Use of Hydrochloric Acid as a Source of Anions for Prevention of Milk Fever[1]

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ABSTRACT

Diets that contain high amounts of K induce milk fever by alkalinizing the blood of the cow, reducing the ability of homeostatic mechanisms to maintain normal blood concentrations of Ca. The addition of anions to the diet induces metabolic acidosis, which counteracts the alkalinizing effect of the high cation diets commonly fed to cows. Currently, anions are usually added as anionic salts, such as $CaCl_2$ or $MgSO_4$, and the pH of urine is often monitored to assess the degree of metabolic acidification resulting from the addition of anions to the diet. An alternative source of anions is HCl. In Experiment 1, the addition of HCl to the diet of cows that were not pregnant and not lactating significantly reduced the pH of urine and blood within 24 h. After HCl was removed from the diet, the pH of urine returned to baseline levels within 48 h. In Experiment 2, the inclusion of HCl into the prepartum ration of Jersey cows entering the third or greater lactation significantly reduced the incidence of milk fever from 63% of control cows to 11% of the treated cows and also reduced the degree of hypocalcemia that was experienced by the cows during the periparturient period. Plasma Ca concentrations at 0.5 d after calving were 5.33 ± 0.52 and 6.69 ± 0.51 mg/dl in the control and the HCl-treated cows, respectively. In Experiment 2, the prepartum consumption of the ration with HCl was greater than the consumption of the control ration. In liquid form, HCl remains dangerous to handle and corrosive to machinery. Commercial preparations of HCl mixed into common feed ingredients as a premix could offer an inexpensive and palatable alternative to anionic salts as a means of controlling the incidence of milk fever in dairy cows.
(**Key words**: milk fever, hypocalcemia, hydrochloric acid)

INTRODUCTION

Current evidence suggests that milk fever can occur in cows as a result of excessive dietary cations. These cations, primarily K and Na, induce a metabolic alkalosis in the cow that impairs Ca homeostatic mechanisms via attenuated responsiveness of tissues to parathyroid hormone (1, 5, 11, 14, 25). During the early 1970s, a group of Norwegian researchers (10) demonstrated that the effects of these cations on the incidence of milk fever could be reduced by adding anions in the form of HCl and H_2SO_4 to the prepartum diet of cows. Handling these corrosive, inorganic acids on the farm, however, presented many problems, and, in subsequent papers, the Norwegian team reported that anionic salts such as $CaCl_2$, NH_4SO_4, $AlSO_4$, and $MgSO_4$ could be used in place of inorganic acids (8). Because anionic salts are relatively easy to handle, they have been the components of choice for overcoming the alkalinizing potential of prepartum diet (2, 4, 22). However, anionic salts can be unpalatable (21) and are always accompanied by a cation, which, depending on its rate of absorption, will negate some of the effects of the anions (16). The present studies were therefore initiated to determine whether HCl, a readily available and inexpensive source of anions, should be reconsidered as a source of anions in the prepartum diet of the dry cow.

Experiment 1 was conducted to assess how long the HCl had to be in the diet to acidify effectively and how quickly the pH of blood and urine returned to baseline levels upon removal of HCl from the diet. In Experiment 2, older Jersey cows that were at risk of developing milk fever were fed diets supplemented or unsupplemented with HCl to determine the effectiveness of HCl in the prevention of hypocalcemia and milk fever.

MATERIALS AND METHODS

Experiment 1: Time Required to Alter Acid-Base Status with Dietary HCl

Eight Jersey cows that were not pregnant and not lactating were fed 5 kg (DM) of the basal ration (Table 1) for 10 d. For the next 7 d, cows were fed the

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TABLE 1. Composition of diets fed to Jersey cows that were not pregnant and not lactating to assess response of blood and urine to the acidifying activity of HCl (Experiment 1).

Composition	Basal ration	HCl
Ingredient, % of DM		
Corn silage	48.0	47.1
Alfalfa hay	29.1	28.6
Beet pulp	21.3	20.9
Magnesium oxide	0.3	0.3
Sodium bicarbonate	1.2	1.2
Vitamin-mineral premix[1]	0.1	0.1
HCl	. . .	1.8
	As analyzed	Calculated
Mineral composition,[2] % of diet		
Na	0.33	0.33
K	1.17	1.17
Ca	0.54	0.54
Mg	0.41	0.41
Cl	0.13	1.88
S	0.15	0.15

[1]Supplied 12,500 IU of retinyl palmitate, 2500 IU of vitamin D_3, 30 IU of tocopherol acetate, and 0.11 mg of Se and met or exceeded NRC (19) requirements for all trace elements.

[2]Basal ration composition analyzed by wet chemistry. The composition of the HCl diet was calculated from addition of a known amount of HCl to the basal ration.

basal ration with 2.5 eq/d of HCl added. Muriatic acid was diluted with water and molasses (acid:water: molasses; 4:3:1, vol/vol/vol) prior to its addition to a mixer wagon. This ration was made in a mixer wagon every 4 d and was stored in an airtight plastic container until it was fed to the cows. The cows were then fed the basal ration for 2 d to assess the recovery from the effects of HCl. Samples of jugular venous blood and urine were obtained to assess the baseline acid-base status and Ca status. The cows were fed at 0800 and 1400 h, and urine samples were obtained daily between 1000 and 1100 h. Urine was also collected at 16 h after the initial offering of the HCl diet. Blood samples were obtained immediately before the diet switch, 24 h and 7 d after initiation of the HCl diet, and 24 h after withdrawal of the HCl diet (d 8).

Experiment 2: HCl to Prevent Milk Fever

Jersey cows entering their fourth or greater lactation were assigned to the control group (n = 11) or the treatment group, which received 1.5 eq of HCl/d (n = 9). Cows were housed in free stalls that were bedded with sand at the National Animal Disease Center (Ames, IA). Cows were moved into maternity pens that were bedded with newspaper when parturition seemed imminent and were again housed in the free-stall area by the 3rd d of lactation. Cows were offered daily 8.4 kg (DM; ~1.9% BW) of a corn silage and alfalfa hay diet (Table 2). Cows were fed at 0800 and 1400 h. The treated cows received 0.75 eq of HCl mixed with 50 ml of molasses and diluted to a final volume of 200 ml with water and hand-mixed with the diet immediately prior to each feeding. The control cows received 50 ml of molasses in 200 ml of water added to the diet at each feeding. The cows were fed using the Calan gate feeding system (American Calan, Inc., Northwood, NH), which permitted determination of feed intake. Feed was placed into feed bins twice daily (0800 and 1400 h), and orts were weighed once daily (0800 h). Cows began receiving the experimental diets 3 wk before the expected date of parturition. The treatments were ended at calving at which time all cows were offered the control ration with 200 g of $CaCO_3$ added and an additional 2.2 kg of grain and 2.2 kg of long-stem alfalfa hay daily for the duration of the experiment. Heparinized samples of jugular blood were obtained daily from approximately 2 wk before calving to 2 wk after calving (with more frequent sampling when calving seemed imminent) and immediately before any treatments for milk fever were administered. Several times before calving, urine samples were ob-

TABLE 2. Composition of control diets and diets with HCl fed during the last 3 wk of gestation of mature Jersey cows.

	Diet	
Composition	Control	HCl
Ingredient, % of DM		
Corn silage	44.9	44.6
Alfalfa hay	5.3	5.2
Beet pulp	24.1	24.0
Soybean meal (44% protein)	14.2	14.2
Distillers grains	10.6	10.5
Magnesium oxide	0.3	0.3
Vitamin-mineral premix[1]	0.5	0.5
Molasses	0.1	0.1
HCl	. . .	0.65
	As analyzed	Calculated
Dietary analysis,[2] % of diet		
NE_L, Mcal/kg	1.76	1.76
CP	15.4	15.4
Na	0.03	0.03
K	0.98	0.98
Ca	0.38	0.38
Mg	0.43	0.43
Cl	0.12	0.75
S	0.23	0.23

[1]Vitamin-mineral premix supplied 125,000 IU of retinyl palmitate, 25,000 IU of vitamin D_3, 3000 IU tocopherol acetate, and 2.4 mg of Se/d, and met or exceeded NRC (19) requirements for all trace elements.

[2]Basal ration composition analyzed by wet chemistry. The composition of the HCl diet was calculated from the addition of a known amount of HCl to the basal ration.

tained from each cow by manual stimulation of the vulva. The pH of the urine sample obtained closest to 2 d before calving was used in the data analysis.

A cow was considered to have milk fever if she was recumbent and her plasma Ca concentration was <5.5 mg/100 ml. Milk fever and any subsequent relapses were treated by the intravenous administration of 10.5 g Ca as Ca borogluconate. A cow was classified as having subclinical hypocalcemia if plasma Ca concentration fell to <7.5 mg/100 ml at any time during the experiment.

Diet Analysis

Samples of the basal ration of Experiments 1 were analyzed for mineral content, and samples of the control ration utilized in Experiment 2 were obtained periodically for analyses of mineral, fiber, energy, and protein (Northeast Dairy Herd Improvement Association Laboratory, Ithaca, NY).

Plasma and Urine Analysis

Plasma and urine concentrations of Ca and Mg were determined by atomic absorption spectrophotometry (24). Plasma concentrations of P (23), hydroxyproline (7), and urine creatinine (6) were determined colorimetrically. Hydroxyproline is an amino acid unique to type I collagen, and the concentration of hydroxyproline in plasma can be utilized as an index of resorption activity of bone collagen. Plasma concentration of 1,25-dihydroxyvitamin D was determined by radioreceptor assay (26). Blood gases and pH were determined on samples of jugular blood, using sodium heparin as an anticoagulant. Samples were maintained at 4°C until analyzed (within 1 h of obtaining the sample). Determinations of urine pH (model 150 pH meter; Corning, Corning, NY) were made within 1 h of urine collection.

Statistical Analysis

For Experiment 1, repeated measures ANOVA with time as the main effect was used to assess how long HCl had to be fed to cows to effect a significant change in urine pH and calcium concentration and in blood pH and bicarbonate concentration. A post hoc Fisher's LSD test was used to compare differences between means for urine pH and blood pH obtained on d 0 with means observed during and after feeding HCl (Statview; Abacus Concepts, Inc., Berkeley, CA).

The effects of HCl on the incidence of milk fever and subclinical hypocalcemia were assessed by chi-square analysis. Dietary HCl effects on plasma constituents of periparturient cows were analyzed by repeated measures ANOVA of data collected between d −3 and +3 (or as stated in the Results Section) around calving with treatment, day around parturition, and the interaction between treatment and day around parturition as the main effects. A Post hoc Fisher's LSD test was used to determine the significance of differences of means for blood parameters as affected by treatment and day around parturition. Determinations of urine pH from 5 d before calving until calving were reduced to a single mean value for each cow prior to statistical analysis. The significance of the effect of diet on urine pH was determined by Student's *t* test.

The values presented for all parameters are the means and standard errors of the mean. Mean differences are considered significant when the probability of wrongly rejecting the null hypothesis is <0.05, unless otherwise stated in the text.

RESULTS

Experiment 1: Time Required to Alter the Acid-Base Status with Dietary HCl

The pH of urine samples obtained 16 h after the addition of 2.5 eq of HCl to the diet was lower than the pH of urine samples obtained before the HCl was added, and the pH remained low throughout the 7 d that HCl was incorporated into the diet (Figure 1A). Within 24 h after the removal of HCl from the diet, the pH of the urine had increased and was no longer different from values obtained prior to HCl treatment. The urinary concentrations of Ca were increased 24 h after HCl addition to the diet and remained high until HCl was removed from the diet (Figure 1B).

By 24 h after the addition of HCl to the diet, the pH of the blood was lower ($P < 0.01$) than that of blood obtained prior to the addition of HCl. The pH of blood on d 7 of HCl supplementation was lower ($P < 0.075$) than before supplementation but not to the same extent as on the 1st d of HCl supplementation (Figure 1C). The pH of blood did not increase to pretreatment levels within the first 24 h after HCl removal from the diet. The blood bicarbonate concentration was lower than pretreatment concentrations by 24 h after HCl addition to the diet but not after 7 d of HCl treatment or 1 d after the removal of HCl from the diet (Figure 1D).

Experiment 2: Effect of HCl on the Incidence of Milk Fever

The addition of HCl to the diet reduced ($P < 0.01$) the incidence of milk fever. Although 63% (7 of 11) of

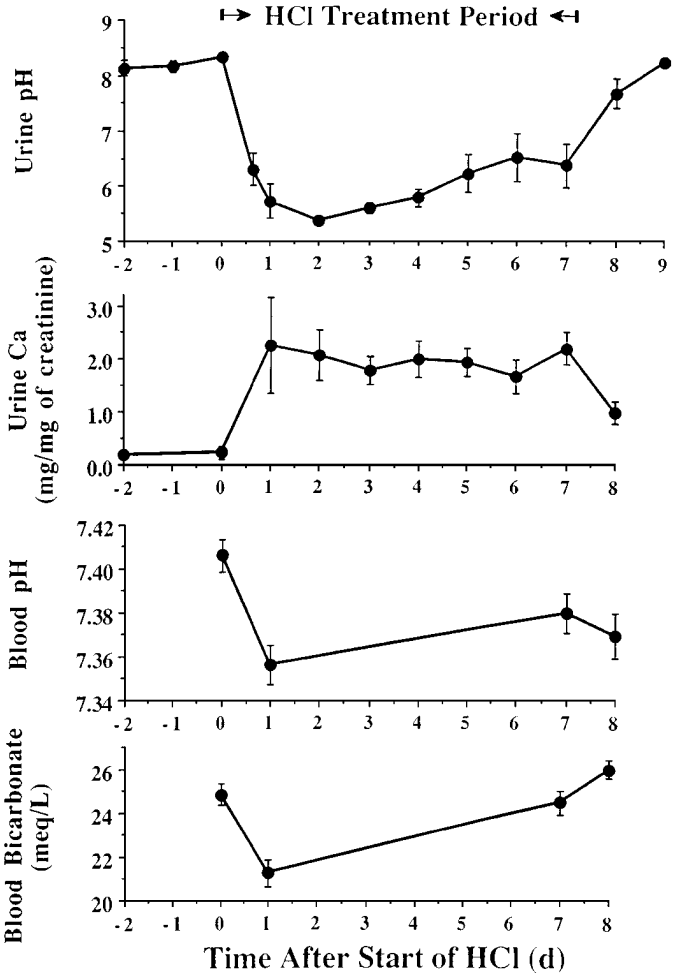

Figure 1. Mean (± SEM) values for urine pH, urinary excretion of Ca, blood pH, and blood bicarbonate of 8 Jersey cows fed the basal ration of Experiment 2, followed by the basal ration with 2.5 eq/d of HCl added for 7 d beginning at d 0.

untreated cows developed milk fever, only 11% (1 of 9) of the cows that were fed the diet with HCl developed milk fever. Of the 7 cows in the control group with milk fever, 5 suffered relapses after initial treatment. A cow that developed milk fever after being fed the HCl diet recovered following a single intravenous injection of Ca.

Both dietary treatment and day around parturition were significant sources of variation of plasma calcium concentration. The interaction between treatment and day around parturition was not significant. The mean plasma concentration of Ca of the cows that were fed HCl (7.48 ± 0.13 mg/dl) was greater ($P < 0.01$) than that of the control cows (6.89 ± 0.18 mg/dl) across the period from 3 d before calving until 3 d after calving (Figure 2A). The plasma concentration of Ca at 0.5 d after calving was 5.33 ± 0.52 mg/dl for the controls and 6.69 ± 0.51 mg/dl for the HCl-treated cows. The day around parturition was the only significant source of variation of plasma concentration of P. The mean plasma concentration of P from 3 d before calving until 3 d after calving (Figure 2B) was not higher ($P = 0.08$) for HCl-treated cows than for control cows (4.14 ± 0.17 vs. 3.91 ± 0.19 mg/dl, respectively). The plasma concentration of P at 0.5 d after calving was 3.50 ± 0.58 mg/dl for HCl-treated cows and 2.82 ± 0.61 mg/dl for control cows. The interaction between treatment and day around parturition was not significant. Both dietary treatment and day around parturition were significant sources of variation of plasma concentration of Mg. The interaction

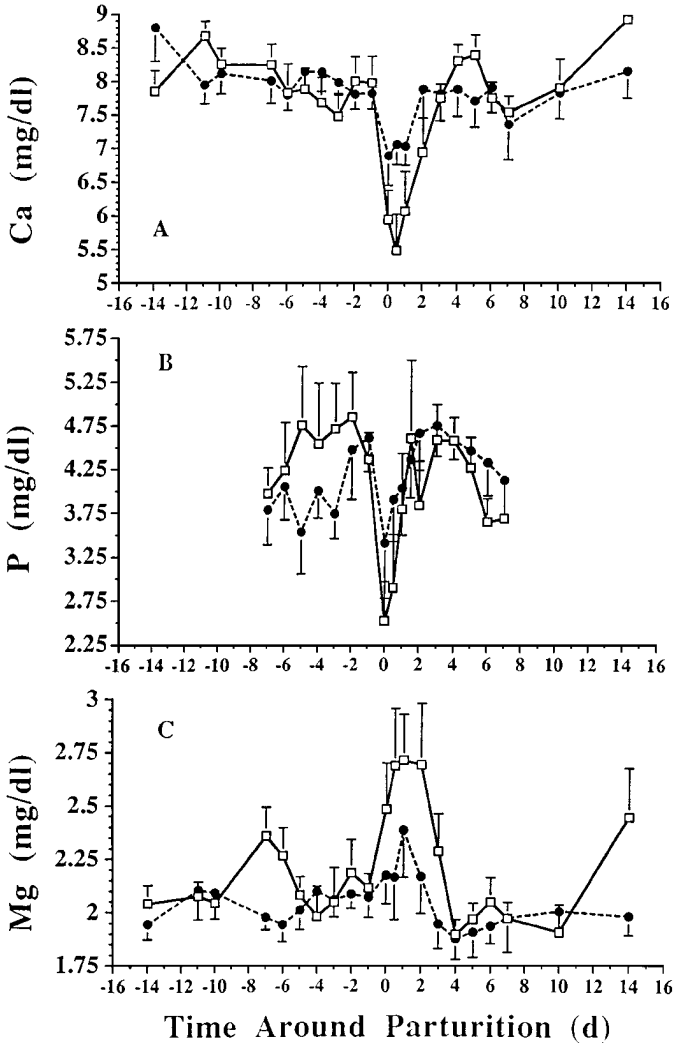

Figure 2. Mean (± SEM) values for plasma concentrations of Ca, P, and Mg in periparturient Jersey cows fed a prepartum diet with (– –●– –; n = 9) or without (— □ —; n = 11) the addition of 1.5 of HCl.

between treatment and day around parturition was not significant. The mean plasma concentration of Mg from 3 d before calving until 3 d after calving (Figure 2C) was higher ($P < 0.01$) for control cows than for HCl-treated cows (2.40 ± 0.08 vs. 2.14 ± 0.05 mg/dl, respectively). The plasma concentration of Mg at 0.5 d after calving was 2.22 ± 0.21 mg/dl for HCl-treated cows and 2.72 ± 0.27 mg/dl for control cows.

Both dietary treatment and day around parturition were significant sources of variation for plasma concentrations of hydroxyproline and 1,25-dihydroxyvitamin D across the time period analyzed (7 d before parturition through 3 d after calving). Plasma concentrations of hydroxyproline and 1,25-dihydroxyvitamin D increased significantly as parturition approached, especially on the first day after calving (Figure 3, A and B). The mean hydroxyproline concentration across the analyzed periparturient period was greater in HCl-treated cows (1.49 ± 0.05 μg/ml) than in control cows (1.35 ± 0.04 μg/ml); however, post hoc testing of daily means could not demonstrate a significant difference in plasma hydroxyproline concentration between HCl-treated and control cows when compared at any given day in the periparturient period. Similarly, the mean plasma 1,25-dihydroxyvitamin D concentration of the control cows across the entire periparturient period analyzed (from 5 d before parturition through 3 d after parturition) was greater than in HCl-treated cows (104 ± 8 and 81 ± 6 pg/ml, respectively); however, post hoc testing of daily means could not demonstrate a significant difference in plasma 1,25-dihydroxyvitamin D concentrations of treated and control cows when treatments were compared at any given day in the periparturient period. The interaction between treatment and day around parturition was not a significant source of variation for either concentration of hydroxyproline or 1,25-dihydroxyvitamin D.

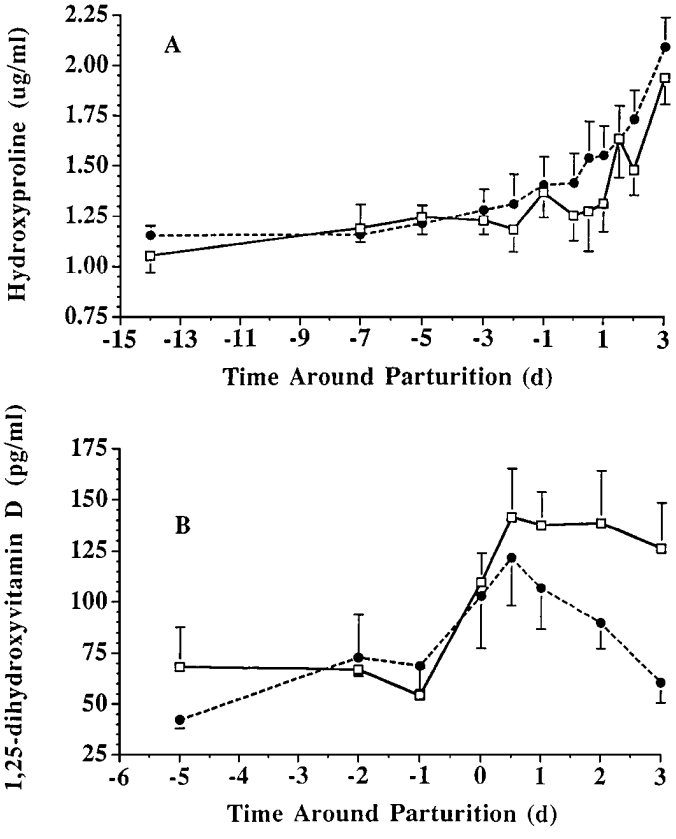

Figure 3. Mean (± SEM) plasma concentrations of hydroxyproline and 1,25-dihydroxyvitamin D in periparturient Jersey cows fed a prepartum diet with (– –●– –; n = 9) or without (— □ —; n = 11) the addition of 1.5 of HCl.

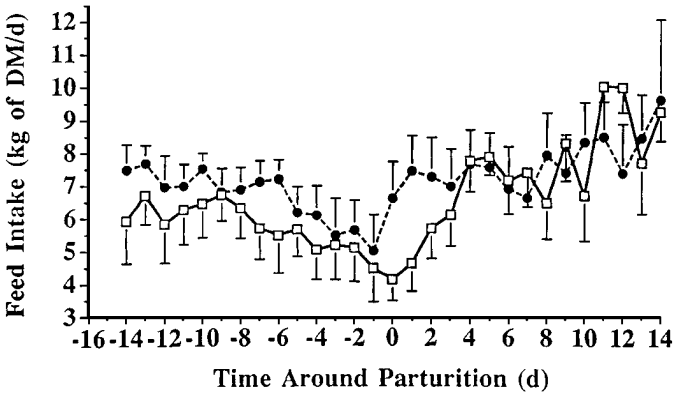

Figure 4. Mean (± SEM) intake of DM of periparturient Jersey cows fed a prepartum diet with (– –●– –; n = 9) or without (— □ —; n = 11) the addition of 1.5 eq of HCl.

Feed intake from 14 d before calving until 14 d after calving was greater for cows fed the diet with HCl than for cows fed the control ration prior to calving (Figure 4). Dry matter intake across this entire period averaged 6.12 ± 0.20 kg/d for the control cows and 7.02 ± 0.19 kg/d for cows fed the HCl diet. The effect of day around calving on feed intake was significant, and both groups experienced a decline in feed intake just before parturition. The interaction between treatment and day around parturition was not significant. Post hoc LSD mean feed intake of control cows was significantly less than the feed intake of cows fed HCl the day of calving and the 1st d of lactation. When the data analyzed included only the feed intakes prior to calving, which is during the time when the experimental rations were fed (from 14 d before calving until the day before calving), the cows fed the HCl diet consumed more DM than did cows receiving the control ration (6.56 ± 0.22 vs. 5.65

± 0.27 kg/d, respectively). No significant interaction between treatment and day around calving was observed for feed intake prior to calving.

The pH of urine within 5 d of calving of the control cows was higher than that for the cows fed HCl (7.69 ± 0.33 vs. 6.61 ± 0.19, respectively).

DISCUSSION

Diets that are high in the readily absorbable cations, K or Na, induce milk fever in dairy cows (8, 12). Diets that are high in K or Na alkalinize the blood and the extracellular fluids of the cow (27), which reduces bone Ca resorption (4, 13) and renal production of 1,25-dihydroxyvitamin D (11, 13, 25). It is now becoming a common practice to add anionic salts to prepartum rations to reduce the degree of hypocalcemia experienced by dairy cows at parturition (3, 4, 22). Growing evidence suggests that anionic salts prevent milk fever by acidifying the blood to restore tissue responsiveness to the parathyroid hormone. Because the pH of urine generally reflects the acid-base state of an animal, monitoring the pH of urine can be an inexpensive and sensitive method to monitor the effect of diet on the pH of blood and assess the risk of milk fever (11, 17).

The first experiment of this study demonstrates that cows respond rapidly to dietary supplementation of anions. Within 24 h of initiation of treatment, the pH of urine can be acidified to between pH 6.0 and 6.8, the range at which cows can be considered to be less likely to develop milk fever (11, 17). If the pH of blood is the major factor determining the ability of the cow to maintain Ca homeostasis at calving, it may be possible to feed anions for only 1 or 2 d before calving to prevent milk fever. Although this procedure is currently not practical because of problems with the prediction of calving date and the introduction of new feedstuffs immediately before calving, the possibility of the prevention of milk fever through rapid acidification is scientifically interesting. This study also suggests that the urine pH, blood pH, and blood bicarbonate increased after several days of HCl supplementation. There was no decline in feed intake during this period, which suggests that cows adapt to the supplemental anions. This adaptation response may be the result of an increased release of cations from the bone to counteract dietary anions (i.e., the bone acting as a buffer of the blood acidity) (18), a response of the respiratory system to the metabolic acidosis induced by the anions, or both. At present, there is no evidence to suggest that this adaptation to anions in the diet reduces or increases the risk of milk fever in dairy cows.

The original discovery that milk fever could be prevented by the addition of anions to a ration was conducted using HCl and H_2SO_4 as sources of anions (8, 9). In the second experiment of this study, we confirmed that the addition of HCl to prepartum diets is a highly effective method to prevent milk fever and that HCl can be an inexpensive source of anions. The use of these acids was abandoned in favor of the salts of these acids primarily because of concerns for the safety of farm personnel and the effect the corrosive nature of HCl has on feed handling equipment. In studies currently being conducted in our laboratory and on cooperating farms, we have greatly reduced these problems by preparing a premix of HCl using beet pulp as a carrier for handling by farm personnel, and we assume that this idea could be developed for commercial purposes using beet pulp or other carriers. In a concentrated liquid form, HCl remains too difficult and dangerous for use on the farm, and we strongly discourage its use in that way.

The addition of traditional anionic salts to prepartum diets can reduce palatability, which sometimes reduces feed intake prior to calving (20), which may precipitate other metabolic diseases in the cow (15). In this experiment, the addition of HCl to the diet actually increased feed intake prior to calving compared with the feed intake of the control cows. However, even in the cows treated with HCl, prepartum DMI averaged only 1.3% of cow body weight, which was considerably less than our target of 1.75 % of body weight. Because of these low feed intakes, we hesitate to state that HCl stimulates DMI. Immediately after calving, the cows that were fed HCl had significantly higher feed intakes than the control cows for the first 3 d of lactation, which was likely due to the prevention of milk fever because those cows that were afflicted with milk fever ate little during this time. The control cows also had more metabolic disease (3 cows developed ketosis and 2 developed left displaced abomasum) than did cows that were fed HCl (1 cow treated for ketosis and concurrent left displaced abomasum), which likely contributed to the poor feed intake after calving in the control cows.

CONCLUSIONS

Hydrochloric acid is an excellent source of the chloride anion and proved to be an effective means of adjusting dietary cation-anion difference of prepartum rations to prevent milk fever. In the liquid form, however, HCl remains dangerous, and problems with handling HCl will have to be overcome to allow widespread use of HCl to prevent milk fever and hypocalcemia.

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