Dietary Cation-Anion Difference and the Health and Production of Pasture-Fed Dairy Cows 2. Nonlactating Periparturient Cows

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ABSTRACT

Anecdotal observations of reduced hypocalcemia due to small reductions in the precalving dietary cation-anion difference (DCAD) are widely reported in Australia and New Zealand. Diets offered to nonlactating, periparturient dairy cows in pasture-based dairy systems in southeastern Australia can vary in their cation-anion difference from 0 to +76 mEq/100 g. The effects of such a range in the DCAD on the health and production of cows, on a pasture-based diet, were examined in an indoor feeding experiment. Four groups of four cows were offered pasture-hay and freshly cut pasture, a periparturient diet typical of that associated with the grazing system in Australia and New Zealand. Varying levels of salt supplementation were used to alter the dietary cation-anion difference, which ranged from -12 to +69 mEq/100 g. Blood and urine pH and mineral concentrations and urine hydroxyproline were measured. The addition of anions to the diet, to produce a negative DCAD, resulted in a nonrespiratory systemic acidosis. With decreasing DCAD, the pH of blood and urine and the strong ion difference of urine decreased curvilinearly, blood bicarbonate decreased linearly and the urinary ratio of Ca to creatinine increased curvilinearly. Although systemic pH was not reduced at a DCAD of +16 mEq/100 g, urine Ca-to-creatinine ratio had begun to rise, probably indicating increased calcium absorption. The absorption and renal excretion of Mg increased with decreasing DCAD. No differences were observed in urine hydroxyproline concentrations and no significant differences in milk production were measured.

(Key words: hypocalcaemia, dietary cation-anion difference, pasture, potassium)

Abbreviation key: Creat = creatinine, DCAD = dietary cation-anion difference, DMD = dry matter digestibility, Hy = hydroxyproline, ICP-ES = inductively coupled plasma emission spectrometry, ME = metabolizable energy.

INTRODUCTION

In the transition from pregnancy to lactation, the clearance of Ca to the placenta ceases, but the lactational Ca demand increases rapidly (Ramberg et al., 1984). Parturient hypocalcemia or paresis is a metabolic disorder caused by the increased demand placed on the plasma Ca pool by the sudden onset of lactation. More commonly known as milk fever, parturient paresis has been shown to affect approximately 2 to 5% of cows in Australia (Caple, 1987) and 2% of cows in New Zealand (McDougall, 2001). Although the mean incidence may be low, this ranges from 2 to 20% on individual farms (J. R. Roche, unpublished).

Plasma Ca concentration is under the control of parathyroid hormone, calcitonin, and the metabolites of vitamin D (Lindsay and Pethick, 1983). The losses of Ca from plasma may be regarded as disturbing signals to which these homeostatic mechanisms must respond to maintain eucalcemia (Ramberg et al., 1984). Hypocalcemia develops in most cows due to delays in these feedback mechanisms. However, in most animals, hypocalcemia is not severe enough to cause clinical signs, and blood Ca levels quickly return to normal (Ramberg et al., 1984; Goff and Horst, 1997b).

Stewart (1983) showed that the addition of anions to a solution decreased pH. The addition of anions to body fluids through dietary supplementation should, therefore, decrease the pH of body fluids. Although blood pH is highly regulated, slight variations can affect Ca metabolism (Bushinsky et al., 1993; Schonewille et al., 1994) and a prepartum diet, with a negative dietary cation-anion difference (DCAD) has been shown to improve periparturient calcium homeostasis (Block, 1984; Goff et al., 1991; Joyce et al., 1997). A low urine pH, an indicator of blood pH (Vagnoni and Oetzel, 1998), has been associated with an increased gastrointestinal

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absorption and urinary output of Ca (Vagg and Payne, 1970; Schonewille et al., 1994). However Schonewille et al. (1994) reported that increased absorption of Ca accounted for only 60% of the increased excretion and concluded that Ca from another source was being added to the urinary Ca pool. The source of the additional Ca remains unclear.

One potential source of Ca is bone, through mobilization of Ca stores. Urinary hydroxyproline has been used in previous research as an indicator of bone resorption (Robins, 1994). However, results to date on the additional source of Ca are inconclusive. For example, in some experiments where DCAD was reduced (Block, 1984; Goff et al., 1991; Goff and Horst, 1997a), an increase in urinary hydroxyproline was reported. The findings of Bushinsky et al. (1993) on the effects of systemic acidosis on bone resorption agree with these results. In contrast, other researchers have found no difference in bone histomorphology or in the concentration of urinary hydroxyproline in nonlactating cows when DCAD was reduced and postulated that the other Ca source was decreased bone accretion (Schonewille et al., 1994; Van Mosel et al., 1994).

The dairying industry of southeastern Australia is largely perennial ryegrass-based and supplemented with cereal grain, pasture-hay, and pasture silage (Doyle et al., 1996). This diet has a very high DCAD (Jacobs and Rigby, 1999; Roche et al., 2000) and differs to that generally investigated as a precalving diet for cows in other countries. The DCAD of this diet can be as high as +76 mEq/100 g (Roche et al., 2000), making a negative DCAD, as is recommended in the literature, difficult and generally impractical to achieve. Nevertheless, the improved Ca homeostasis reported by Roche et al. (2002) in grazing animals receiving a DCAD of +15 to +20 mEq/kg and anecdotal reports of reduced milk fever incidence when DCAD is marginally reduced demand an investigation of the DCAD concept as a means of preventing periparturient hypocalcemia in pasture-based cows. The objectives of this experiment were to compare a range of DCAD (−10 to +65 mEq/100 g) on a perennial ryegrass and pasture hay diet before calving, examining the effects on systemic pH, milk fever, and subsequent lactation performance.

MATERIALS AND METHODS

Experimental Design and Treatments

A total of 16 multiparous Holstein cows, 19 ± 5 d precalving, were randomly allocated to four dietary treatments in a randomized block design. Cows were allocated randomly to treatment using Baird's method (Harville, 1974; Baird, 1994) on the basis of total production for the previous lactation, and age. The cows

were about to begin (mean $\pm$ SD) lactation number 4 ± 2.5 and had produced 5500 ± 900 kg of milk the previous lactation with $4.4 \pm 0.53\%$ fat and $3.2 \pm 0.14\%$ protein.

Feeds

All cows were offered a daily diet of 5 kg DM of pasture hay in the morning and 5 kg DM of freshly cut pasture in the afternoon for 24 ± 10 d before calving. After calving, the cows were switched to a ration of 6 kg DM of barley and ad libitum freshly cut pasture for 3 wk. Ad libitum was defined as 110% of the pasture that had been eaten the previous day. The nutritive characteristics and mineral concentrations of the feeds offered are given in Table 1.

Dietary Cation-Anion Difference

The DCAD [(Na + K) − (Cl + S); Tucker et al., 1992] offered before calving was manipulated by adding salts of either Na, Cl, or S such that the four treatments received a planned DCAD of −10, +15, +40, and +65 mEq/100 g DM.

Table 2 shows the salts used and the daily quantities that were administered before calving. The basal DCAD, based on previous work (Roche et al., 2000), was assumed to be approximately +30 mEq/100 g DM. MgSO_4 ($\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$), MgCl_2 ($\text{MgCl}_2 \cdot 6\text{H}_2\text{O}$), and CaCl_2 ($\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$) were used to reduce the DCAD, and NaHCO_3 was used to increase it. NaHCO_3 was chosen to increase the DCAD as it is the most commonly supplemented cationic salt in Australian situations. Furthermore, the findings reported by Goff and Horst (1997) showed that Na or K had similar effects on calcium homeostasis. MgO and CaCO_3 were used to balance the dietary Mg and raise the dietary Ca concentrations (approximately 0.5 and 1.0%, respectively).

Before calving, the cows received their appropriate salt mixture, twice daily, at 0600 and 1500 h. If DCAD is to be accurately manipulated in pasture-based systems, it will be through supplementation of cows with appropriate salt mixtures once or twice daily, as the consistent and predictable manipulation of DCAD has been shown to be very difficult (Roche et al., 2002). Furthermore, the pasture-based system, where cows are generally a considerable distance from supplementation facilities precludes a more frequent supplementation strategy. In support of this method of supplementation, Roche (1999) showed increased Ca absorption and urinary Ca excretion and little or no diurnal variation in urine pH when anionic salts were supplemented twice daily to reduce the DCAD to −20 mEq/100 g, thereby suggesting that twice daily supplementation of anionic salts in pasture-based systems had similar effects to continuous supplementation in a TMR.

Table 1. Mean DM (%) and nutrient analysis (% DM) of the feeds offered.

	DM	CP	DMD [†]	ME [‡]	K	Na	Cl	S	Mg	Ca	P
Pasture	16.2	20.3	80	2.77	3.37	0.35	1.21	0.28	0.23	0.41	0.35
Barley	89.0	12.7	79	2.72	0.49	0.11	0.18	0.11	0.12	0.04	0.26
Hay	82.5	7.2	60	1.96	1.64	0.55	1.84	0.17	0.23	0.28	0.16

[†]DMD = In vitro DM digestibility.

[‡]ME = Metabolizable energy (Mcal/kg – calculated as ME = ((DMD * 0.17) – 2)/4.186) — Standing Committee on Agriculture, 1990.

As the DCAD of the diet was high, and the amounts of salts required to reduce or increase it to the predetermined levels were so great, the salts used were administered orally in solution using a stomach tube, as described by Roche et al. (2003).

Animal Management

Cows were individually fed for the duration of the experimental period. Feeds were offered daily for two 5-h periods. At 1200 to 1445 h and 2100 to 0545 h, the cows were held as a single group on a bare paddock where water was available ad libitum.

After receiving their salt supplement, precalving, the cows received their morning and afternoon feed. They calved indoors and were monitored to determine the duration of calving. Calving time was deemed to be the time from first sign of the calf's hooves until the calf was born.

After calving, the cows were milked daily at 0600 and 1500 h. They were then allowed access to half their daily allocation of barley for 20 min before being allowed access to fresh pasture.

The pastures used in this experiment had not been grazed for 6 to 7 wk and were harvested at a mass of approximately 2500 kg DM/ha. The pasture was cut to approximately 40 mm, twice daily, and collected using a loader wagon. The cut pasture was not chopped further so as to best represent grazed pasture. The sward consisted of approximately 70% perennial ryegrass (*Lolium perenne* L.), 25% other grasses (*Dactylus Glomerata*, *Holcus Lanatus* and some *Poa* species), 2.5% white clover (*Trifolium repens*) and 2.5% weeds on a

DM basis. The hay was chopped to approximately 3 to 5 cm using a New Holland 717 flail harvester, and the barley was dry rolled prior to feeding.

Measurements

Cows were manually stimulated to urinate 3 × each week before and after calving, and a sample of urine, from midstream, was collected in a 30-ml container. Within 30 min of collection, pH was measured, and 10 ml of sample was frozen for mineral, hydroxyproline, and creatinine (**Creat**) analysis. Urine minerals were determined by inductively coupled plasma emission spectrometry (**ICP-ES**), having been diluted with 1% nitric acid. Urine hydroxyproline was determined by a method developed by Parekh and Jung (1970), using a microplate reader (Biorad 550, BioRad, Hercules, CA). Urine creatinine was determined by a procedure modified from Bartels et al. (1972), using an autoanalyzer (Boehringer Mannheim Hitachi 911).

Blood from the jugular vein was collected from each cow twice weekly for the duration of the experiment. Two samples were taken at each sampling. One sample was taken using a 10-ml heparinized syringe. Any excess air was immediately removed along with approximately 1 ml of blood, and the syringe was stoppered and placed on ice. This aliquot was used to determine blood pH, pCO₂ and pO₂, within 20 min of sampling, using a blood gas analyzer (Geprufte Sichereit, Italy). The second sample, taken using a 10-ml heparinized evacuated tube, was centrifuged for 10 min at 1120 × g. Following centrifugation, the plasma was pipetted off and frozen awaiting mineral analysis. Plasma minerals were determined by ICP-ES having been digested using nitric acid-assisted microwave digestion (Milestone Application Laboratory, 1995).

Individual milk yields were recorded daily, postcalving (Alpro milk meter system, Alfa Laval, Sweden). Fat, protein, and lactose concentrations of milk were determined by Milkoscan (Foss Electric, Hillerød, Denmark) on individual p.m. and a.m. aliquot samples collected for 2 d each week.

Dry matter intake were measured for each cow daily, and representative samples of each feed offered and

Table 2. Quantities of salts (g) administered daily to alter the dietary cation-anion difference (mEq/100 g) of a pasture:pasture hay diet and balance the diets for Ca and Mg concentration.

Treatment	–12	+16	+51	+69
NaHCO ₃	0	0	90	300
MgSO ₄ ·7H ₂ O	120	175	0	0
MgCl ₂ ·6H ₂ O	100	0	0	0
CaCl ₂ ·2H ₂ O	140	0	0	0
MgO	0	10	40	40
CaCO ₃	100	160	160	160

refused were dried at 105°C to constant weight to determine DM content. Samples of all feeds were bulked on a weekly basis and dried at 65°C for 72 h, ground to pass through a 0.5-mm sieve (Christy Lab Mill, Ipswich, UK), and analyzed for in vitro DM digestibility (**DMD**), nitrogen, and macrominerals. Dry matter digestibility was determined by the method of Clarke et al. (1982). Metabolizable energy (**ME** – Mcal/kg DM) was calculated from DMD; $ME = ((DMD \times 0.17) - 2) / 4.186$; Standing Committee on Agriculture, 1990).

The nitrogen content was determined by a Kjeldahl method using Buchi Kjeldahl nitrogen apparatus, with CP being calculated from nitrogen ($CP = N \times 6.25$). Macrominerals were determined using X-ray spectroscopy (Hutton and Norrish, 1977; Norrish and Hutton, 1977).

Calculations and Statistical Analysis

Urinary minerals (Ca, Mg, Na, Cl, S) and hydroxyproline (**Hy**) are expressed as ratios to Creat concentration to overcome variations in urine volume among animals (Roche et al., 2002).

The strong ion difference (**SID**) is the difference, in milliequivalents, between certain cations and anions in body fluids and is calculated by subtracting the milliequivalents of Cl and S from the milliequivalents of Na and K, in either plasma or urine ($[SID_b]$ and $[SID_u]$, respectively).

The DCAD was calculated using the equation of Tucker et al. (1992) using the quantities of the salts administered and the measured mineral concentrations and DMI of all feeds.

$$DCAD = (Na + K) - (Cl + S) \text{ (mEq/100 g DM)}.$$

Blood bicarbonate concentration was calculated from blood pH and pCO_2 using the equations outlined by Tietz (1987).

For each cow, daily milk yields were averaged over a 17-d period beginning 4 d after calving. All data were analyzed using Genstat 5.4.1 (Genstat V, 1997) software, with cows as the experimental unit, according to a statistical model, which included effects for the four levels of treatment, linear, quadratic, and cubic trends with increasing DCAD and a covariate for previous season milk yield.

RESULTS

The DCAD eaten by cows on each treatment were –12, +16, +51, and +69 mEq/100 g precalving, and +33, +34, +29 and +35 mEq/100 g, postcalving.

The effect of treatment on precalving blood pH, bicarbonate concentration, pCO_2 , pO_2 and $[SID_b]$ and plasma

Mg and Ca concentrations are shown in Figure 1. The curvilinear decrease in blood pH with decreasing DCAD approached significance ($P < 0.1$), and cows receiving the lowest DCAD had a significantly lower blood pH than the other three treatments, which did not differ from each other. Precalving DCAD did not significantly affect blood pCO_2 , pO_2 , plasma Ca, or $[SID_b]$, although bicarbonate concentration declined linearly with declining DCAD. The linear increase ($P < 0.1$) in plasma Mg concentrations precalving with decreasing DCAD (Figure 1) is consistent with there being a real trend. There were no significant effects of treatment on any other plasma mineral concentrations precalving, and there were no residual effects of precalving treatment on subsequent post-calving blood pH, pCO_2 , pO_2 or $[SID_b]$ or plasma minerals (Table 3).

Treatment effects on urine variables are shown in Figure 2 and Table 4. Precalving urine pH and $[SID_u]$ decreased curvilinearly ($P < 0.001$ and 0.1, respectively) with decreasing DCAD, but when offered a common diet after calving, there were no residual effects of precalving treatment on either (Table 4). The curvilinear increase in Ca/Creat and Mg/Creat ($P < 0.1$ and 0.1, respectively) with decreasing DCAD (Figure 2) is consistent with there being a real trend. Cl/Creat and S/Creat also increased, precalving, as DCAD decreased ($P < 0.01$ and 0.05, respectively). Precalving Na/Creat increased curvilinearly with increasing DCAD ($P < 0.01$). There was no effect, either precalving or postcalving, of treatment on Hy/Creat. Postcalving, Ca/Creat was significantly higher in cows receiving a precalving DCAD of +16 mEq/100 g compared with cows that received a precalving DCAD of +69 mEq/100 g. There was no significant difference between the other treatments. There were no significant differences in the postcalving urinary concentrations of any of the other minerals (Table 4).

Precalving DMI did not decline until the day before calving, and varying the DCAD before calving did not significantly affect either pre- or postcalving total DMI or the yield or concentration of milk (Table 5). In the low DCAD treatment, two cows suffered clinical parturient paresis within 24 h of calving, while no cows in the other groups were clinically affected. There was no apparent trend in the effect of treatment on the duration of calving, although the cows receiving +69 mEq/100 g calved in significantly less ($P < 0.05$) time than cows receiving either +16 mEq/100 g or +52 mEq/100 g. Duration of calving ranged from 2 min to 60 min.

DISCUSSION

Based on the lower blood and urine pH, blood bicarbonate concentration and $[SID_u]$, with no obvious effect

Figure 1. The effect of precalving dietary cation-anion difference (DCAD; mEq/100 g) on blood pH, bicarbonate (HCO_3^- ; mEq/L), pCO_2 (Torr) and pO_2 (Torr), plasma Mg (mg/L) and Ca (mg/L) and the Strong Ion Difference of blood [SID]_b, precalving.

Table 3. Effects of precalving dietary cation-anion difference (DCAD) on blood pH, pCO₂ (Torr), pO₂ (Torr), bicarbonate (HCO₃⁻; mEq/L) and strong ion difference [SID_b] and plasma mineral concentration (mg/l) of dairy cows after calving.

DCAD – mEq/100 g	-12	+16	+51	+69	SEM
pH	7.400	7.402	7.393	7.402	0.008
HCO ₃ ⁻	27.2	27.6	26.3	26.7	0.6
pCO ₂	45.6	45.9	45.0	44.6	0.9
pO ₂	30.6	27.8	31.1	29.9	1.6
Ca	87.5	91.6	92.6	89.8	1.0
Mg	14.3	16.1	17.8	13.1	1.9
K	219	234	223	224	8
Na	2828	2758	3033	2808	172
Cl	3394	3442	3350	3767	166
S	1022	984	1003	1036	30
[SID _b]	-31.4	-31.9	-31.3	-46.6	7.5

on the partial pressure of either CO₂ or O₂ in blood, it was established that a reduction in the DCAD below +16 mEq/100 g caused a nonrespiratory systemic acidosis. This supports the theory of Stewart (1981, 1983), that an increased dietary concentration of Cl and S would reduce the pH of body fluids. Blood mineral balance is tightly controlled, and so [SID_b] was not significantly affected by treatment. This illustrates that more variables are involved in the control of systemic acid-base balance than [SID], as discussed by Stewart (1983) and Roche (1999). However, the significant decrease in urine pH and [SID_u] as blood pH decreased show urine parameters to be strong indicators of the systemic acid-base status of the animal, a point also considered by Roche et al. (2000). In the current experiment, blood and urine pH and urine Ca/Creat were not affected unless the DCAD was reduced below +16 mEq/100 g. This supports the conclusions of Beede (1992) and those discussed by Roche et al. (2000), that a negative DCAD is required to alter blood pH and, hence, prevent milk fever. This requirement for such a low DCAD to alter systemic pH and urine Ca/Creat (calcium absorption) and the lack of a change in these variables at higher DCAD was not known from previous research. These findings are of significant importance for pasture-based systems, where eucalcaemic benefits have been suggested from small changes in DCAD (reducing DCAD from +50 to +30 mEq/100 g). These results are also useful in determining the practicality of the DCAD concept as a means of preventing milk fever in pasture-based systems, as the volume of anionic salts required to reduce the DCAD sufficiently to alter calcium homeostasis would be large and, practically, very difficult to administer.

The concentration of Mg in plasma increased with decreasing DCAD, suggesting that metabolic acidosis increases the absorption of Mg. The curvilinear increase in the renal excretion of Mg (Mg/Creat) as DCAD decreased supports this. This was not evident from previ-

ous work. The concentration of Ca in urine (Ca/Creat), an indicator of the Ca status of the animal, is also dependent on the net acid excretion of the body. Precalving Ca/Creat increased with the decrease in DCAD, although there was no increase in plasma Ca concentration. While Schonewille et al. (1994) and Van Mosel et al. (1994) have reported a similar result, others have measured an increase in plasma Ca at calving (Block, 1984) and for a short period (1 or 2 d) postcalving. There is still debate as to the source of the additional Ca when such a diet is fed. Schonewille et al. (1994) measured an increase in absorption of Ca, but found that it only accounted for approximately 60% of the increased excretion of Ca. Another theoretical source of the excreted Ca may be the acidification of the medium surrounding bone, which can result in the release of Ca from the bone (Bushinsky et al., 1993). Many researchers have used the concentration of hydroxyproline in urine as an indicator of bone resorption (Robins, 1994; Russell, 1997). In this present work, no significant difference in Hy/Creat between treatments was found. This is supported by the work of Schonewille et al. (1994) and Van Mosel et al. (1994). Van Mosel et al. (1994) reported a decrease in bone accretion when DCAD was reduced but found no histomorphometric evidence of a change in bone resorption.

Chu et al. (1975) and Kim and Linkswiler (1979) found that an 'acidifying' diet, increased the urinary output of Ca due to an increased intestinal absorption, an increased glomerular filtration rate, and a reduced renal tubular reabsorption of Ca, and not an increased resorption of bone. On the other hand, Block (1984), Leclerc and Block (1989) and Goff et al. (1991) measured an increase in urinary Hy as a result of feeding an acidogenic diet, suggesting that a reduction in blood pH did increase bone resorption. Hy may be derived from several different tissue sources such as the breakdown of collagen during uterine involution (Kaidi et al., 1991). A more likely cause of the increased urinary Hy

Figure 2. The effect of precalving dietary cation-anion difference (DCAD – mEq/100 g) on urine pH, urinary mineral and hydroxyproline (Hy) to creatinine (creat) ratios and the strong ion difference of urine [SID]_u, precalving.

Table 4. The effect of precalving dietary cation-anion difference (DCAD) on urine pH and urinary mineral and hydroxyproline (Hy) to creatinine ratios (/Creat) after calving.

DCAD – mEq/100 g	–12	+16	+51	+69	SEM
Urine pH	7.76	8.04	8.02	7.90	0.11
Ca/Creat	0.17	0.16	0.14	0.09	0.06
Mg/Creat	0.78	0.61	1.02	0.44	0.21
K/Creat	35.6	41.6	47.4	37.5	4.7
Na/Creat	3.8	2.0	5.9	3.2	1.9
Cl/Creat	17.6	16.0	19.1	17.5	2.3
S/Creat	3.2	3.2	2.9	2.9	0.5
Hy/Creat	2.17	2.19	1.97	2.43	0.36

concentration in the prepartum cow is an increased rate of hydrolysis of extraskeletal connective tissue components (Galambos et al., 1976). Therefore, an effect of treatment on urine Hy concentration cannot be assumed to be solely due to bone resorption without additional measurements to support this.

Despite the low DCAD treatment group having a higher Ca/Creat, it was the only treatment to experience clinical hypocalcemia (two cows). This is in direct contrast to previous research findings where a reduction in DCAD before calving was associated with a reduced incidence of hypocalcemia (Block, 1984; Oetzel et al., 1988). Perennial ryegrass-dominant pastures in southeast Australia typically have 3 to 4.5 g Ca/kg DM, while the main supplement, barley, has less than 1 g of Ca/kg DM (Jacobs and Rigby, 1999). This diet is marginal in its ability to meet the Ca requirements of freshly-calved cows. However, animals fed a diet high in strong anions reabsorb less Ca in the renal tubules (Kim and Linkswiler, 1979) and, thus, probably require more dietary Ca to maintain a positive Ca balance (Linkswiler et al., 1981). In this experiment, therefore, a reduction in the DCAD offered to dry cows may have created a greater demand for Ca postcalving in order to prevent an accession of milk fever. This finding was

not evident from previous work. In essence, although postcalving Ca supplementation should be regarded as important in all periparturient cows, it appears to be more important if cows are fed a negative DCAD or have been supplemented with anionic salts prior to calving.

The consequences of substantially altering the DCAD on perennial ryegrass-based systems as are found in Australia, New Zealand, and Western Europe were not known from previous work.

CONCLUSIONS

In pasture-based dairying systems, blood pH and urine pH are reduced when a ration with a negative DCAD is fed. Achievement of a diet with a negative DCAD also resulted in an increased urinary output of Ca. No significant differences in milk production through altering the precalving DCAD were detected. Due to the high DCAD in the base diet offered to pasture-fed dairy cows and the requirement for a negative DCAD to increase Ca absorption, it is unlikely that the DCAD concept is a practical means of preventing milk fever in pasture-fed cows.

Table 5. The effect of precalving dietary cation-anion difference (DCAD) on 17-d mean milk yield and composition, DMI and duration of calving.

DCAD – mEq/100 g	–12	+16	+51	+69	SEM
Yield, kg/c/d					
Milk	26.3	29.5	29.8	28.4	1.9
Fat	1.10	1.24	1.22	1.16	0.13
Protein	0.79	0.99	0.93	0.96	0.05
Lactose	1.24	1.51	1.46	1.46	0.06
Concentration, %					
Fat	4.34	4.07	4.21	4.37	0.25
Protein	3.04	3.37	3.18	3.40	0.11
Lactose	4.77	5.09	4.92	5.14	0.13
Precalving total DMI, kg/c/d	7.9	9.3	8.7	8.9	0.5
Postcalving total DMI, kg/c/d	11.8	14.0	12.4	13.0	1.2
Time spent calving, min	23 ^{ab}	37 ^b	33 ^b	4 ^a	8.4

^{a,b}Means in a row with different superscripts differ significantly ($P < 0.05$).

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