high and low dietary calcium were associated with slightly lower incidence rates of milk fever in this analysis.[18] However, dietary sulfur, a strong anion and dietary acidifier, was the most important single dietary measure related to milk fever.[18] Diets high in sulfur were associated with very low incidence rates of milk fever (see Figure 2). High concentrations of dietary potassium (a strong cation and dietary alkalinizer) caused milk fever in a recent study, but differing levels of dietary calcium had no effect on milk fever incidence (see Table 2).[20]

Two mechanisms have been proposed to explain why acidogenic diets help prevent milk fever. Both mechanisms involve increased resorption of calcium from bone, which is then used to support blood calcium concentrations. Acidogenic diets first promote bone mobilization by stimulating osteocytic bone resorption. This occurs because bone acts as a buffer against excessive systemic acidity by exchanging calcium ions for hydrogen ions from the bloodstream. Acidogenic diets have also been shown to increase the amount of 1,25 dihydroxyvitamin D produced per unit increase in parathyroid hormone.[21] This increases osteoclastic resorption of calcium from bone. When bone is already

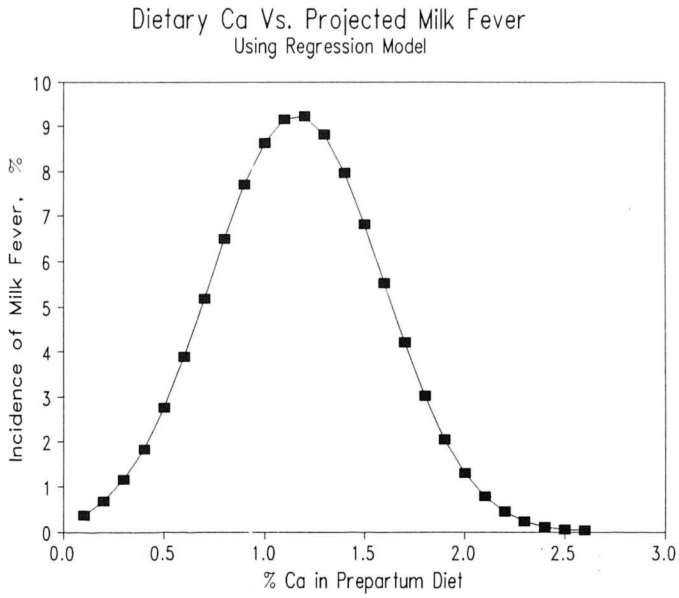

Figure 1. Sample relationship of dietary Ca to the incidence rate of milk fever using a logistic regression model. Points plotted were calculated for mixed breed cows, lactation number = 5, Na = .20%, and S = .35%. Adapted from Oetzel.[18]

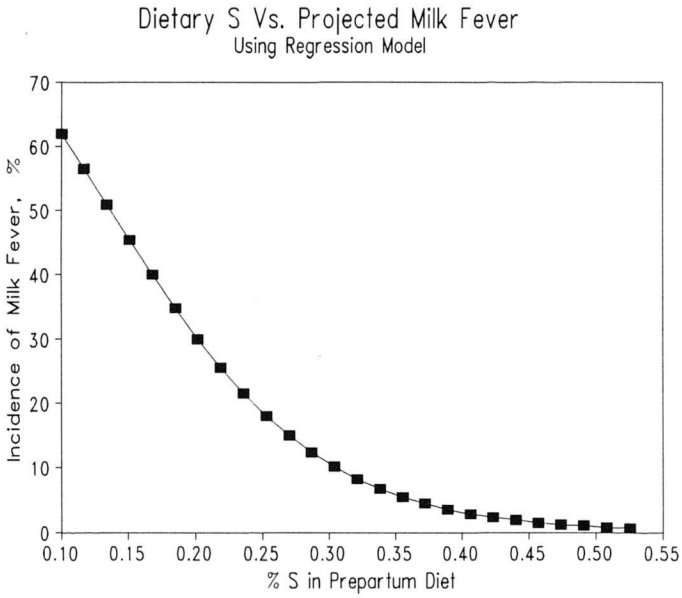

Figure 2. Sample relationship of dietary S to the incidence rate of milk fever using a logistic regression model. Points plotted were calculated for mixed breed cows, lactation number = 5, Na = .20%, and Ca = .75%. Adapted from Oetzel.[18]

being mobilized via these two mechanisms, cows are better able to respond to the sudden calcium demand of early lactation.

The potential of a diet to be either acidogenic or alkalogenic can be estimated by calculating its DCAD (Dietary Cation-Anion Difference). Understanding how DCAD affects systemic acid-base balance first requires

Table 2. Effect of dietary potassium and calcium on milk fever and urinary pH in Jersey cows. Adapted from Goff *et al.*[20]

Diet	DCAD[a] (meq/kg)	Milk fever cows/ Total cows	Milk fever treatments/Diet	Urinary pH (Mean ± SEM)
Low Ca[b] - Low K[c]	-150	0/9	0	5.8 ± .22
Low Ca - Medium K[c]	+150	4/11	9	8.0 ± .08
Low Ca - High K[c]	+450	7/9	15	8.1 ± .11
High Ca[b] - Low K	-150	2/10	2	5.7 ± .09
High Ca - Medium K	+150	6/9	9	7.9 ± .11
High Ca - High K	+460	3/12	4	8.2 ± .06

[a] DCAD = Dietary Anion-Cation Difference, expressed as (Na + K) - (Cl + S), milliequivalents per kilogram of diet dry matter.
[b] Low Ca diets were .49% Ca and high Ca diets were 1.5% K.
[c] Low K diets were 1.1% K, medium K diets were 1.8% K, and high K diets were 2.5% K. Potassium was added as potassium bicarbonate.

a short review of chemistry. Dietary electrolytes can be classified as either anions or cations. Anions have a negative charge; cations have a positive charge. The charge carried by these electrolytes affects acid-base balance and ultimately calcium metabolism. Important dietary cations are sodium, potassium, calcium, and magnesium; important dietary anions are chloride, sulfur, and phosphorus. Sodium, potassium, sulfur, and chloride are thought to exert the strongest ionic effects on acid-base balance and are referred to as the "strong ions".[22] Strong (or "fixed") ions are highly bioavailable and are not metabolized within the body.[23]

Dietary cation-anion difference can be used to quantitate the relationship between strong cations and anions and thus predict whether a diet will evoke an acidic or alkaline response when fed to a dairy cow. Several methods of calculating DCAD of a diet have been utilized, including the following equations:

$$DCAD\ (meq) = (Na + K + Ca + Mg) - (Cl + SO_4 + H_2PO_4 + HPO_4)$$

$$DCAD\ (meq) = (Na + K + Ca + Mg) - (Cl + S + P)$$

$$DCAD\ (meq) = (Na + K + .38\ Ca + .30\ Mg) - (Cl + .60\ S + .50\ P)$$

$$DCAD\ (meq) = (Na + K) - (Cl + S)$$

$$DCAD\ (meq) = (Na + K) - (Cl)$$

Based on the known physiologic effects of strong ions,[22] the fourth equation [(Na+K) -(Cl+S)] should be a reasonably accurate equation in predicting whether a diet will evoke an acidic or alkaline response. This equation also had the highest correlation to the incidence rate of milk fever in one study.[18] It has become the *de facto* standard for calculating DCAD among dairy nutritionists.[17] The third equation[17] takes into account the average bioavailability of all of the potential strong ions and theoretically should be more accurate than the other equations. The equations used to calculate DCAD and the units used to express it vary among ration evaluation software programs. Some programs differ by counting ions only if they are from certain types of feed ingredients (eg., mineral supplements vs. other ingre-

dients). The ideal DCAD calculation would take into account the actual bioavailability of all of the fixed ions within each feed ingredient and would adjust for different feeding conditions. Unfortunately, this specific information is not available.

Calculation of the DCAD of a diet, regardless of the equation employed, requires using the equivalent weights of the electrolytes. This is necessary because acid-base balance is affected by electrical charge rather than mass. The equivalent weight is equal to the molecular weight divided by the valence (electrical charge strength). The term milliequivalent (meq) is used to express equivalent weights; one milliequivalent equals 1/1000th of an equivalent. Table 3 provides reference values for calculating equivalent weights of important electrolytes and converting from percent of diet dry matter (DM) to milliequivalents per kilogram. Once milliequivalents concentrations are calculated, DCAD can then be computed by subtracting the anions from the cations.

Table 3. Molecular weights, equivalent weights, and conversions from percent to milliequivalents of anions and cations used in calculating DCAD. Adapted from Oetzel.[15]

Element	Molecular Weight	Valence	Equivalent Weight	To convert from % diet DM to meq, multiply by:	
	(g)	---	(g)	(meq/kg)	(meq/lb)
Sodium (Na)	23.0	1	23.0	434.98	197.72
Potassium (K)	39.1	1	39.1	255.74	116.25
Chloride (Cl)	35.5	1	35.5	282.06	128.21
Sulfur (S)	32.1	2	16.0	623.75	283.52
Calcium (Ca)	40.1	2	20.0	499.00	226.82
Magnesium (Mg)	24.3	2	12.2	822.64	373.93
Phosphorus (P)	31.0	1.8[a]	17.2	581.14	264.15

[a] The valence of P is 1.8 based on the normal distribution of mono-hydrogen and di-hydrogen forms of phosphorus in the body.

Manipulation of DCAD does not usually result in clinically significant changes in blood pH, because both kidney and bone compensate to maintain normal blood pH. For example, strongly anionic diets (low DCAD) are acidogenic, but blood pH remains nearly constant because urinary pH is reduced from about 8.0 to about 6.5. Similarly, cationic diets (high DCAD) are alkalogenic but have little effect on blood pH because the urine becomes more alkaline.

Most typical diets fed to dry cows will have an DCAD [using the formula (Na + K) - (Cl + S)] of about +100 to +250 meq/kg DM. Addition of a cationic salt (such as sodium bicarbonate) to the dry cow diet increases DCAD and would increase the incidence rate of milk fever. Adding an anionic salt or a mixture of anionic salts (minerals high in Cl and S relative to Na and K) to the diet lowers the DCAD and reduces the incidence of milk fever. Examples of different anionic salts, their equivalent weights, and costs are given in Table 4.

Table 4. Approximate retail costs and properties of anionic salts used in prevention of milk fever. Adapted from Oetzel.[15]

Anionic salt	Molecular Weight (g)	Equivalent Weight (g)	Mg (%)	Ca (%)	NPN[a] (%)	CP[b] (%)	Cl (%)	S (%)	Cost:[c] ($/cwt)	(¢/eq)
$MgCl_2 \cdot 6H_2O$	203.3	101.7	11.96	—	—	—	34.87	—	92.50	20.7
$MgSO_4 \cdot 7H_2O$	246.5	123.3	9.86	—	—	—	—	13.01	30.00	8.1
$CaCl_2 \cdot 2H_2O$	147.0	73.5	—	27.26	—	—	48.22	—	21.00	3.4
$CaSO_4 \cdot 2H_2O$	172.2	86.1	—	23.28	—	—	—	18.62	19.00	3.6
NH_4Cl	53.5	53.5	—	—	26.2	163	66.26	—	40.00	4.7
$(NH_4)_2SO_4$	132.1	66.1	—	—	21.2	133	—	24.26	21.50	3.1

[a] NPN = Nonprotein nitrogen.
[b] CP = Crude protein.
[c] Approximate wholesale prices of anionic salts; gathered from numerous feed sources.

Practical Questions About Feeding Anionic Salts

Are the anionic salts safe? It is known that force-feeding large amounts of these salts can be detrimental. However, lack of palatability limits the likelihood of toxicity if the salts are overdosed. A combination of salts is probably best, because it decreases the potential of toxicity from the cation (Mg, NH_4, Al, etc.) that must necessarily accompany each salt. It is possible to exceed NRC maximum tolerable amounts[24] of sulfur (.40%), magnesium (.50%) and NPN (.50%) by feeding large amounts of any single anionic salt.

Are the anionic salts palatable? It appears that the salts are not very palatable and that they are best fed in a total mixed ration (TMR) rather than in a grain or mineral mix alone. Palatability problems are minimal (not statistically significant) when the salts are added to a TMR.[7,9,19,25]

Palatability of the salts in component-fed diets is not good. It is best if the salts can be hand-mixed with a wet forage (corn silage or alfalfa haylage). If only dry forages or pasture are used, then the salts can be added to a grain mix, but with some difficulty. It appears that the salts must be mixed with more than at least 5 lbs. of a grain mix, and even then palatability may still be impaired.[26] Pelleting a mixture of anionic salts does not appear to increase their palatability,[26] but it does provide advantages in product formulation and in preventing separation of the anionic salts in a concentrate mixture. Pre-mixing loose salts with a carrier that has a strong flavor of its own (dried distillers grains or molasses) may be helpful and is commonly practiced. Ammonium salts pre-mixed into a concentrate mixture during warm weather may result in the release of ammonia gas and feed refusal. In addition, most of the salts are very hygroscopic and attract moisture, which may lead to caking.

Research[26] also indicates that $MgSO_4$ is the most palatable of the commonly used salts, and that $CaCl_2$ is the least palatable. Sulfates appear to have an advantage in palatability over chlorides. However, care must be taken to avoid over-feeding sulfur in the total diet,

and chlorides appear to be better urinary acidifiers than sulfates.[17,25]

What combination of anionic salts is most effective? Direct comparisons of the abilities of the individual anionic salts to prevent milk fever have not been done (such trials would require extremely large sample sizes). Indirect evidence[18] suggests that sulfates may be more effective in preventing milk fever than chlorides. Estimates of each salt's potential to prevent milk fever (measured as acid-base balance and calcium metabolism) have been made.[25] All of the salts tested in one study had significant effects on calcium metabolism and acid-base balance (see Table 5); ammonium chloride had a slight advantage over the other salts in its acidifying ability.[25] Interactions among anionic salt combinations have not been evaluated; however, there is no theoretical basis to suspect significant interactions. Selection of a mixture of anionic salts is generally made on the basis of price, availability, palatability, and avoidance of potential toxicity.

Table 5. Effect of anionic salt treatments on diet, acid-base balance, and calcium metabolism. Adapted from Oetzel *et al.*[25]

	Diet (control or with added anionic salts)						
Item	Control	$MgCl_2 \cdot 6H_2O$	$MgSO_4 \cdot 7H_2O$	$CaCl_2 \cdot 2H_2O$	$CaSO_4 \cdot 2H_2O$	NH_4Cl	$(NH_4)_2SO_4$
Salt added, g/d	0	204	246	147	172	107	132
Salt added, eq/d	0	2.0	2.0	2.0	2.0	2.0	2.0
Added NPN, %	0	0	0	0	0	.24	.24
DCAD,[a] meq/kg	-4	-172	-171	-170	-171	-172	-175
DM intake, % BW	1.70	1.70	1.65	1.63	1.68	1.68	1.67
Blood pH	7.38	7.38	7.38	7.38	7.38	7.37	7.38
Urinary pH	8.10	7.66	7.96	7.65	7.51	7.39	7.76
FECa,[b] %	.79	2.17	1.32	1.49	2.71	1.71	1.29

[a] DCAD = dietary cation-anion difference, calculated as [(Na+K) -(Cl+S)].
[b] FECa = fractional excretion of ionized calcium.

How long must the salts be fed before parturition? The time period of feeding the salts in previous trials has ranged from 21 to 45 days before expected parturition. It may be possible to feed the salts for a shorter time period; however, this theory has not been tested. The author's experience suggests that cows must consume the salts for at least 10 days in order to receive maximal benefit. There have been no reports of detrimental effects of feeding anionic salts for the entire dry period.

Will use of the anionic salts cause udder edema? A large field trial[7] found no differences in umbilical-udder edema scores taken 1 to 2 weeks postpartum between cows receiving an anionic salts mixture and cows which did not. Another trial showed slightly reduced udder edema scores in first lactation heifers fed calcium chloride.[27] Because udder edema is a sporadic disease of poorly understood etiology, there is a tendency to blame the anionic salts for any case of udder edema that occurs after the onset of their use.

Are the anionic salts cost-effective? Current costs of 2 to 3 equivalents of the salts are about 20 to 35 cents per cow per day. Costs of milk fever (both clinical and subclinical) are substantially greater than this. If gains of milk production of 3 to 7% can be expected, then the economic return from feeding the salts is about 10 to 1 for increased milk production alone.[7]

Formulation Strategies for Feeding Anionic Salts

Anionic salts have been added to prepartum diets either by using standard doses or by systematic calculation of mineral content and DCAD. An example of a standard daily dose is 4 ounces $MgSO_4 \cdot 7H_2O$ plus 4 ounces NH_4Cl (3.0 equivalents of anions). While standard doses of anionic salts may perform satisfactorily in many herds, this dosing strategy does not properly adjust for the existing mineral content of the prepartum diet. A systematic approach[28] to the calculation of the dose and type of anionic salts added to a prepartum diet has been proposed (Figure 3). Dry matter intake depression may occur when >300 meq of anions/kg of diet DM (about 3.5 eq/d at 26 lbs of DM intake) are added.[29]

No studies have been conducted to titrate the exact dose required to satisfactorily decrease the incidence rate of milk fever. Most of the studies to date have used doses of about 2 to 3 eq/d of anionic salts. It may be useful to adjust the dose of salts to a desired final DCAD (typically about 0 to -150 meq/kg DM). However, the optimal final DCAD is not known, and a wide range of DCAD's have shown apparent effectiveness in milk fever prevention. Difficulties in laboratory analysis for chlorine and sulfur (or use of reference values for these electrolytes) may limit the accuracy of calculated DCAD values. It has been suggested that monitoring urinary pH after feeding anionic salts may be the most direct and useful approach to establishing the optimal dose of anionic salts within a herd.[30,31] Mean urinary pH values in a group of close-up dry cows should be between 5.5 and about 6.5 if anionic salts are fed and the diet is properly formulated and delivered.

There is some uncertainty regarding the optimal concentration of dietary calcium that should be used when anionic salts are fed. Research has not definitively answered this question. There is evidence that the salts work best when dietary calcium is high.[7,19] Clinical experience strongly suggests that the anionic salts should <u>not</u> be used when dietary calcium is very low (less than about 60 g per day).

There is also uncertainty regarding the optimal

SYSTEMATIC APPROACH TO ANIONIC SALTS SUPPLEMENTATION

Step in Formulation:	Mineral Supplement:
1. Analyze available forages and concentrates for Na, K, Cl, and S.	—
2. Select feed ingredients with low DCAD (especially forages low in K).	—
3. Balance dietary Mg at .40%, dry matter basis.	$MgSO_4 \cdot 7H_2O$
4. Balance dietary S at .40%, dry matter basis.	$CaSO_4 \cdot 2H_2O$ (if needed) $(NH_4)_2SO_4$ (if needed)
5. Add dietary Cl to lower DCAD to -150 meq/kg, dry matter basis, or a total of <3.0 eq/day total anionic salts; if DCAD cannot be lowered to <0 meq/kg with <3.0 eq of anionic salts, then consider new feed ingredients with lower DCAD.	$CaCl_2 \cdot 2H_2O$ NH_4Cl
6. Check dietary NPN and DIP; if NPN is >.50%, or if DIP >70% of CP, then reduce the amount of ammonium salts added to the diet.	—
7. Add dietary Ca to a daily intake of at least 150 g; add dietary P to a daily intake of ~50 g.	limestone[a] monocalcium phosphate dicalcium phosphate monosodium phosphate
8. Monitor dry matter intake of the transition group. Consider more palatable salts or reduced dose of salts if dry matter intake is depressed.	—
9. Monitor urinary pH of the transition group. Adjust dose of anionic salts to achieve average urinary pH between about 5.5 and 6.5.	—

[a] The use of limestone has been questioned because it may be somewhat alkalogenic.[29]

Figure 3. A systematic approach to supplementing anionic salts in prepartum diets for dairy cattle. Adapted in part from Byers.[28] *DCAD* = Dietary cation-anion difference; *NPN* = Non-protein nitrogen; *DIP* = Degradable intake protein; *CP* = Crude protein.

concentration of dietary phosphorus. Dry cow diets high in phosphorus (>80 g of PO_4 per day) will increase the risk of milk fever due to inhibitory effects on vitamin D metabolism.[29] Most nutritionists provide about 40 to 50 g of daily phosphorus in dry cow diets containing anionic salts; however, there are reports of dry cow rations formulated to contain 70 to 90 g of daily phosphorus that have been fed with apparent success in preventing milk fever. These diets also contained high concentrations of calcium.

If dry cows are fed individually rather than as a group, it would be advantageous to feed the salts only to those cows at highest risk for milk fever, such as older cows and cows with previous episodes of milk fever. If only a few cows are fed the salts, then the extra labor of hand-preparing a TMR could be justified. If the anionic salts mixture must be part of a grain mix, then measures should be taken to improve the palatability of the mixture of salts (premixing, etc.). Dry cows should be brought up gradually, over a three day period, to the full feeding rate of the anionic salts mixture. If DM intake drops when the cow receives the full dose of the salts, then decrease the dose to the point that DM intake is acceptable. Excessive loss of DM intake just prior to calving is very undesirable and may lead to ketosis and/or fatty infiltration of the liver.

Oral Calcium Supplementation around Calving

Oral supplementation of dairy cattle with calcium chloride gels for milk fever prevention began over 20 years ago in Sweden.[32] Formulations have evolved from simple aqueous solutions of calcium chloride to thin gel solutions to thick paste preparations and recently to encapsulated (bolus) formulations.[33] Each improvement in the formulation has resulted in an increase in safety for the cow receiving the calcium chloride. There have also been new formulations using oils and propylene glycol. Positive results of recently published research trials have sparked interest in the use of calcium chloride supplements to prevent and even treat milk fever.

Calcium chloride has been used effectively as a source of supplemental calcium for parturient dairy cattle because of the high calcium content of the compound and because of the excellent availability of calcium in this form. Other dietary forms of calcium such as limestone (calcium carbonate) are relatively poorly absorbed into circulation compared to calcium chloride.[34] Calcium propionate can also be used as a source of calcium for oral supplementation;[34] however, relatively large doses are required to achieve similar increases in blood calcium concentration observed with oral calcium chloride administration.[35,36] The irritating nature of calcium chloride preparations may trigger partial closure of the esophageal groove following oral administration; this closure increases absorption of calcium from oral dosing,[34] since the main site of calcium absorption is apparently the small intestine rather than the rumen. Another advantage of calcium chloride in providing supplemental calcium is its acidic nature.[25]

The combination of high availability, partial bypass of the rumen, and acidic nature of calcium chloride results in surprisingly rapid increases in blood calcium concentrations following oral administration.[34,37] Administration of a typical commercial preparation containing calcium chloride results in peak blood calcium concentrations about 3 mg/dl higher than baseline concentrations.[34,37] Peaks occur about 30 minutes post-administration.[37] Blood calcium concentrations return to normal within about 24 hours. Even greater increases in blood calcium concentrations can be achieved by using calcium chloride in water instead of a gel (presumably due to greater closure of the esophageal groove);[34] however, the risk of aspiration pneumonia is too great to recommend administration in an aqueous solution.

There are three general uses of calcium chloride products in parturient dairy cattle. First, calcium chloride may be administered prophylactically to assist in milk fever prevention. The beneficial effect of preventive treatment has been demonstrated in a recent research trial;[38] prophylactic use of a calcium chloride gel product (Balance™, Bayer, Inc., Shawnee Mission, KS) significantly decreased the incidence of clinical milk fever from 11.8 to 4.9%. Use of the product also significantly decreased the incidence of subclinical milk fever (52.0 vs. 29.4%) and displaced abomasum (7.8 vs. 1.0%).

Oral calcium chloride has also been successfully used to treat clinical cases of milk fever in cases where the total blood calcium deficit was less than about 4 grams.[34] The risk of allowing a cow to remain recumbent any longer than necessary precludes the use of oral calcium chloride for treatment of recumbent milk fever cases. However, research suggests that it may be effective and practical to use oral calcium chloride products to treat early milk fever cows (stage 1) which are still standing.

Oral calcium chloride also shows promise for preventing relapses of clinical cases of milk fever that have responded initially to intravenous treatment. About 25% of such cases will relapse and become recumbent again within 12 to 24 hours unless preventive measures are taken.[10] Subcutaneous administration of 500 ml of a calcium-containing solution has been shown to reduce the rate of hypocalcemic relapses;[39] however, there are practical problems with this procedure, such as risk of infection. Oral calcium has the advantages of being easier, faster, and safer than subcutaneous administration; however, drug costs are greater with oral calcium supplementation.

Because calcium chloride is a very caustic compound that irritates tissues, there are definite risks associated with its oral administration. If some of the calcium chloride solution is aspirated into the lungs, the resulting irritation to the pulmonary tree will almost certainly cause a severe aspiration pneumonia. The use of thick gel or paste formulations of calcium chloride greatly reduces the risk of aspiration compared to calcium chloride given in water.

If the mucosal lining of the pharyngeal area is lacerated during the administration of a product containing calcium chloride, then the tissues will become severely inflamed by the presence of the calcium chloride. Severe infections, abscesses, and death may result. Extra caution should be exercised when orally administering a caustic substance such as calcium chloride.

There have been some concerns regarding possible rumen tissue irritation and/or ulceration following oral administration of calcium chloride. It appears that any rumen mucosal irritation is mild and transient. Using an oil carrier reduces the tissue irritation associated with calcium chloride in a gel form;[40] however, calcium absorption into the bloodstream is very poor when an oil carrier is used.[41]

Oral administration of calcium chloride gels can be an important tool in milk fever prevention. Their value is generally under-appreciated by dairymen and veterinarians. For cows known to be prone to milk fever, prophylactic treatment could start prior to calving (ideally 12 hours prior to delivery) and continue at 12-hour intervals for two or three treatments after calving. It would also be reasonable to use calcium chloride treatments for cows showing early signs of milk fever. This could save the owner a veterinary call and would save both the cow and owner the hassle of an intravenous treatment. Finally, it appears that the judicious use of calcium chloride products after successful intravenous treatment of recumbent cows has promise to reduce the incidence of hypocalcemic relapses. A reasonable protocol would appear to be to give two treatments 12 hours apart, with the first treatment given as soon as the cow is standing and has regained her swallowing reflexes.

Conclusions

Decreased reproductive performance in dairy cattle will result if milk fever cannot be controlled in a dairy herd. Approaches to milk fever prevention include dietary calcium restriction in the dry period, dietary acidification in the dry period, and oral calcium supplementation before and after calving. Choice of approach(es) to use depends on the dairy's management scheme, feeding system, forage availability, and degree of existing milk fever within the herd.

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Abstract

Necrotising enteritis in suckled calves

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Necrotising enteritis is a newly recognized disease affecting two to three-month-old calves in Scotland. A cohort of 10 calves from an affected herd was closely monitored from birth until the risk period was over, and one case occurred. In addition, all the cases of dysentery in suckled calves reported to SAC Veterinary Services, St Boswells, from April to August of 1992 were investigated and a further five outbreaks of necrotising enteritis were identified. The clinical pathology, gross and histological findings and results of microbiological investigations are described. No aetiological agent was identified and although the condition bore a superficial resemblance to mucosal disease the histological changes were distinct from those of mucosal disease and no bovine viral diarrhoea virus antigen was detected.