

Evaluation of models of acute and subacute acidosis on dry matter intake, ruminal fermentation, blood chemistry, and endocrine profiles of beef steers¹

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ABSTRACT: Crossbred steers (n = 20; 316 ± 4 kg BW), each fitted with a ruminal cannula, were used to evaluate the effects of acute acidosis (AA) and subacute acidosis (SA) on DMI, ruminal fermentation, blood chemistry, and endocrine profiles. Animals were blocked by BW and assigned to treatments including 1) intraruminal (via cannula) steam-flaked corn (3% of BW; AA); 2) intraruminal dry-rolled wheat:dry-rolled corn (50:50; 1.5% of BW; SA); 3) offering forage-adapted steers ad libitum access to a 50% concentrate diet (AA control; AC); and 4) offering 50% concentrate diet-adapted steers ad libitum access to a 50% concentrate diet (SA control; SC). Samples of ruminal fluid and whole blood were collected on the day of the challenge (d 0) and 3, 7, 10, and 14 d after the challenge. Daily DMI responded quadratically ($P < 0.01$) through d 7 for AA and SA steers and increased linearly ($P < 0.01$) for AC steers. Dry matter intake by AA steers reached a nadir (< 3 kg/d) on d 3 and gradually increased to a level similar to other treatments (7 kg/d) by d 10, whereas DMI by SA steers increased through d 3. Blood pH, bicarbonate, base excess, and total CO₂ were decreased ($P < 0.03$) for AA steers and increased ($P < 0.03$) for SC

steers through d 7. Ruminal pH decreased quadratically ($P < 0.01$) in AA and AC steers and increased ($P = 0.01$) in SA steers through d 7. Ruminal total lactate concentration and osmolality responded quadratically ($P < 0.01$) for AA and AC steers. Ruminal total lactate peaked on d 3 for AA steers and on d 0 for AC and decreased to basal concentrations by d 7. Plasma NEFA concentration increased ($P < 0.04$) on d 3 and 7 for AA steers. Serum Na decreased ($P < 0.05$) on d 0 for AA and SA steers and on d 7 and 14 for AA steers. Serum P decreased ($P = 0.01$) for AA steers through d 7 and decreased quadratically ($P = 0.01$) for AC steers through d 7. Serum albumin and cholesterol decreased ($P < 0.02$) for AA and AC steers through d 7. Area under the GH curve decreased ($P = 0.02$) for AA and AC steers through d 7. Considerable variation was evident in the ability of an animal to cope with a carbohydrate challenge. Results of data modeling generally suggest that serum amylase activity, cholesterol and potassium concentrations, and plasma NEFA concentrations were useful in distinguishing between steers classified as experiencing subacute acidosis or not affected by a carbohydrate challenge.

Key Words: Acidosis, Cattle, Hydrocortisone, Metabolites, Somatotropin

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Introduction

The influence of ruminal acidosis on ruminal microbiology and metabolite production has received considerable attention, but less is known about systemic manifestations that arise from ruminal acidosis. In grain-

challenged buffalo, acute ruminal acidosis results in ruminal ulceration, atrophy of secretory epithelium of pancreatic acini, degranulation and atrophy of pancreatic β -cells, depletion of glycogen in vacuolated hepatocytes, hypertrophy and(or) degranulation of catecholamine-secreting cells in the adrenal medulla, and emphysema of the lungs (Randhawa et al., 1980, 1981, 1982). Similar pathology has been described for cattle (Jensen et al., 1954; Ahrens, 1967; Dirksen, 1970). Hence, ruminal acidosis likely involves changes in both blood chemical constituents and endocrine profiles.

Acute ruminal acidosis in sheep is generally accompanied by decreased plasma or serum Ca, K, and Mg (Irwin et al., 1979; Patra et al., 1993) from 58 to 120 h after carbohydrate loading, which seems to result from

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increased urinary mineral excretion (Harmon and Britton, 1983). The syndrome of acidosis has been associated with endocrine involvement (Bruce and Huber, 1973; Randhawa et al., 1980; Brown et al., 1999), alterations in serum enzyme activity (Lal et al., 1991; Brown et al., 1999), creatinine, total protein (Brown et al., 1999), and decreased ruminal organic acid absorption (Krehbiel et al., 1995). Previous experiments have generally focused on short-term (< 5 d) changes; therefore, our objectives were 1) to comprehensively characterize both short- and longer-term effects of acute and subacute acidosis on ruminal fermentation, blood chemistry, and endocrine profiles of beef steers and 2) to model these comprehensive data to identify variables that might reliably indicate the severity of acidosis.

Materials and Methods

Animals and Diets. Twenty Hereford × Angus steers (initial BW = 316 ± 4 kg) were each fitted with a ruminal cannula under local anesthetic (Lidocaine HCl; American Regent Laboratories, Shirley, NY) using the one-stage technique of Dougherty (1955). Animals were handled and cared for according to a protocol approved by the Pharmacia and UpJohn Corporate Animal Welfare Committee. Antibiotic therapy using penicillin/dihydrostreptomycin (Combiotic; Pfizer Animal Health, New York) was initiated on the day of surgery and continued for 5 d. At the time of surgery and 6 wk later, steers received Fulvicin U/F boluses (Schering-Plough Animal Health, Madison, NJ) to treat ringworm. Animals were allowed a 6-wk recovery period and were placed in individual indoor tie stalls on d -53. Facilities were monitored to maintain a 16:8 h light:dark cycle, and indoor temperature was ambient (18 to 20°C). Animals were allowed ad libitum access to drinking water at all times.

Steers were weighed on d -40 and blocked by BW into five blocks. Blocks were assigned randomly to groups of adjacent tie stalls, and steers were assigned randomly to stalls and treatments within each block. Treatments were subacute acidosis (SA), subacute acidosis control (SC), acute acidosis (AA), and acute acidosis control (AC; Hibbard et al., 1995). Briefly, SA and SC steers were adapted to a 50% concentrate diet (Table 1) from d -31 to d -3, which was designated as the preconditioning period. Level of DMI was restricted to 1.7% of BW/d, and feed was offered in two equal meals at 0700 and 1500. On d -2, feed was offered to SA and SC steers at 0900 and 2300, whereas feed was not offered on d -1. At 0700 on d 0, SA steers were intraruminally dosed with a challenge ration (Table 1) at the rate of 1.5% of BW, whereas feed was withheld from SC steers until d 1. The preconditioning period for AA and AC steers occurred from d -31 to d -4, during which steers were offered ad libitum access to coarsely chopped grass hay (7.1% CP, 45.0% ADF). On d -3, AA and AC cattle were fed 0.5% of BW of chopped grass hay at both 0700 and 1500. One meal of grass hay (0.5% of BW) was offered

Table 1. Ingredient composition of the basal diet and subacute acidosis challenge diet and chemical composition of the basal diet

Item	% of DM
Basal diet ingredient composition	
Corn silage	60.0
Alfalfa hay	20.0
Rolled corn	11.9
Soybean meal (49% CP)	6.8
Premix ^a	1.3
Basal diet chemical composition	
CP	13.0
ADF	20.9
Ca	0.47
P	0.34
K	1.46
Challenge diet ingredient composition	
Soft white, rolled wheat	50.0
Rolled corn	47.6
Premix ^b	2.4

^aPremix contained: dicalcium phosphate, 29.24%; NaCl, 21.97%; KCl, 21.97%; tallow, 13.97%; Se (0.2 mg/g), 7.27%; trace mineral premix, 2.88%; vitamin A (30,000 IU/g), 0.3%; vitamin D (15,000 IU/g), 0.3%; and vitamin E (275,000 IU/kg), 2.1%.

^bPremix contained: limestone, 37.5%; NaCl, 35.42%; tallow, 22.72%; Se (0.2 mg/g), 0.63%; trace mineral premix, 2.06%; vitamin A (30,000 IU/g), 0.83%; vitamin D (15,000 IU/g), 0.21%; and vitamin E (275,000 IU/kg), 0.63%.

on d -2, whereas feed was withheld on d -1 for AA and AC steers. The challenge ration for AA steers consisted of steam-flaked corn (59.3% gelatinization [Xiong et al., 1990]; Ag Analysis, Hereford, TX) administered intraruminally in four equal parts (0.75% of BW) at hourly intervals on d 0; the total dose was equivalent to 3.0% of BW. Steam-flaked corn was hydrated with 39°C tap water (1.5% vol/wt) for 1.5 h before dosing. As with the SC steers, feed was withheld from AC steers on d 0. All steers were allowed ad libitum access to a 50% concentrate diet (Table 1) from d 1 to 14. Feed was offered twice daily (0730 and 1630), and orts were weighed and discarded daily.

Laboratory Methods. Animals were fitted with jugular catheters on d -33 and -4 to facilitate collection of venous whole blood. Rectal temperature measurements, ruminal fluid, and venous whole blood were sampled on two occasions during the preconditioning period (d -32 and -3) to determine whether covariate adjustment for response variables was appropriate. Similar samples also were collected on d 0, 1, 3, 4, 7, 10, and 14. Within a day, sample collection times and response variables measured included the following: daily DMI; rectal temperature at 3, 6, and 9 h; ruminal pH, osmolality, and D(-)- and L(+)-lactate at 1, 3, 5, 7, and 9 h; blood packed cell volume hourly at 1 through 9 h; blood pH, pCO₂, pO₂, HCO₃⁻, total CO₂, and base excess at 1, 3, 5, 7, and 9 h; plasma NEFA, glucose, and L(+)-lactate hourly at 1 through 9 h; serum amylase, cholesterol, glutamine-oxaloacetate transaminase (GOT), glutamine-pyruvate transaminase (GPT), alkaline

phosphatase, lactate dehydrogenase, albumin, urea, Na, Cl, K, Ca, P, triiodothyronine, and thyroxine at 1, 3, 5, and 7 h; serum cortisol at 20-min intervals from 1 through 3 h and from 6 through 8 h; serum GH and insulin at 20-min intervals from 1 through 8 h; and serum IGF-I at 1, 3, 5, and 7 h.

Rectal temperature was measured using a mercury thermometer. Ruminal pH was determined immediately using a pH meter (No. ϕ 32, Beckman Instruments, Palo Alto, CA) equipped with an immersible probe (No. 450C, Sensorex Corp., Westminster, CA) on triplicate samples of ruminal contents (approximately 50 mL) obtained from the anterior, middle, and posterior regions of the rumen. Ruminal contents were strained through four layers of cheesecloth, and fluid was transferred into 15- × 100-mm polypropylene centrifuge tubes and transported to the laboratory. Aliquots (1 mL) were transferred to microcentrifuge tubes and centrifuged twice at 16,000 × *g* for 10 min in an Eppendorf microcentrifuge (Brinkman Instruments, Westbury, NY); osmolality of the supernatant fluid was determined by freezing point depression using a Micro-Osmometer (No. 3 MO; Advanced Instruments, Needham Heights, MA). Ruminal fluid was also collected into 17- × 100-mm polypropylene centrifuge tubes as noted previously and immediately frozen on dry ice. Samples were stored at -20°C until they were analyzed for D(-)- and L(+)-lactate (Hibbard et al., 1995). Samples of dietary ingredients were collected weekly and analyzed for Kjehldahl N, Ca, P, K (AOAC, 1990), and ADF (Goering and Van Soest, 1970).

Venous whole blood collected into Li-heparin tubes (10 mL) was used to determine blood gases, pH, HCO₃⁻, base excess, and packed cell volume immediately after collection. Duplicate heparinized capillary tubes were filled, centrifuged for 6 min (IEC Clinical Centrifuge, Damon/IEC Division, Needham Heights, MA), and analyzed for packed cell volume. Blood gases and pH were determined, and HCO₃⁻, total CO₂, and base excess were calculated using a pH/Blood Gas Analyzer (Corning model 168; Ciba-Corning Diagnostics, Medfield, MA). These samples were subsequently centrifuged at 545 × *g* for 15 min at 4°C (Sorvall RT6000B, DuPont Instruments, Wilmington, DE), and plasma was harvested and stored at -20°C until it was assayed for NEFA concentration (NEFA-C; Waco Chemical USA, Dallas, TX). Plasma was harvested from a second venous whole blood sample collected into 5-mL (NaF/EDTA) tubes following centrifugation and stored at -20°C until it was assayed for L(+)-lactate (Hibbard et al., 1995) and glucose (DACOS Chemistry Analyzer, Coulter Electronics, Hialeah, FL). Serum was harvested from venous whole blood (10 mL) allowed to clot at room temperature for 2 h and stored at -20°C until assayed for chemical constituents (DACOS Chemistry Analyzer, Coulter Electronics) cortisol, insulin (Coat-A-Count, Diagnostic Products, Los Angeles, CA), triiodothyronine, thyroxine (ELISA, Boehringer Mannheim Corp., Indianapolis, IN), growth hormone (Moseley et

al., 1982), and IGF-I (Dahl et al., 1993). Intra- and interassay coefficients of variation for hormone assays were less than 9 and 10%, respectively.

Data Analyses. Because AA and SA steers were challenged on d 0 and AC and SC steers on d 1, collection days were designated as d 0 (challenge day) and 3, 7, 10, and 14 d after the challenge. With the exception of serum insulin and GH, data were summarized by calculating means within a day before analysis. Mean GH, area under the GH curve, and serum GH pulsatility were determined using Cluster analysis software (Veldhuis and Johnson, 1986). Serum insulin was summarized by calculating area under the curve using the trapezoidal summation method.

Data obtained during the preconditioning period were evaluated for use as covariates. When the effects of treatment or treatment × day were significant (*P* < 0.05), the d -3 value was used as a covariate in subsequent analyses. When these effects were not different (*P* > 0.05), the mean value of d -31 and -3 was used as a covariate in subsequent analyses. For subsequent analyses, nonsignificant (*P* > 0.05) covariates were removed from the model. The model included the effects of block and treatment for all individual variables analyzed.

Individual summary variables for d 10 and 14 were subjected to repeated measures analysis of variance using GLM procedures of SAS (SAS Inst. Inc., Cary, NC) separately from d 0 through 7 because two AA steers were removed from the experiment after d 7 and repeated measures analysis in GLM omits experimental units with missing observations. Treatment sums of squares were partitioned into the contrasts of AA vs AC, SA vs SC, and AA vs SA. Polynomial contrasts were used to describe linear and quadratic responses across time for d 0 through 7 when a day × contrast interaction (*P* < 0.05) occurred, and day × treatment means for d 10 and 14 were separated using Fisher's LSD.

Two AA- and four SA-challenged steers did not exhibit clinical signs of acute or subacute ruminal acidosis, respectively. Therefore, all steers were qualitatively classified regardless of initial treatment for modeling purposes. Each steer was placed in one of three classes: AA, SA, or not affected (NA). Steers were initially classified as AA, SA, or NA when average daily ruminal pH on at least 1 d was ≤ 5.00, 5.01 to 5.60, or ≥ 5.61, respectively. Ruminal total lactate, plasma L(+)-lactate, and DMI were subsequently considered to evaluate this initial classification.

Subsets of individual summary variables (e. g., endocrine, blood acid/base status, blood inorganic elements, blood enzyme activities, other blood metabolites, and ruminal fermentation variables) within day were subjected to regression analysis (PROC REG; SAS Inst. Inc., Cary, NC) to select variables that distinguished among the three classes. Within a subset, the variable with the highest variance inflation factor (measure of collinearity; SAS Inst. Inc.) was removed from the subset; all variables remaining displayed a variance infla-

tion factor < 10. Class served as the dependent variable, whereas class comparisons were AA vs SA, AA vs NA, and SA vs NA. Only a maximum of two classes could be evaluated in the analysis at one time; therefore, the two classes included for a comparison were coded 0 or 1. Variables selected from each subset were then combined and the process was repeated. The final variables selected were used to develop a discriminant function placing each steer in the class with the smallest generalized squared distance (PROC DISCRIM; SAS Inst. Inc.). Options were included in the model statement to request parametric methods for multivariate normal distributions, Bartlett's test of homogeneity of within-group covariance matrices, and to evaluate the performance of the discriminant criterion by estimating probabilities of misclassification of future observations (SAS Inst. Inc.). In the event of steer misclassification using variables derived from the best regression model (based on adjusted r^2 and Mallow's C_p statistic), variables contained in the next best model were tested. This process was repeated until no further misclassification was evident.

Results and Discussion

Two AA steers were removed from the experiment on d 7 because of inappetence; one steer was euthanatized, and the second steer recovered. All data for these steers were collected on d 7, except for ruminal D(-)- and L(+)-lactate. The steer that was euthanatized displayed shallow, rapid respiration, blood pH = 7.311, base excess = -1.2 mEq/L, hematocrit = 54%, and serum creatinine = 13.1 mg/dL. Necropsy revealed emphysema of the lungs, a shrunken spleen containing little blood, a hemorrhagic area in the ruminal wall, and several kidney infarcts.

Feed Intake. Dry matter intake (Figure 1; day \times treatment, $P < 0.01$) responded quadratically ($P < 0.01$) through d 7 for AA. Dry matter intake increased linearly ($P < 0.01$) across days for AC steers, whereas DMI responded quadratically ($P < 0.01$) for SA steers. Dry matter intake by AA steers reached a nadir (< 3 kg/d) on d 3 and gradually increased to a level similar to other treatments (7 kg/d) by d 10, whereas DMI by SA steers increased through d 3.

Feed intake on d 0 for AA and SA steers included their respective intraruminal dose. A dramatic decrease was evident on d 3 and 7 for AA steers, with a subsequent increase as mentioned previously. However, this increased DMI was partly a function of removing two AA steers with low DMI from the experiment after d 7. In contrast, AC steers showed a steady increase in DMI through d 10, whereas DMI of SA steers increased markedly by d 3. Decreased DMI in response to an increased ruminal acid load has been described previously (Dirksen, 1970; Fulton et al., 1979; Harmon et al., 1985). Huber (1976) reviewed the literature and indicated that the frequency and amplitude of ruminal contractions decrease as ruminal pH decreases to 5.0,

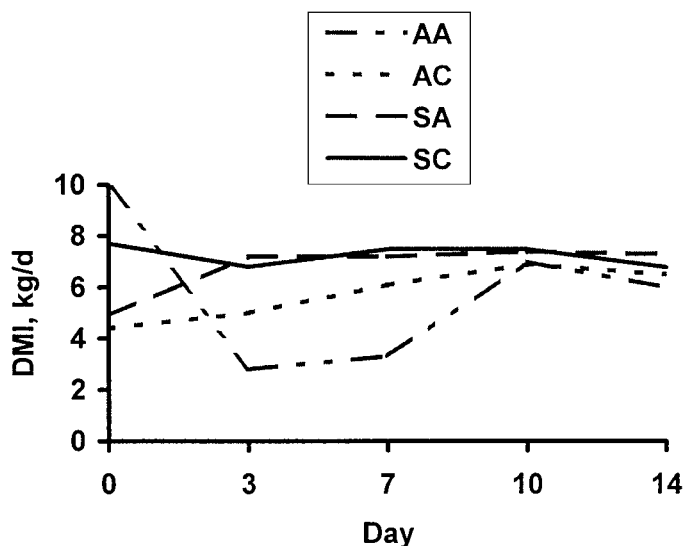


Figure 1. Effect of acute and subacute acidosis challenges on DMI by beef steers. Day \times treatment interaction was significant ($P < 0.01$). Treatments are coded as follows: AA = acute acidosis, AC = AA control, SA = subacute acidosis, SC = SA control, and DMI on d 0 includes the intraruminal dose given to AA and SA. The SEM across time was .8 kg/d, with five observations per data point through d 7 and a minimum of three observations per data point on d 10 and 14. Dry matter intake responded quadratically for AA (AA vs AC \times quadratic, $P < 0.05$) through d 7 and increased linearly for AC (AA vs AC \times linear, $P < 0.05$) through d 7. Dry matter intake by SA increased quadratically (SA vs SC \times quadratic, $P < 0.05$) through d 7, whereas DMI by SC did not differ across time (SA vs SC \times linear, $P < 0.05$).

ultimately resulting in stasis. Although the mechanism by which ruminal acidity, tonicity, and(or) organic acids collectively might decrease intake is not clear, the duration of time that ruminal pH is below 5.2 seems important in facilitating the shift in products of microbial fermentation from VFA to lactate (Slyter, 1976).

Ruminal Fermentation. Ruminal pH (Table 2) of AA and AC steers decreased quadratically ($P < 0.01$) through d 7. Ruminal pH of SA steers increased linearly ($P = 0.01$) through d 7, whereas pH of SC steers was unchanged through d 7 ($P > 0.05$). On d 10 and 14, ruminal pH was highest for SA steers and lowest for AC steers ($P < 0.05$). Ruminal D(-)-lactate for AA and AC steers responded quadratically ($P = 0.05$) through d 7; however, the pattern differed between these two treatments. Ruminal D(-)-lactate peaked on d 3 for AA steers, whereas D(-)-lactate was highest on d 0 and decreased thereafter for AC steers. For SA and SC steers, D(-)-lactate was unchanged ($P = 0.54$) through d 7. Ruminal L(+)-lactate responded quadratically ($P < 0.01$) for AA and AC steers; L(+)-lactate increased on d 3 and decreased on d 7 for AA steers, whereas L(+)-lactate was decreased after d 0 for AC steers. Ruminal L(+)-lactate was unchanged across time ($P = 0.61$) for

SA and SC steers. Ruminal total lactate responded quadratically across days ($P = 0.01$) for AA and AC steers. Total lactate increased on d 3 and decreased on d 7 for AA steers, whereas total lactate decreased for AC steers after d 0. Ruminal total lactate was unchanged ($P = 0.56$) across time for SA and SC steers. Generally, basal concentrations of total lactate were evident for all treatments by d 7. Ruminal osmolality responded quadratically through d 7 ($P < 0.01$) for AA and AC steers. For AC steers, osmolality was highest on d 0, lowest on d 3, and intermediate on d 7. For AA steers, osmolality was lowest on d 0, highest on d 3, and intermediate on d 7. Osmolality was unchanged ($P = 0.15$) through d 7 for SA and SC steers. Osmolality was highest for SC and lowest for AA steers on d 10 ($P < 0.05$), whereas osmolality on d 14 was highest for SA steers and lowest for both AA and AC steers ($P < 0.05$).

Ruminal pH reached a nadir for AA and AC steers on d 3, whereas ruminal pH for SA steers was lowest on d 0. These data seem to reflect a more persistent and severe acid insult for AA and AC steers, which is supported by ruminal lactate data. Ruminal lactate concentration was increased through d 3 for AA steers, despite the omission of two experimental units. Although AC steers displayed ruminal total lactate concentrations approaching 50 mM on d 0, mean ruminal pH remained above 5.6, and DMI progressively increased through d 10. This response seems noteworthy because the AC treatment was intended to simulate forage-fed cattle during the initial exposure to ad libitum access to a 50% concentrate diet. For SC steers (previously adapted to a 50% concentrate diet), ruminal total lactate on d 0 exceeded 15 mM, but neither ruminal pH nor osmolality was influenced. In agreement

Table 2. Effect of acute and subacute acidosis challenges on ruminal pH, lactate concentration, and osmolality in beef steers^a

Item and day	Treatment ^b				SE ^c	n ^c	Contrast ^d
	AA	AC	SA	SC			
Ruminal pH							
0	6.32	6.11	5.68	6.25	0.14	5	
3	5.04	5.78	6.05	6.01	0.22	5	
7	5.50	5.96	6.21	6.21	0.16	5	u, v, w, y, z
10	6.00 ^{ef}	5.97 ^e	6.22 ^f	6.07 ^{ef}	0.10	3	
14	6.10 ^{ef}	6.00 ^e	6.25 ^f	6.12 ^{ef}	0.10	3	
D(-)-lactate, mM							
0	3.9	10.0	2.8	9.6	3.4	5	
3	21.8	0.4	0.6	0.3	8.8	5	
7	0.3	0.3	0.4	0.4	0.03	3	v
10	0.3	0.5	0.3	0.4	0.1	3	
14	0.2	0.3	0.4	0.2	0.2	3	
L-(+)-lactate, mM							
0	14.6	38.1	1.7	7.9	7.5	5	
3	15.3	0.2	0.3	0.2	6.2	5	
7	0.1	0.1	0.2	0.2	0.03	3	u, v
10	0.2	0.3	0.1	0.3	0.08	3	
14	0.1	0.1	0.1	0.1	0.06	3	
Total lactate, mM							
0	18.4	48.1	4.6	17.5	10.4	5	
3	37.1	0.5	0.9	0.4	15.0	5	
7	0.5	0.4	0.5	0.6	0.06	3	v
10	0.5	0.8	0.4	0.7	0.2	3	
14	0.3	0.4	0.6	0.3	0.2	3	
Osmolality, mOsm/kg							
0	261	326	330	311	6	5	
3	284	276	312	312	5	5	
7	271	287	309	305	8	5	u, v, z
10	283 ^e	296 ^{ef}	305 ^{fg}	311 ^g	6	3	
14	293 ^e	297 ^e	308 ^f	302 ^{ef}	4	3	

^aDay × treatment ($P < 0.02$) for all variables.

^bAA = acute acidosis (3% of BW as steam-flaked corn), AC = AA control (offered forage-adapted steers ad libitum access to a 50% concentrate diet), SA = subacute acidosis (1.5% of BW as dry-rolled wheat:dry-rolled corn [50:50]), SC = SA control (offered concentrate-adapted steers ad libitum access to a 50% concentrate diet).

^cSE = standard error; n = minimum number of observations per mean.

^du = (AA vs AC) × linear ($P < 0.05$), v = (AA vs AC) × quadratic ($P < 0.05$), w = (SA vs SC) × linear ($P < 0.05$), x = (SA vs SC) × quadratic ($P < 0.05$), y = (AA vs SA) × linear ($P < 0.05$), and z = (AA vs SA) × quadratic ($P < 0.05$) interactions of treatment comparisons with time from d 0 through 7.

^{e,f,g}Row means differ ($P < 0.05$); analysis was done only for d 10 and 14.

with previous data (Suber et al., 1979; Krehbiel et al., 1995), plasma L(+)-lactate concentration did not differ among treatments, lending support to observations of a low correlation between ruminal and plasma L(+)-lactate (Suber et al., 1979). Ruminal osmolality was lower than expected for AA steers, considering the dose administered (Huber, 1971; Carter and Grovum, 1990). Because both the dose used and method of hydrating of steam-flaked corn given intraruminally were validated previously (Hibbard et al., 1995), an explanation for the lack of a higher ruminal osmolality of AA steers is not apparent. Moreover, the intraruminal dose for SA steers was not hydrated in the present experiment.

Blood Acid/Base Status. Time \times treatment interactions ($P < 0.05$) were observed for blood gases, acid/base status, and packed cell volume (Table 3). Blood pH decreased linearly ($P < 0.01$) through d 7 for AA steers, whereas blood pH increased linearly ($P < 0.01$) for AC and SC steers. Blood pH responded quadratically ($P < 0.01$) for SA steers; blood pH was highest on d 0, lowest on d 3, and intermediate on d 7. On d 10, blood pH was lower in AA and SA steers than in AC steers ($P < 0.05$). Blood bicarbonate decreased linearly ($P = 0.03$) in AA steers and increased linearly ($P = 0.03$) in SC steers; however, bicarbonate of AC and SA steers did not differ ($P > 0.08$) through d 7. Blood base excess decreased linearly ($P = 0.01$) in AA steers, increased linearly ($P = 0.01$) in SC steers, and was unchanged through d 7 ($P = 0.20$) in AC steers. Blood base excess of SA steers responded quadratically ($P = 0.03$) through d 7, whereas blood base excess was increased ($P < 0.05$) for AC steers on d 10. Blood pCO₂ was unchanged through d 7 ($P > 0.23$) for AA and SC steers. Blood pCO₂ decreased linearly ($P < 0.01$) for AC steers and increased linearly ($P = 0.02$) for SA steers. Blood total CO₂ decreased linearly ($P = 0.03$) for AA steers, increased linearly ($P = 0.03$) for SC steers, and was unchanged through d 7 ($P = 0.09$) for SA steers. Blood total CO₂ for AC steers tended ($P = 0.08$) to respond quadratically through d 7; total CO₂ was decreased on d 3 relative to d 0 and 7. On d 10, total CO₂ was higher in AC and SC steers than in AA steers ($P < 0.05$). Packed cell volume increased quadratically ($P = 0.05$) for AA steers, decreased linearly ($P = 0.05$) for AC steers, and was unchanged ($P = 0.34$) through d 7 for SA and SC steers. Blood pO₂ (40.4, 43.8, 45.0, and 41.9 \pm 1.2 mm of Hg for AA, AC, SA, and SC steers, respectively) responded similarly across days (day \times treatment, $P = 0.24$). Blood pO₂ was lower ($P = 0.02$) in AA than in SA steers and tended ($P = 0.07$) to be greater in AA than in AC steers.

Trends for blood bicarbonate, base excess, and total CO₂ suggested a more persistent systemic acid/base disturbance (through d 10) that extended beyond the nadir of ruminal pH for AA steers, whereas steers on the remaining treatments generally displayed compensation by d 7. Decreased blood pH, bicarbonate, and base excess for SA steers also occurred following the nadir in ruminal pH. These results agree with those of Horn et al. (1979), who found that changes in blood

acid/base status are small during subacute acidosis. However, Goad et al. (1998) reported a more marked decrease in blood bicarbonate and base excess during subacute acidosis than was evident in the present experiment.

Blood Metabolites. Plasma L(+)-lactate concentration (day \times treatment, $P = 0.42$) did not differ ($P > 0.14$) among treatments (1.09, 0.49, 0.46, and 0.53 \pm .28 mM for AA, AC, SA, and SC steers, respectively). Similarly, plasma glucose concentration averaged across time (day \times treatment, $P = 0.38$; 96, 71, 78, and 77 \pm 9 mg/dL) did not differ among treatments ($P = 0.07$). Plasma NEFA concentration (Table 4) responded quadratically ($P < 0.01$) for AA steers through d 7; NEFA was highest on d 0, lowest on d 3, and intermediate on d 7. Plasma NEFA decreased linearly ($P < 0.01$) for AC steers, whereas NEFA was unchanged ($P = 0.68$) through d 7 for SA and SC steers. Plasma NEFA was decreased for AC and SA steers on d 10 ($P < 0.05$), whereas NEFA on d 14 was highest for SA and SC steers and lowest for AC steers ($P < 0.05$). Serum Na concentration responded quadratically ($P < 0.01$) through d 7 for AA steers; Na was increased on d 0 and 3 and decreased on d 7. Serum Na decreased linearly ($P < 0.01$) for AC steers through d 7. Serum Na did not differ ($P = 0.38$) between SA and SC steers through d 7, but Na was highest for SA and SC and lowest for AA and AC steers on d 10 ($P < 0.05$). Serum P concentration decreased linearly through d 7 ($P = 0.01$) for AA steers and was lower ($P < 0.05$) for AA than for SA steers on d 10. Serum P responded quadratically ($P = 0.01$) in AC steers; P increased on d 0 and decreased on d 3 and 7. Serum P of SA and SC steers was unchanged ($P = 0.35$) through d 7.

Serum albumin concentration decreased linearly across days ($P < 0.02$) for AA, AC, and SC steers, whereas albumin did not differ through d 7 ($P = 0.10$) for SA steers. On d 10, albumin was lower ($P < 0.05$) in AA steers than in the remaining treatments. Serum total cholesterol concentration followed a pattern similar to albumin. Cholesterol decreased linearly ($P < 0.01$) for AA and AC steers, whereas cholesterol did not differ ($P = 0.49$) through d 7 for SA steers. Serum cholesterol responded quadratically ($P = 0.02$) in SC steers; cholesterol was increased on d 0, decreased on d 3, and was intermediate on d 7. On d 10 and 14, cholesterol was lower ($P < 0.05$) in AA and AC than in SA or SC steers. Serum Ca and Cl concentrations (day \times treatment, $P > 0.10$) were lower ($P < 0.01$) in AA than in AC steers averaged through d 7, whereas serum Ca concentration was lower ($P < 0.01$) in AA than in SA steers. Observed means for AA, AC, SA, and SC steers were 4.7, 4.9, 4.8, and 4.9 \pm 0.03 mEq of Ca/L and 99, 103, 102, and 103 \pm 0.9 mEq of Cl/L, respectively. Rectal temperature (day \times treatment, $P = 0.57$) averaged across time was greater ($P = 0.02$) in AC than in AA steers (38.9 vs 38.7 \pm 0.1°C).

Greater plasma NEFA concentrations for AA and AC steers in the present experiment seem to agree with increased mean GH and area under the GH curve on d 0 (described below), as well as decreased DMI on d 3 and

Table 3. Effect of acute and subacute acidosis challenges on blood gases, acid/base status, and packed cell volume in beef steers^a

Item and day	Treatment ^b				SE ^c	n ^c	Contrast ^d
	AA	AC	SA	SC			
Blood pH							
0	7.396	7.332	7.402	7.340	0.008	5	
3	7.365	7.333	7.357	7.371	0.014	5	
7	7.364	7.369	7.380	7.385	0.015	5	u, w, x
10	7.364 ^e	7.391 ^f	7.359 ^e	7.375 ^{ef}	0.010	3	
14	7.405	7.378	7.398	7.376	0.014	3	
Bicarbonate, mEq/L							
0	31.3	28.0	29.5	26.5	0.7	5	
3	29.2	25.4	27.7	28.5	1.3	5	
7	28.6	28.2	29.3	29.6	0.8	5	u, w
10	26.9 ^e	30.3 ^f	27.6 ^e	28.5 ^e	0.7	3	
14	30.5	29.0	29.8	27.8	1.1	3	
Base excess, mEq/L							
0	6.44	2.12	5.21	1.20	0.67	5	
3	3.94	0.13	2.63	3.59	1.32	5	
7	3.49	3.27	4.48	4.88	0.98	5	u, w, x
10	2.17 ^e	5.54 ^f	2.55 ^e	3.70 ^e	0.68	3	
14	6.06	4.16	5.36	3.21	1.09	3	
Packed cell volume, %							
0	33	35	27	30	0.7	5	
3	38	32	29	29	2.4	5	
7	36	31	26	27	2.2	5	v
10	28	29	27	28	0.9	3	
14	27	28	27	29	1.1	3	
pCO ₂ , mm Hg							
0	51.8	53.5	48.2	50.0	1.2	5	
3	51.7	48.8	49.9	50.1	1.5	5	
7	51.5	49.7	50.5	50.5	1.0	5	u, y
10	48.0	50.9	49.8	49.6	1.5	3	
14	49.5	50.2	49.4	48.4	1.7	3	
Total CO ₂ , mm Hg							
0	32.7	29.5	30.9	27.9	0.7	5	
3	30.7	26.8	29.2	29.9	1.3	5	
7	30.0	29.6	30.8	31.1	0.8	5	u, w, y
10	28.2 ^e	31.7 ^f	29.0 ^{ef}	29.9 ^f	0.8	3	
14	31.8	30.4	31.2	29.2	1.1	3	

^aDay × treatment ($P < 0.05$) for all variables.

^bAA = acute acidosis (3% of BW as steam-flaked corn), AC = AA control (offered forage-adapted steers ad libitum access to a 50% concentrate diet), SA = subacute acidosis (1.5% of BW as dry-rolled wheat:dry-rolled corn [50:50]), SC = SA control (offered concentrate-adapted steers ad libitum access to a 50% concentrate diet).

^cSE = standard error; n = minimum number of observations per mean.

^du = (AA vs AC) × linear ($P < 0.05$), v = (AA vs AC) × quadratic ($P < 0.05$), w = (SA vs SC) × linear ($P < 0.05$), x = (SA vs SC) × quadratic ($P < 0.05$), and y = (AA vs SA) × linear ($P < 0.05$) interactions of treatment comparisons with time from d 0 through 7.

^eRow means differ ($P < 0.05$); analysis was done only for d 10 and 14.

7. Similar observations have been reported for nutrient-restricted animals (Yambayamba et al., 1996; Hornick et al., 1998). The direction of the trends for serum Na concentration for AA and AC steers are in contrast to reports from challenge experiments of shorter duration. Serum Na was lowest on d 7 and returned to d-0 concentrations by d 14 in the present experiment, whereas serum Na has been reported to increase (5 to 12 mEq/L) and then decrease to initial concentrations by 8 to 14 h after a carbohydrate challenge (Irwin et al., 1979; Cao et al., 1987; Patra et al., 1993; Brown et al., 1999). Hypophosphatemia seemed to occur earlier and to a similar

magnitude for AC compared to AA steers, which is in contrast to the hyperphosphatemia characteristic of lactic acidosis of shorter duration or observation (Oster et al., 1978; Perez et al., 1980; Brown et al., 1999). Hypophosphatemia was not accompanied ($P > 0.37$) by changes in plasma alkaline phosphatase activity (98, 92, 99, and 99 ± 6 U/L for AA, AC, SA, and SC steers), whereas Harmon and Britton (1983) reported increased alkaline phosphatase activity and total P excretion in response to intravenous lactate administration.

The linear decrease of albumin in AA, AC, and SC steers disagrees with a previous report of increased

serum albumin with an increasing severity of metabolic acidosis (Brown et al., 1999). In addition, the linear decrease of serum total cholesterol through d 7 in AA and AC steers continued numerically through d 10, in contrast to results of Brown et al. (1999). Feed intake did not differ among treatments by d 10, whereas serum total cholesterol for AA and AC steers had not yet increased to a level comparable to SA and SC steers by d 14. Increased triglyceride synthesis in liver would be expected based on plasma NEFA concentrations, and it seems likely that serum cholesterol reflects the slow output of VLDL, and thus LDL and HDL formation (Grummer, 1993).

Endocrine Profiles. Area under the curve did not differ among treatments for insulin ($P = 0.18$), triiodothyronine ($P = 0.11$), or thyroxine concentration ($P = 0.16$; data not shown). Mean GH concentration (Table 5) de-

creased linearly across days ($P = 0.02$) in AC and AA steers, whereas mean GH for SA and SC steers was unchanged through d 7 ($P = 0.55$). The response for area under the GH curve (Table 5) mirrored that of mean GH, whereas the number of pulses/7 h (day \times treatment, $P = 0.23$) did not differ ($P > 0.10$) among treatments. Averaged across time (day \times treatment, $P > 0.39$), neither serum cortisol (1.38, 0.37, 0.45, and 0.77 ± 0.41 $\mu\text{g/dL}$) nor serum IGF-I (121, 128, 152, and 137 ± 12 ng/mL) was influenced ($P > 0.12$) by the AA, AC, SA, or SC treatments.

Although serum insulin and plasma glucose concentration did not differ, the AA steer that was euthanized displayed severe hyperglycemia (300 mg/dL plasma glucose) concomitant with hypoinsulinemia. Randhawa et al. (1980) reported similar results for buffalo experiencing acute ruminal acidosis. Pulsatility of

Table 4. Effect of acute and subacute acidosis challenges on blood metabolites in beef steers^a

Item and day	Treatment ^b				SE ^c	n ^c	Contrast ^d
	AA	AC	SA	SC			
Plasma NEFA, $\mu\text{Eq/L}$							
0	433	662	62	129	61	5	
3	221	66	81	60	46	5	
7	273	60	54	74	72	5	u, v, z
10	125 ^e	75 ^f	89 ^f	120 ^e	15	3	
14	77 ^{ef}	70 ^e	86 ^f	86 ^f	8	3	
Serum Na, mEq/L							
0	139	143	140	142	0.4	5	
3	139	139	141	140	0.8	5	
7	132	138	140	140	2.4	5	v, y
10	138 ^e	138 ^e	140 ^f	141 ^f	0.9	3	
14	142	143	143	142	1.6	3	
Serum P, mEq/L							
0	4.7	5.0	3.9	4.5	0.2	5	
3	4.3	3.2	3.8	4.0	0.2	5	
7	4.2	3.3	3.9	4.0	0.4	5	v
10	3.1 ^e	3.6 ^{ef}	3.9 ^f	3.7 ^{ef}	0.3	3	
14	3.5	3.8	3.9	3.8	0.3	3	
Serum albumin, g/dL							
0	3.2	3.3	2.9	3.2	0.05	5	
3	3.0	3.1	3.0	3.1	0.07	5	
7	2.9	3.0	2.9	3.0	0.05	5	w, y
10	2.8 ^e	2.9 ^f	2.9 ^f	2.9 ^f	0.05	3	
14	3.0	3.0	3.0	3.1	0.06	3	
Serum cholesterol, mg/dL							
0	81.6	84.6	71.8	89.5	2.7	5	
3	65.2	64.4	69.9	62.5	4.7	5	
7	56.8	56.4	70.7	69.7	4.5	5	w, x, y
10	51.6 ^e	49.3 ^e	71.0 ^f	73.4 ^f	5.4	3	
14	61.7 ^e	53.8 ^e	77.4 ^f	80.3 ^f	6.5	3	

^aDay \times treatment ($P < 0.05$) for all variables, covariate ($P < 0.01$) for albumin and cholesterol.

^bAA = acute acidosis (3% of BW as steam-flaked corn), AC = AA control (offered forage-adapted steers ad libitum access to a 50% concentrate diet), SA = subacute acidosis (1.5% of BW as dry-rolled wheat:dry-rolled corn [50:50]), SC = SA control (offered concentrate-adapted steers ad libitum access to a 50% concentrate diet).

^cSE = standard error; n = minimum number of observations per mean.

^du = (AA vs AC) \times linear ($P < 0.05$), v = (AA vs AC) \times quadratic ($P < 0.05$), w = (SA vs SC) \times linear ($P < 0.05$), x = (SA vs SC) \times quadratic ($P < 0.05$), y = (AA vs SA) \times linear ($P < 0.05$), and z = (AA vs SA) \times quadratic ($P < 0.05$) interactions of treatment comparisons with time from d 0 through 7.

^{e,f}Row means differ ($P < 0.05$); analysis was done only for d 10 and 14.

Table 5. Effect of acute and subacute acidosis challenges on serum metabolic hormones in beef steers

Item and day	Treatment ^a				SE ^b	n ^b	Contrast ^c
	AA	AC	SA	SC			
Serum insulin, ng/mL ^d							
0	0.8	0.3	1.6	0.8	0.2	4	
3	1.6	2.2	2.5	3.3	0.7	4	
7	1.1	1.9	1.2	1.2	0.2	4	
10	1.9	1.0	1.1	1.0	0.6	3	
14	1.6	0.8	1.7	1.4	0.3	3	
Mean GH, ng/mL ^e							
0	11.1	13.7	4.7	3.4	1.5	5	
3	7.6	6.4	2.0	3.1	2.4	5	
7	4.2	1.8	2.9	2.6	0.7	5	u, y
10	1.1	1.6	2.1	2.2	0.6	3	
14	1.8	1.4	1.6	1.9	0.4	3	
Area under the GH curve ^e							
0	4,814	5,968	2,016	1,485	670	5	
3	3,154	2,627	882	1,286	994	5	
7	1,807	793	1,264	1,041	308	5	u, y
10	521	669	918	942	255	3	
14	790	613	672	836	164	3	
GH pulses/7 h							
	1.8	1.1	1.5	1.5	0.3	5	

^aAA = acute acidosis (3% of BW as steam-flaked corn), AC = AA control (offered forage-adapted steers ad libitum access to a 50% concentrate diet), SA = subacute acidosis (1.5% of BW as dry-rolled wheat:dry-rolled corn [50:50]), SC = SA control (offered concentrate-adapted steers ad libitum access to a 50% concentrate diet).

^bSE = standard error; n = minimum number of observations per mean.

^cu = (AA vs AC) × linear ($P < 0.05$) and y = (AA vs SA) × linear ($P < 0.05$) interactions of treatment comparisons with time from d 0 through 7.

^dArea under the curve ($P = 0.18$).

^eDay × treatment ($P < 0.01$).

GH did not differ, whereas mean GH concentration and area under the GH curve were increased for AA and AC, and both decreased linearly through d 7. The influence of carryover effects from the feed withdrawal period on serum GH cannot be eliminated because the duration of this period was not equalized among treatments. Although the feed withdrawal period before the challenge was slightly longer for SA than for AC steers (1 d) and feed intake was similar for SA and AC steers on d 0, mean GH concentration and area under the GH curve were considerably higher for AC steers on d 0. These observations suggest that other factors might be involved in stimulating GH secretion and/or altered GH half-life of AA and AC steers. Huber (1976) indicated that ruminal pH < 5.4 can result in an increased ruminal concentration of bacterial endotoxin. Endotoxin challenges in sheep have increased plasma GH, prolactin, and cortisol concentrations (Coleman et al., 1993), whereas intrathoracic yeast injection has increased plasma GH (Moore et al., 1995). However, Brown et al. (1999) reported that serum prolactin was not influenced by an increasing severity of metabolic acidosis resulting from ruminal glucose administration in ewes.

In summary, steers challenged with 3% of body weight as steam-flaked corn in an attempt to produce acute acidosis displayed a more persistent decrease in

serum sodium, phosphorus, albumin, total cholesterol, feed intake, and ruminal and blood acid/base status than steers challenged with 1.5% of body weight as wheat:corn (50:50) in an attempt to produce subacute acidosis. In the early stages, acute acidosis was further characterized by increased area under the growth hormone curve.

Data Modeling. Acute ruminal acidosis has generally been clinically defined as ruminal pH ≤ 5.00 and ruminal total lactate ranging from > 40 (Owens et al., 1996) to > 90 mM (Hibbard et al., 1995). Subacute ruminal acidosis has generally been clinically defined as ruminal pH of 5.01 to 5.60 and ruminal total lactate ranging from < 10 (Harmon et al., 1985; Burrin and Britton, 1986; Goad et al., 1998) to approximately 50 mM (Horn et al., 1979). Several previous reports (Huber, 1971; Dougherty et al., 1975; Suber et al., 1979) document the marked variation in animal responses to substrates administered on a BW basis. For example, Dougherty et al. (1975) administered 70 g of grain (75:25 whole corn:whole oats)/kg of BW intraruminally to three steers; two experienced acute acidosis (one steer was euthanized), whereas ruminal pH of a third steer did not decrease below 5.5. As mentioned previously, two steers initially on the AA treatment and four steers initially on the SA treatment did not clinically develop acute or subacute ruminal acidosis, respectively, ac-

ording to these criteria. Therefore, data from the present experiment were modeled to identify response variables that might reliably discriminate among animals presenting clinical signs of acute and subacute ruminal acidosis and animals that did not seem to be clinically affected, regardless of a challenge with carbohydrate.

Characteristics of steers qualitatively classified as not affected (NA), or as experiencing acute (AA) or subacute (SA) ruminal acidosis regardless of initial treatment are presented in Table 6. Animals were initially classified as AA, SA, or NA when the lowest average daily ruminal pH was ≤ 5.00 , 5.01 to 5.60, or ≥ 5.61 . The day that this occurred varied from d 0 (day of the challenge) to d 3 after the challenge. Lowest average daily ruminal pH was used because it provided the highest correlation with DMI on the day following the insult ($r = 0.843$), compared with ruminal pH 5 h after the challenge ($r = 0.834$) or the lowest 2-h interval ruminal pH ($r = 0.824$). Ruminal total lactate, plasma L(+)-

lactate, and DMI were subsequently considered to evaluate the initial classification. Because these classifications were somewhat subjective, observed values and calculated means within each group are presented, as well as initial treatment assignment (Table 6).

The final classification of steers indicated that one steer initially challenged with 3% of BW as steam-flaked corn was classified as SA and a second steer as NA. Four steers challenged with 1.5% of BW as 50:50 wheat:corn were classified as NA. Moreover, ruminal total lactate of steers classified as SA or NA encompassed a similar range (15 to 61 mM for SA and 4 to 66 mM for NA). Calculated means for ruminal pH, DMI, plasma L(+)-lactate concentration, and ruminal total lactate concentration of steers within each classification through d 7 are represented in Figure 2 (a through d, respectively).

Results of variables used in discriminant analysis that provided a probability of misclassification of future

Table 6. Characteristics of individual beef steers classified as experiencing acute or subacute acidosis or not affected by a carbohydrate challenge

Class ^a and steer ID	Initial treatment ^b	Ruminal pH ^c	Plasma L(+)-lactate, mM ^c	Peak ruminal total lactate, mM ^d	DMI, kg ^e
Acute					
a	AA	4.46	1.1	111.4	0.08
n	AA	4.33	1.1	92.0	0.16
r	AA	4.26	1.6	126.6	1.49
Average		4.35	1.3	110.0	0.58
Subacute					
b	AC	5.48	0.6	54.0	5.51
e	AA	5.04	0.8	22.6	1.51
h	SA	5.34	0.5	14.6	5.95
t	AC	5.42	0.4	61.4	5.20
Average		5.32	0.6	38.1	4.54
Not affected					
c	SA	5.70	0.4	4.3	7.63
d	SC	5.94	0.5	12.7	7.14
f	AC	5.78	0.5	31.0	6.79
g	SC	5.91	0.4	7.0	7.25
i	SA	5.76	0.5	6.1	8.19
j	AA	5.63	2.3	14.5	5.13
k	SC	5.99	0.4	8.7	8.16
l	AC	5.83	0.4	66.5	6.86
m	SC	5.69	0.4	52.6	6.78
o	SA	5.74	0.4	4.9	7.51
p	AC	5.75	0.5	27.4	6.56
q	SC	6.03	0.4	9.8	6.25
s	SA	5.85	0.5	3.8	6.70
Average		5.81	0.6	19.2	7.00

^aSteers were initially classified as acute, subacute, or not affected when average daily ruminal pH on at least 1 d was ≤ 5.00 , 5.01 to 5.60, or ≥ 5.61 . Ruminal total lactate and plasma L(+)-lactate concentration and DMI were subsequently considered to evaluate this initial classification.

^bAA = acute acidosis (3% of BW as steam-flaked corn), AC = AA control (offered forage-adapted steers ad libitum access to a 50% concentrate diet), SA = subacute acidosis (1.5% of BW as dry-rolled wheat:dry-rolled corn [50:50]), SC = SA control (offered concentrate-adapted steers ad libitum access to a 50% concentrate diet).

^cActual average ruminal pH and plasma L(+)-lactate used to classify animals that was observed on the first day pH was ≤ 5.00 , 5.01 to 5.60, or ≥ 5.60 for acute, subacute, and not affected, respectively.

^dHighest ruminal total lactate concentration observed for each animal.

^eDMI for the day following the first day that ruminal pH was ≤ 5.00 , 5.01 to 5.60, or ≥ 5.61 for acute, subacute, and not affected, respectively.

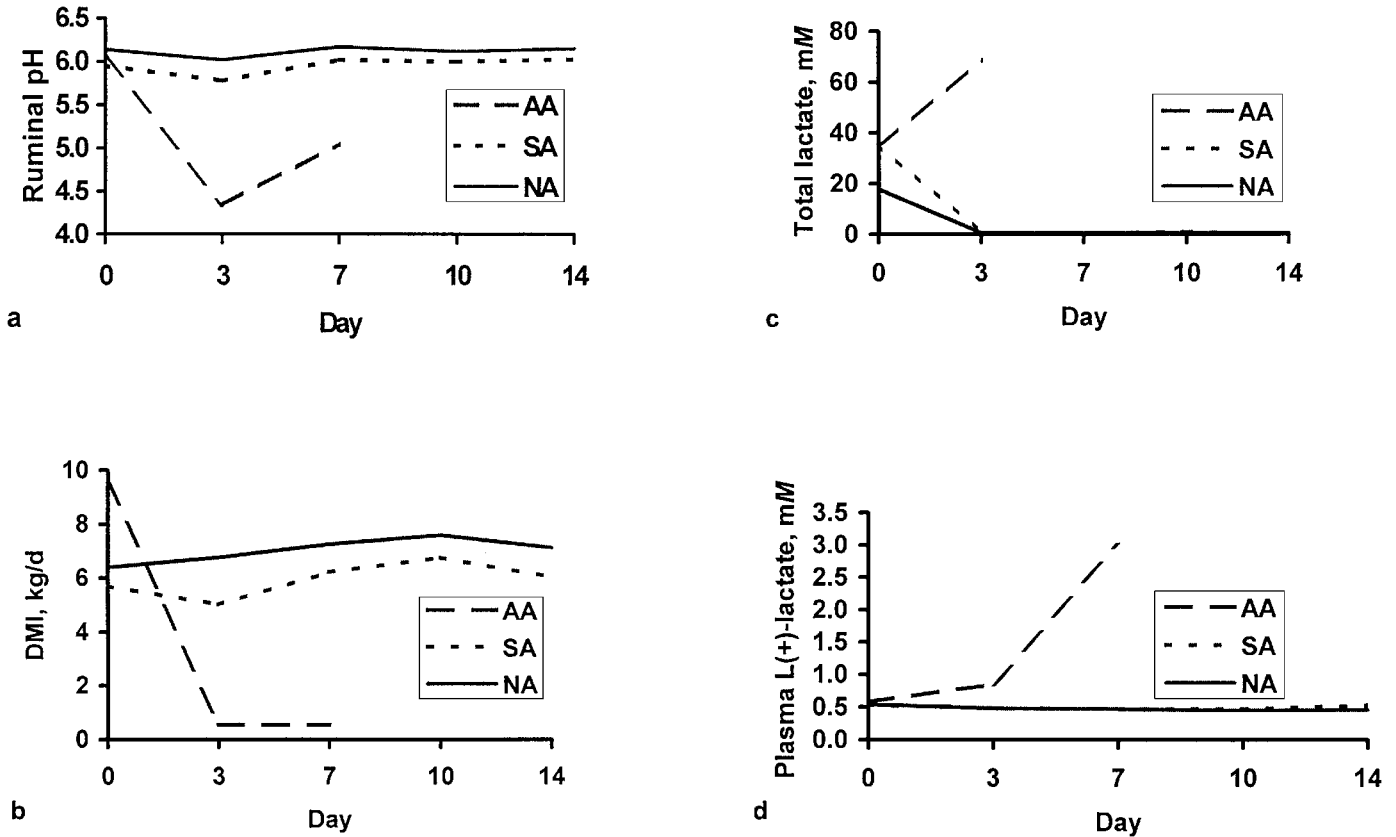


Figure 2. Ruminal pH (panel a), DMI (panel b), ruminal total lactate (D + L) concentration (panel c), and plasma L(+)-lactate concentration (panel d) of beef steers classified as experiencing acute (AA) or subacute (SA) acidosis, or not affected (NA) by a grain challenge. There were three observations per mean through d 7 for AA, four observations per mean for SA, and 13 observations per mean for NA. Data for AA on d 10 and 14 were omitted because two steers were removed from the experiment as a result of initial treatment.

observations of 0.0 are presented in Table 7. Ruminal total lactate concentration, blood pH, blood bicarbonate, blood base excess, and serum triiodothyronine were not selected in any of the comparisons. Moreover, ruminal pH and DMI were selected infrequently. These results suggest that variables others than those commonly associated with ruminal acidosis can more reliably distinguish among the classes designated previously (Table 6).

Comparisons of AA vs SA and AA vs NA steers on d 10 and 14 were not conducted because two of the three steers classified as AA were removed from the experiment after d 7. Each variable selected to differentiate AA and SA steers occurred on only 1 d, whereas blood $p\text{CO}_2$, serum albumin, serum cholesterol, and ruminal osmolality distinguished between AA and NA steers on d 3 and 7. A greater number of response variables were selected across a greater number of collection days for distinguishing between SA and NA than for AA and NA or AA and SA steers. Serum amylase activity was included in the model on four of five collection days, whereas plasma NEFA, serum cholesterol, and serum K concentrations were included in the model on three of five collection days. All of the variables listed on each day for each comparison were necessary to develop each

discriminant function, and caution is warranted regarding the use of a single variable or subset of those variables to distinguish between the designated classes. However, means for certain variables that were included in the discriminant function on multiple days for AA vs NA and SA vs NA steers are presented to indicate the direction of change in Table 8.

In contrast to present observations, Krehbiel et al. (1995) reported that plasma amylase activity did not differ between lambs offered a 50% concentrate diet and those intraruminally dosed with 0, 6, 12, or 18 g of glucose/kg of BW. Brown et al. (1999) indicated that serum cholesterol was not influenced by an increasing severity of metabolic acidosis. Brown et al. (1999) reported decreased serum K concentration as the level of ruminal glucose infusion increased, whereas circulating serum aldosterone concentration was increased for ewes dosed with 10 g of glucose/kg of BW. Irwin et al. (1979) and Patra et al. (1993) also reported decreased serum K after inducing acute ruminal acidosis. However, Lal et al. (1992) observed increased serum amylase activity in goats intraruminally dosed with 100 g of whole wheat/kg of BW.

No single variable measured in the present experiment displayed a consistent response across time that

Table 7. Summary of variables that distinguished between steers classified as experiencing acute or subacute acidosis or not affected by a carbohydrate challenge^a

Comparison and day	Variables included
Acute vs subacute	
0	Serum lactate dehydrogenase activity (LDH), serum urea N, plasma L(+)-lactate, ruminal pH, serum amylase activity
3	Serum insulin, serum glutamine-oxaloacetate transaminase activity (GOT), plasma NEFA
7	Serum IGF-I, serum thyroxine, blood pCO ₂ , serum Ca, albumin
Acute vs not affected	
0	Blood pO ₂ , plasma glucose, serum Na, serum glutamine-pyruvate transaminase activity (GPT), DMI
3	Serum cortisol, serum IGF-I, blood pCO ₂ , serum albumin, serum cholesterol, ruminal osmolality
7	Area under the GH curve, blood total CO ₂ , blood pCO ₂ , serum Ca, serum albumin, serum cholesterol, ruminal osmolality
Subacute vs not affected	
0	NEFA, serum IGF-I, serum amylase activity, serum Cl, blood pCO ₂ , GOT, serum cholesterol, LDH, serum insulin
3	NEFA, ruminal pH, serum Ca, serum urea N, serum Na, serum cholesterol, blood total CO ₂
7	Serum amylase activity, LDH, serum K
10	Serum cortisol, serum amylase activity, blood pCO ₂ , blood pO ₂ , hematocrit, GPT, plasma L(+)-lactate, NEFA, serum K, serum Na
14	Serum amylase activity, blood pCO ₂ , serum alkaline phosphatase, serum albumin, serum cholesterol, serum K, serum P, serum urea N, DMI, rectal temperature, ruminal osmolality

^aProbability of misclassification of future observations = 0.0.

Table 8. Observed means of variables selected on multiple days that distinguished between beef steers classified as experiencing acute acidosis (AA) or not affected (NA) or as experiencing subacute acidosis (SA) or not affected (NA) by a carbohydrate challenge

Item	Day					n ^a
	0	3	7	10	14	
AA vs NA						
pCO ₂ , mm Hg						
AA	53.3	53.9	53.6	—	—	3
NA	49.9	49.5	50.1	49.3	48.7	13
Albumin, mg/dL						
AA	3.22	3.19	2.97	—	—	3
NA	3.12	3.00	2.94	2.93	3.04	13
Ruminal osmolality, mOsm/kg						
AA	265	281	253	—	—	3
NA	311	301	302	303	301	13
SA vs NA						
Amylase, U/L						
SA	47.9	45.9	51.7	57.8	57.9	4
NA	40.9	40.5	43.1	46.3	47.2	13
Cholesterol, mg/dL						
SA	89.1	73.2	72.9	67.2	67.4	4
NA	79.5	62.3	63.3	62.6	71.6	13
Potassium, mEq/L						
SA	4.2	4.1	4.1	4.2	4.2	4
NA	4.0	3.9	3.8	3.9	3.8	13
NEFA, μEq/L						
SA	732.6	102.1	70.0	78.7	76.7	4
NA	224.5	67.4	68.5	101.4	82.5	13

^aMinimum number of observations per mean.

could be used to identify acute or subacute ruminal acidosis. The syndrome of acidosis seems to involve numerous tissues and generally results in complex and transient changes in physiology across time. Although considerable variation was evident in the ability of an animal to cope with a carbohydrate challenge, results generally suggest that serum amylase activity, cholesterol and potassium concentration, and plasma NEFA concentration were useful in distinguishing between steers classified as experiencing subacute acidosis or not affected by a carbohydrate challenge.

Implications

Considerable variation was evident in the ability of an animal to cope with a carbohydrate challenge. Results of data modeling generally suggest that serum amylase activity, cholesterol and potassium concentration, and plasma nonesterified fatty acid concentration might be useful in distinguishing between steers classified as experiencing subacute acidosis or not affected by a carbohydrate challenge, but further research is needed. Research directed toward describing the biological attributes of animals metabolically capable of coping with a carbohydrate challenge and(or) describing predisposing or "risk" factors of acute and subacute acidosis seems warranted.

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