

Metabolic Alterations Associated with an Attempt to Induce Laminitis in Dairy Calves

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ABSTRACT

The objective of this study was to investigate metabolic alterations in young ruminating calves associated with the sudden introduction of readily fermentable diets in an attempt to induce laminitis. Sixteen dairy bull calves, at 17 wk of age were fed equal amounts of one of four diets that contained either 71 or 81% total digestible nutrients (TDN) and 15 or 20% crude protein in a 2 × 2 factorial with time as a factor. Jugular blood and ruminal fluid were sampled, and hoof temperature was measured postfeeding at frequent intervals over a subsequent 2-d period. Hooves were examined for abnormalities and the orientation of the pedal bone radiographed prior to the experiment, 48 to 72 h into the experiment and at 3 and 7 mo later. Calves responded acutely to the 81% TDN diets by inappetence, stiffness, and diarrhea. Ruminal pH was lower and both D- and L-lactate concentrations were greater in the rumens of calves fed the 81% TDN diets. Total ruminal volatile fatty acid concentration decreased as pH declined. Whole blood L-lactate did not differ across treatments, but blood D-lactate increased in calves fed the 81% TDN diets, peaking at 32 h (7.2 mM). Hoof temperature responses could not be explained by dietary treatments. Laminitis was not detected despite the reduction of ruminal pH and a manyfold increase in blood D-lactate. (**Key words:** L(+)-lactate, D(-)-lactate, ruminal acidosis, laminitis)

Abbreviation key: HE = high energy diet; HP = high protein diet; LE = low energy diet; LP = low protein diet.

INTRODUCTION

Acute laminitis is an aseptic inflammation of highly vascularized corium in the hoof and is a major cause of lameness in dairy cattle. Lameness ranks third as a basis for culling dairy cows (6). Symptoms of acute lami-

nititis observed in fattening steers were reluctance to rise, arched back, and a lowered head; limb posture depended on which feet were the most painful (5). Deviation of the pedal bone (7) and its distal rotation may follow the acute phase of laminitis in cattle (5). Rotation of the distal bone is irreversible (8).

The true mechanistic cause of laminitis is not known, leading researchers to speculate that the disease is multifactorial (23). Nutritional factors are considered to be especially important to the etiology of the disease (23). Diets containing rapidly digestible carbohydrates and over 70% TDN have been recognized as one of the most important factors that leads to the onset of laminitis (8, 12, 21, 22). Diets containing 78.5% TDN resulted in toe and heel hemorrhages in calves and heel hemorrhages in yearling cattle (8). Bargai et al. (1) claimed that highly digestible protein was responsible for laminitis in calves.

Lactic acid is present in two isomeric forms, L(+)- and D(-)-lactate. Sudden introduction of diets with high concentrations of rapidly digestible carbohydrate can cause accumulation of lactic acid in the rumen and blood. Dunlop and Hammond (4) found an exponential decline of ruminally infused lactate (11.4 mM/kg of BW), with a half-life of 33 min.

Rate of absorption of L- and D-isomers from the rumen into the blood appears to be equal (3, 10). The increase in blood concentration of D-lactate observed after absorption are explained by low activity of a D-specific lactic dehydrogenase in the liver (10). Metabolic half-life for injected L-isomer was 22 min, in contrast to 108 min for the D-form of lactate (3).

By definition, chronic laminitis lasts for more than 6 wk (1). Changes caused by chronic laminitis may be seen as concavity of the dorsal hoof wall, irregular hoof shape, and hoof overgrowth. Flattening of the hooves relative to the walking surface, usually associated with hoof overgrowth, is also an indicator of chronic laminitis (7). Associated with laminitis is the formation of ridges on the dorsal wall (2, 7). Evidence of massive hemorrhage may be observed on the sole surface which indicates that some time previously hemorrhage may have interfered with normal horn synthesis. Complications

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Table 1. Ingredients and chemical composition of diets fed to dairy calves.

Composition	Diet ¹			
	LE+LP	LE+HP	HE+LP	HE+HP
	(% of DM)			
Ingredient				
Alfalfa silage	30.4	30.6	6.4	6.4
Corn silage	25.7	25.9	5.7	5.7
Corn meal	16.3	9.1	38.6	31.7
Barley, ground	16.3	9.1	38.6	31.7
Soybean meal, 48% CP	8.2	22.2	8.7	21.5
Molasses, cane	2.9	2.9	1.8	2.7
Salt	0.2	0.2	0.2	0.2
Chemical ²				
DM, %	61.5	61.5	80.6	80.8
CP	15.0	20.0	15.0	20.0
TDN	71.3	71.2	81.3	81.2
ADF	22.0	22.8	9.4	10.0
NDF	33.7	33.6	19.6	19.2
Ca	0.53	0.57	0.18	0.22
P	0.31	0.35	0.37	0.41

¹LE = Low energy (71% TDN); HE = high energy (81% TDN); LP = low protein (15% CP); and HP = high protein (20% CP).

²Estimated by Dair4 updated library file (20), except for silages, which were analyzed.

of chronic laminitis include white line disease and ulceration of the sole that is subject to bacterial invasion of vascular tissues of the hoof following a breakdown of hoof horn integrity.

The objective of this study was to investigate metabolic alterations caused by the sudden introduction of diets containing different concentrations of protein and fermentable carbohydrates that have been implicated in the development of laminitis.

MATERIALS AND METHODS

Calves, Diets, and Experimental Design

Sixteen Holstein bull calves, 17 wk old and 130 kg of BW, were fed an adjustment diet that contained 71% TDN and 15% CP (low energy and low protein diet; Table 1) for 2 wk in individual pens (1.2 × 2.0 m). Feed was offered twice daily at 0800 and 1600 h. To train calves to meal feeding, the calves were allowed 2 h to eat after each feeding. Water was always available. So that age would not be a variable, experiments were conducted in groups of four when calves reached the designated age.

The calves were randomly assigned to receive one of four treatments. Experimental diets contained two concentrations of energy, 71 (LE) or 81% TDN (HE) and two concentrations of CP, 15 (LP) or 20% (HP). Ingredients and chemical composition of the diets are shown in Table 1. On the last day that the adjustment diet was fed (d 14), calves were given a normal amount

of feed in the morning only, then deprived of feed until 0800 h the following morning. Diets were fed initially for 2 d at 0800 and 1600 h. Calves that became severely acidotic were fed a mixture of their assigned diet plus hay for up to 1 wk. Beyond that, only the assigned diets were fed for 2 mo. Orts were removed each day at 2000 h. This study was reviewed and approved by the University Animal Care Committee.

Samplings and Measurements

Just before feeding of the assigned diet on the first day, blood and ruminal samples were collected, and hoof temperature was measured at 0800 h (0 h) and at 4, 8, 12, 16, 24, 28, 32, 36, and 48 h after feeding. Blood was collected into Vacutainer tubes (Becton-Dickinson, Rutherford, NJ). Two types of tubes were used: one contained 8.0 mg of potassium oxalate and 10 mg of sodium fluoride and was used for lactate measurements, and the other contained 100 USP units of sodium heparin and was used for VFA analysis. Upon sampling, the tubes were submerged in ice and taken to the laboratory. To prepare for lactate measurement, 1 ml of whole blood was mixed with 2 ml of cold 8% perchloric acid. After centrifugation (IEC Centra 8R centrifuge; International Equipment Company, Needham Heights, MA) at 3000 × *g* for 15 min at 4°C, supernatants were stored in duplicate at -20°C until analyzed.

Ruminal fluid was aspirated via an esophageal tube fitted with a stainless steel strainer. Of the 100 ml collected, approximately 10 ml was retained as a sample and placed in ice for transport to the laboratory. The pH was measured within 30 min with an Accumet pH meter (Fisher Scientific, Raleigh, NC) equipped with a Ross Sure-Flow combined pH electrode (Orion Research Inc., Boston, MA). One milliliter of ruminal fluid was deproteinized with 2 ml of 8% perchloric acid. After centrifugation, as described for blood, the supernatant was stored at -20°C until lactate analysis. The remaining ruminal fluid was split in two subsamples, 2.5 ml each. For each tube, 0.5 ml of 25% H₃PO₄ was added, and 0.5 ml of 30 mM isocaproic acid (internal standard) was added to one tube for VFA analysis; both were stored at -20°C.

The left rear leg of each calf was radiographed twice from a lateral position to observe the angle of the pedal bone, using a portable device (MINIXRAY, Inc., Northbrook, IL). This procedure was completed during the period of feed deprivation and within 24 h after the last 48-h sampling. The dorsal hoof wall on the lateral claw of the rear foot was cleaned, and the surface temperature was measured with a digital infrared scanner (model D501; Exergen Corp., Newton, MA), at each sampling time (0 through 48 h). Hooves were radio-

graphed again approximately 3 and 7 mo later. At the same time, hooves were examined for presence of hardship groove, hoofwall separation, previous hemorrhage of the sole, sole or heel erosion, and double sole.

Analyses

Whole blood was analyzed for L(+)- and D(-)-lactate, and ruminal fluid was assayed for lactates and VFA. All samples were analyzed in duplicate. Chemicals were purchased from Sigma Chemical Company (St. Louis, MO). Absorbencies were measured with a Titertek Multiskan MCC/340 microplate reader (EFLAB, Finland) equipped with Skan Soft program.

Deproteinized supernatants from the whole blood and ruminal fluid were analyzed for L(+)- and D(-)-lactate by the enzymatic procedure outlined in the Sigma Technical Bulletin (No. 826-UV, Sigma, St. Louis, MO). L-lactate was analyzed with L-lactate dehydrogenase (EC 1.1.1.2.7) from beef heart, and D-lactate was analyzed with D-lactate dehydrogenase (EC 1.1.1.2.8) from *Lactobacillus leichmanii*. The original procedure was modified by reducing the total reacting volume by 10-fold, which accommodated a 96-well microplate. Prior to analysis, 6.1 ml of a reactant solution was prepared containing 2.0 ml of glycine buffer, 4.0 ml of water, 0.10 ml of appropriate lactate dehydrogenase, and 10.0 mg of NAD. To each microplate, 0.29 ml of the reactant solution plus 10 μ L of the deproteinized iced sample was added. Lactate concentration was determined from standard curves prepared on each plate. The reactant mixture was incubated for about 15 min at 37°C, and absorbance was measured at a wavelength of 340 nm. All samples were analyzed within 48 h of storage at -20°C.

Ruminal VFA were analyzed by injecting 0.5 μ l of sample into a Varian Vista 6000 gas chromatograph (Varian Instruments, Palo Alto, CA) equipped with a flame ionization detector, a Varian Vista 4270 integrator (Varian Instruments), and a 180 \times 0.6 cm o.d. and 2-mm i.d. glass column (Supelco Inc., Bellefonte, PA) packed with GP 10% SP-1200/1% H₃PO₄ on 80/100 Chromosorb WAW. The analysis was isothermal; temperatures were 115°C for the column, 170°C for the injector, and 180°C for the detector. Nitrogen, used as a carrier, had a flow rate of 80 ml/min, and hydrogen and air as detector gases had flow rates of 40 and 60 ml/min, respectively.

Statistical Analyses

Data were analyzed by ANOVA for a two-factor experiment (energy and protein) with repeated measurements on time using the repeated option of the GLM

procedure of SAS (16). Interaction of diet and time was included.

$$y_{ijk} = u + \alpha_i + \pi_{j(i)} + \beta_k + \alpha\beta_{ik} + \varepsilon_{ijk}$$

where y_{ijk} , u , α_i , $\pi_{j(i)}$, β_k , $\alpha\beta_{ik}$ are effects corresponding to observations for dependent variables, mean, main effects for diet, calf (diet), time, the interaction of treatment and time, and residual error, respectively.

All results are shown as least squares means and pooled standard errors of the mean. Contrasts for treatment means included effects caused by energy (LE vs. HE) and protein (LP vs. HP). Time within each treatment was tested for linear and quadratic response.

RESULTS

Because feed had been withheld for 22 h, eager consumption of feed by all calves was observed at 0800 h when the assigned diets were initially offered, resulting in high DMI (Table 2). When compared with their mean daily DMI during the adjustment period, calves fed HE+LP, HE+HP, LE+LP, and LE+HP, increased consumption by 61, 49, 20, and 8%, respectively. The following day, however, calves offered the HE diets refused to eat (Table 2).

Symptoms of anorexia and depression in calves fed HE+LP and HE+HP occurred as early as 12 h after the feed was offered, indicating metabolic dysfunction. Discomfort, lethargy, stiffness, muscular tremor, and diarrhea were observed. Affected calves stood motionless, apparently unaware of their environment for a period of time; finally these calves became recumbent and unable to rise, usually between 16 to 24 h. Six of eight calves fed HE+LP and HE+HP were recumbent by 24 h. Two calves, one each fed HE+LP and HE+HP consumed less feed than others (3.3 and 3.4 kg, respectively) but demonstrated symptoms of mild diarrhea and stiffness. One calf fed LE+HP showed similar behavior after consuming 5.8 kg of the diet. At 48 h, affected calves fed HE+LP and HE+HP were medicated with an oral balanced electrolyte solution containing alkalizing agents (Entrolyte; Smith Kline Beecham Animal Health, West Chester, PA). In general, this therapy proved beneficial for speeding recovery of affected calves.

Ruminal fluid changed color, consistency, and odor in HE calves beginning at 8 h. The fluid became a yellow, watery liquid with a progressively sour odor. This change coincided with a quadratic decline ($P < 0.01$) in ruminal pH (Figure 1). Ruminal pH declined most in animals fed HE diets ($P < 0.01$). Responses for HE+LP and HE+HP were nearly identical between 12 and 28 h; thereafter pH recovered faster in HE+HP than in

Table 2. Dry matter consumption of calves fed diets varying in TDN and CP.

Day ²	Diet ¹				SEM
	LE+LP	LE+HP	HE+LP	HE+HP	
	(% of BW)				
1	3.93 ^{bc}	3.25 ^c	5.33 ^a	4.30 ^b	0.24
2	2.95 ^a	2.38 ^a	0.00 ^b	0.00 ^b	0.29

^{a,b,c}Means within a row lacking a common superscript differ ($P \leq 0.05$).

¹LE = Low energy (71% TDN); HE = high energy (81% TDN); LP = low protein (15% CP); and HP = high protein (20% CP).

²Day after abrupt change to experimental diets.

HE+LP. Dietary energy explained most of the observed decline in pH.

L-Lactate in ruminal fluid was very low in all calves at 0 h (Figure 2). L-Lactate increased remarkably in animals fed HE diets ($P < 0.01$) and generally showed a quadratic response over time ($P < 0.01$). At 12 h, concentrations of L-lactate were nearly equal in HE treatments.

D-Lactate in ruminal fluid followed a pattern similar to L-lactate but peaked earlier and at lower concentrations (Figure 3). Differences in dietary response were significant ($P < 0.01$) and again showed a quadratic time effect ($P < 0.01$). The increase in HE+LP paralleled the increase in HE+HP but was delayed for approximately 4 h.

The ratio of L-lactate to D-lactate in the rumen in HE diets varied according to rates of production and absorption (data not shown), but were not different ($P > 0.05$) with respect to protein amount. Small differences were observed at both ends of the sampling window.

However, since the absolute amounts of the isomers at these points were very small, they are not considered physiologically important.

Ruminal VFA concentration doubled within 4 h in calves fed all diets (Figure 4), followed by an overall decline ($P < 0.05$) in calves that differed among diets. The decline was not explained by linear or quadratic functions. However, significant differences by diet are distinctly shown in Figure 4 with the wide separation of concentrations of VFA for the calves fed HE versus LE.

By contrast, in those calves fed the HE diets, ruminal lactate concentration continued to increase and exceeded VFA concentrations by 16 h postfeeding. By 36 to 48 h, ruminal lactate had decreased to near initial base concentrations.

L-Lactate in blood remained fairly insensitive ($P > 0.05$) to changes in L-lactate in the rumen (Figure 5). Major peaks in the blood occurred at 4 h after each meal and appeared to be a normal response to feeding. D-Lactate in blood, however, followed an entirely differ-

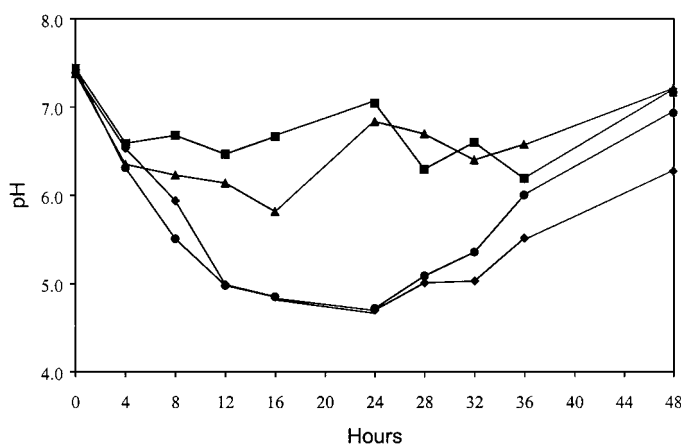


Figure 1. Serial response of ruminal pH in calves after engorgement of diets differing in TDN and CP content. Low energy (LE)+low protein (LP) = 71% TDN, 15% CP (■); LE + high protein (HP) = 71% TDN, 20% CP (▲); high energy (HE) + LP = 81% TDN, 15% CP (◆), HE + HP = 81% TDN, 20% CP (●). Feed was consumed beginning at 0 h. SEM = 0.26.

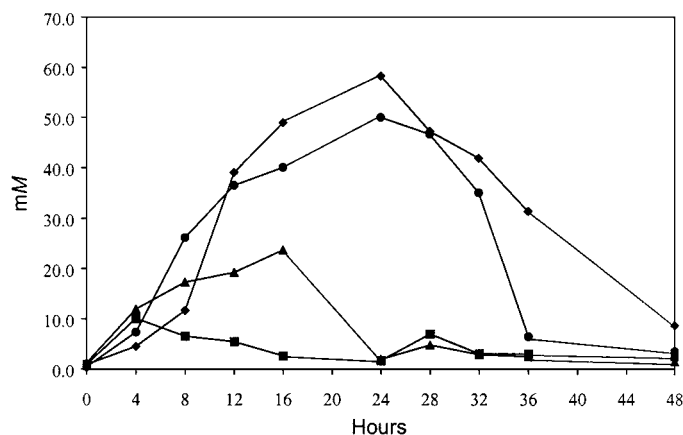


Figure 2. Serial response of ruminal L-lactate concentration in calves after engorgement of diets differing in TDN and CP content. Low energy (LE)+low protein (LP) = 71% TDN, 15% CP (■); LE + high protein (HP) = 71% TDN, 20% CP (▲); high energy (HE) + LP = 81% TDN, 15% CP (◆), HE+HP = 81% TDN, 20% CP (●). Feed was consumed beginning at 0 h. SEM = 7.6 mM.

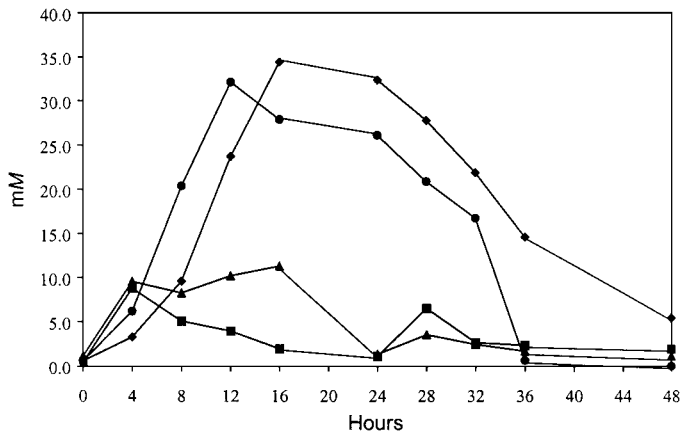


Figure 3. Serial response of ruminal D-lactate concentration in calves after engorgement of diets differing in TDN and CP content. Low energy (LE) + low protein (LP) = 71% TDN, 15% CP (■); LE + high protein (HP) = 71% TDN, 20% CP (▲); high energy (HE) + LP = 81% TDN, 15% CP (◆); HE + HP = 81% TDN, 20% CP (●). Feed was consumed beginning at 0 h. SEM = 5.7 mM.

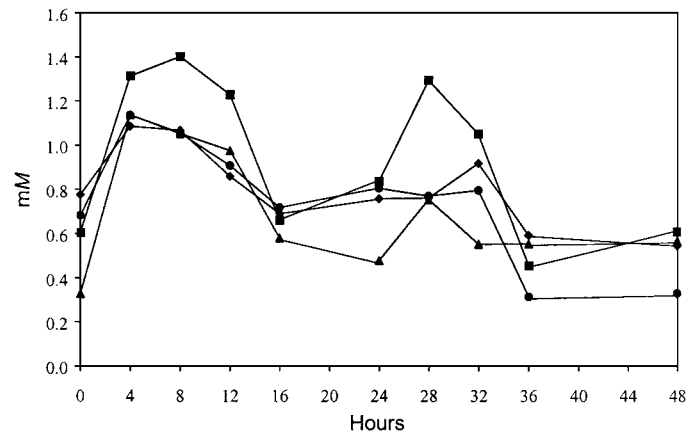


Figure 5. Serial response of blood L-lactate in calves after engorgement of diets differing in TDN and CP content. Low energy (LE) + low protein (LP) = 71% TDN, 15% CP (■); LE + high protein (HP) = 71% TDN, 20% CP (▲); high energy (HE) + LP = 81% TDN, 15% CP (◆); HE + HP = 81% TDN, 20% CP (●). Feed was consumed beginning at 0 h. SEM = 0.12 mM.

ent pattern than L-lactate (Figure 6) and the response was due to HE diets ($P < 0.01$). From negligible amounts, measured at 0 and 4 h, D-lactate increased in calves fed HE diets beginning by 8 h and continued to 28 and 32 h. Both linear and quadratic components were significant ($P < 0.05$). Blood D-lactate continued to increase for 20 h after having peaked in the rumen.

Hoof temperature was quite variable, generally lower in HE treatments, but with great variation. Biphasic changes coincided with changes in daily temperature and could not be explained by dietary treatments. Radiographs did not reveal any significant changes in hoof

angle that could be associated with dietary treatments (data not shown). Hooves were inspected 3 and 7 mo after engorgement for any deviant morphology. At 3 mo, the hoof inspection indicated possible hoof damage during feed engorgement (Table 3). Separation of the hoof wall, erosion of the sole and the heel, and double sole were found. A hardship groove was present at the dorsal hoof wall, which resulted in a concavity of the dorsal wall, similar to that described by Ossent et al. (15). The groove was positioned between 2 and 3 cm from the coronary band, which indicated its relationship with time of engorgement.

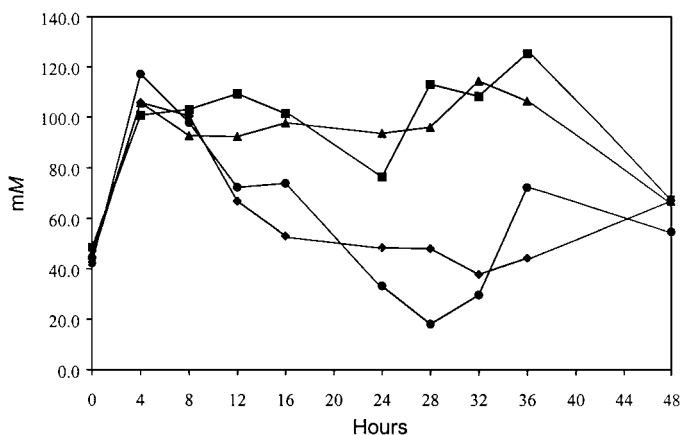


Figure 4. Serial response of ruminal total VFA concentration in calves after engorgement of diets differing in TDN and CP content. Low energy (LE) + low protein (LP) = 71% TDN, 15% CP (■); LE + high protein (HP) = 71% TDN, 20% CP (▲); high energy (HE) + LP = 81% TDN, 15% CP (◆); HE + HP = 81% TDN, 20% CP (●). Feed was consumed beginning at 0 h. SEM = 14.9 mM.

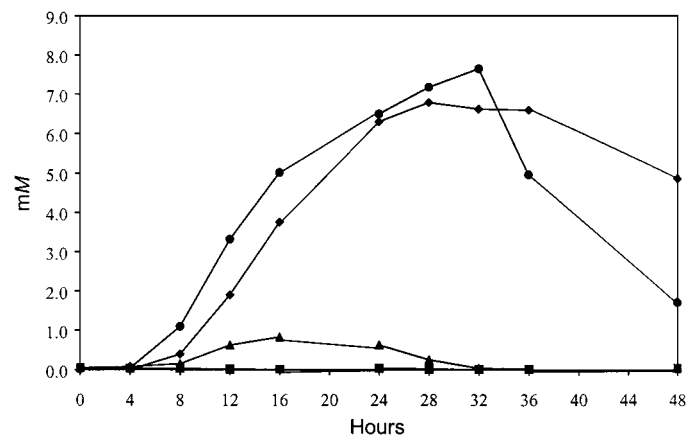


Figure 6. Serial response of blood D-lactate in calves after engorgement of diets differing in TDN and CP content. Low energy (LE) + low protein (LP) = 71% TDN, 15% CP (■); LE + high protein (HP) = 71% TDN, 20% CP (▲); high energy (HE) + LP = 81% TDN, 15% CP (◆); HE + HP = 81% TDN, 20% CP (●). Feed was consumed beginning at 0 h. SEM = 0.81 mM.

Table 3. Hoof characteristics of calves at 3 and 7 mo after consuming diets varying in CP and TDN.¹

Diet ³	Observed morphology ²											
	Hardship Groove		Hoof wall separation		Hemorrhage of the sole		Erosion of the sole		Erosion of the heel		Double sole	
	3 mo	7 mo	3 mo	7 mo	3 mo	7 mo	3 mo	7 mo	3 mo	7 mo	3 mo	7 mo
LE+LP	4	4	1	0	0	0	1	0	1	0	1	0
LE+HP	3	3	0	0	0	1	1	0	1	0	0	0
HE+LP	4	2	3	0	0	0	1	0	1	0	0	0
HE+HP	4	2	1	0	0	0	2	1	2	1	0	0

¹Four calves per diet.

²Number of animals with the observed morphology.

³LE = Low energy (71% TDN); HE = high energy (81% TDN); LP = low protein (15% CP); and HP = high protein (20% CP).

At the second inspection, an improvement in hoof condition was observed. The hardship groove had extended further distally and was positioned between 4 and 6 cm from the coronary band. In some calves, the groove had disappeared because of wear. Beyond that point the dorsal hoof wall appeared to be free of any irregularities. Earlier abnormalities in the hoof horn were completely corrected with regrowth of a new horn.

DISCUSSION

Sudden inclusion of readily fermentable carbohydrate into the diet can result in lactic acidosis, which through a sequence of events may result in laminitis. However, the mechanism by which diet causes laminitis is incompletely understood. Attempts to induce lactic acidosis based on engorgement of large amounts of readily digestible carbohydrates have been reported (14, 18, 19, 21). Feed withdrawal for different lengths of time (24 to 96 h) to create hunger and increase intake was employed. Because an extended period of feed withdrawal could alter the microbial population in the rumen, we limited feed deprivation to 22 h. In some studies, calves were engorged by placing an allotment of feed into the rumen via a permanent fistula. Concentrate intake equaled 17 g/kg of BW for calves fed LE+LP, 15 g/kg for calves fed LE+HP, 50 g/kg for calves fed HE+LP, and 42 g/kg for calves fed HE+HP. Consumption rates for calves fed HE+LP and HE+HP are comparable with the engorgement rates used in other studies (4, 13, 21).

The first observed symptoms of illness were behavioral changes that were displayed during sampling. Nonaffected calves resisted sampling, and ill calves became lethargic, perhaps because of approaching acidotic coma and neural depression. Respiration rate increased in affected calves. Ruminal fluid pH in engorged calves decreased significantly by 8 h after feeding and at 16 h was below 5.0. The pattern and degree of changes in ruminal fluid pH were similar to those ob-

tained by Harmon et al. (9) who infused a glucose solution into the rumen of steers. In that study pH declined below 5.0 at 12 h and remained less than 5.0 for 30 h. In the present study, pH was less than 5.0 at 12 h and remained less than 5.0 for an additional 12 h. Nagaraja et al. (13) measured a pH of 4.4 at 12 h in engorged sheep and cattle.

Sharp increases in the concentrations of L-lactate in the rumen have been followed by either a further gradual increase (9) or decrease (4, 19, 21). In the present study, a severalfold increase could have occurred in the 8-h interval between 16 and 24 h. The maximal concentrations of ruminal L-lactate obtained were somewhat lower than those measured by Harmon et al. (9) (61 mM) and Wilson et al. (24) (63 mM) but were higher than those measured by Rumsey (18) (14 mM). Peak of L-lactate (161 mM) measured by Dunlop and Hammond (4) occurred at 6 h in an engorged steer. Slyter and Rumsey (19) measured the increase of L-lactate in an engorged steer at 6 and 24 h (50 and 90 mM, respectively).

D-Lactate concentrations in the rumen was similar in pattern over time, but not in quantity, to L-lactate. A similar pattern was observed by Rumsey (18). Dunlop and Hammond (4) and Harmon et al. (9) determined that D-lactate at 30 h was 90 and 47 mM, respectively. In the present study, a sharp decrease of D-lactate occurred at 36 and 48 h in calves fed HE+HP and HE+LP, respectively. Differences in quantity might be due to different rates of either synthesis or removal from the rumen. In general, the rate of absorption for both isomers is thought to be the same (4). However, others (9) measured a severalfold increase in D-lactate absorption and little increase for L-lactate. Racemization of D-lactate to L-lactate in the rumen is a less likely explanation because this reaction was not very extensive in the rumen (19).

The ratio of ruminal fluid L-lactate to D-lactate observed in calves fed HE+LP was unaffected by change

in dietary treatments. Other authors reported either increases (9, 11, 18, 24) or decreases (4) in the proportion of D-lactate. Huntington and Britton (11) observed a significant shift in the proportion between the two isomers in the rumen at d 35 (5 d after receiving a 90% concentrate diet), from predominantly L-lactate to almost all D-lactate.

In general, ruminal VFA declined similar to ruminal pH. Initially, an increase of VFA coincided with a decrease in ruminal fluid pH. Later, as lactate accumulated, both VFA and pH declined. The observed changes in total VFA in this study agree with the results of Wilson et al. (24) and Slyter and Rumsey (19) and disagree with the results of Rumsey (18). Others (9, 14) have reported an overall decline of total VFA in the rumen of animals with metabolic disturbances. Moreover, Slyter and Rumsey (19) showed a dramatic reduction of total VFA in engorged steers compared with that in normally fed steers. Rumsey (18), however, measured a steady increase of total VFA concentration in steers, from a baseline created by feed deprivation for 96 h. Acetate decreased less than propionate in this study. In a few cases, propionate was nearly absent at certain sampling times.

Amounts of L-lactate in blood were not related to diet changes and did not correspond directly with ruminal L-lactate. Harmon et al. (9) observed a slight increase in L-lactate in blood that coincided with the lowest ruminal pH. Nagaraja et al. (13) measured a rapid accumulation of L-lactate in blood beginning at 15 h, and a transitory increase at 28 h (4). In contrast, others (21) observed a decrease in L-lactate in plasma of engorged steers when compared with that in plasma of normal steers. L-Lactate may have been absorbed into the blood at a slower rate than was D-lactate or L-lactate was metabolized more rapidly. Prins et al. (17), measured faster metabolism for L-lactate than for D-lactate in the ruminal epithelium.

When pH declined to less than 5.0, a rapid accumulation of D-lactate in blood occurred. Below pH 5.0, lactate may be absorbed in undissociated form, and because of slow metabolism, concentration in blood may increase (5, 18). Dunlop and Hammond (4) and Prins et al. (17) have proposed that D-lactate may accumulate in blood in the presence of hemoconcentration. Researchers have usually measured L-lactate in the blood of animals affected by acute laminitis but have neglected D-lactate. D-Lactate has reached peak concentrations of 3 to 8.4 mM in engorged cattle (4, 9, 13). No data have reported the measurement of D-lactate in blood during laminitis.

In this study ruminal fluid and blood changes observed were primarily induced by HE. Dietary protein in LE+HP may have modified some parameters in the

first 16 to 24 h after feeding, but may be due to the diet fermentability and not protein per se.

Inspecting hooves at 3 and 7 mo after the primary insult would allow sufficient time to observe hoof hemorrhages that occurred previously (15). Hardship groove, double sole, separation of the hoof wall, erosion of the sole, and the heel and sole hemorrhage are symptoms of laminitis. Few signs of chronic laminitis were observed on second inspection, suggesting full recovery. Based on this study, lactic acidosis induced by gorging did not result in any permanent hoof abnormalities.

CONCLUSIONS

Gorge consumption of HE diets resulted in severe acute lactic acidosis in bull calves, resulting in anorexia, lethargy, diarrhea, and stiffness. Ruminal fluid pH declined below 5.0 within 12 h of feeding as a result of a rapid fermentation. Both isomers of lactic acid increased manifold and peaked in the rumen at 24 h. Lactate increased modestly post-feeding in calves fed LE diets. Concentrations of L-lactate were always greater than concentration of D-lactate in the rumen. Ruminal VFA declined in acidotic calves, which indicated a disruption of normal ruminal activity. L-Lactate in blood did not differ among diets, but D-lactate in blood accumulated in acidotic calves peaking between 28 and 32 h. Acute laminitis was not observed. Hooves, examined 3 mo after acute acidosis showed limited evidence of laminitis. On examination at 7 mo, no evidence of damage was found. This study shows that acute ruminal acidosis and extremely elevated blood D-lactate does not necessarily result in acute laminitis in cattle.

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