# ACIDOSIS, RATE OF STARCH DIGESTION AND INTAKE

R. A. Britton and R. A. Stock

Department of Animal Science
University of Nebraska-Lincoln

R. A. (Bud) Britton

## INTRODUCTION

Acidosis and the rate of starch digestion are closely intertwined. Acidosis usually occurs in feedlots with cattle fed high energy diets. Therefore, the relationship between rate of starch digestion, acidosis and intake is important in determining whether the level of performance obtained is at the level predicted for the cattle and feedstuffs used.

The relationship between intake regulation and rumen energy metabolism is imperfect as evidenced by the problem of acidosis. Cattle evolved as grazing animals consuming diets consisting mainly of forages. Ruminal production, absorption and metabolism of organic acids from fermentation of plant cell walls is slow because of the inherent slow rate of fermentation of most forage cell walls. Intake in the grazing animal is most likely governed by ruminal fill or a combination of ruminal fill and energy sensing mechanisms (chemostats) coordinated in the brain. The animal did not adapt or evolve in environments where it had to cope with the problem of large quantities of starch being fermented in the rumen and the ensuing problem of disposing of the organic acids being produced. If the rate of starch digestion is fast, production of organic acids in the rumen will overcome the ability of the tissues or other microbes to absorb and utilize them. Subsequently, rumen pH declines and reduced intake follows.

Our definition of acidosis is: biochemical and physiological stresses caused by rapid production and absorption of ruminal organic acids and endotoxins caused by overconsumption of readily fermentable carbohydrates. This definition encompasses many problems that are related to low rumen pH, but are not necessarily classical acidosis. We chose the term organic acid production rather than just lactic acid production because the problem of acidosis is caused by all organic acids produced in the rumen and not just lactic acid and certainly not just the D-isomer of lactic acid.

## IMPORTANCE OF ACIDOSIS

Most cattle being marketed today are fed grain in feedlots. These cattle have to deal with rate of starch digestion in the rumen every day. Because of this acidosis will continue to be an important problem for the feedlot industry. Adjusting cattle to high grain diets is a very critical time as the rumen microbes are adapting to the changes in energy and this ends up being an unstable period in the rumen environment and a likely time for acidosis. We are in the middle of a

large grain surplus with rather low prices which supports feeding as much grain to livestock as possible. But even in times of high grain prices, grain continued to be fed to cattle during the finishing period. The response of the feedlot industry to high grain prices was to shorten the grain feeding period. This results in the grain adjustment period being a greater proportion of the total feeding period. Relieving acidosis during this adaptation period becomes critical for maximum performance of those cattle.

Roughage is used as a means of controlling acidosis in feedlots. But feeding roughages causes problems to the feedlot because of handling and mixing bulky feeds. Therefore, the trend has been to feed diets with minimal amounts of roughage and usually of small particle size. Both low roughage levels and small particle size of the roughage are generally associated with acidosis.

Figures of economic losses associated with acidosis are not available, but consideration of other feedlot problems related to acidosis suggest these economic losses are large. Brent (1976) detailed founder, polioencephalomalacia and ruminitis as problems related to acidosis. In an American Society of Animal Science symposium in 1984, we added sudden death syndrome, off-feed syndrome, malabsorption, liver abscesses and clostridial infections to this list. There are probably other disorders that are related that we have not included or are unaware of at this time.

The effect of liver abscesses on intake and feed efficiency has been detailed by Brink and coworkers (1985). They listed four categories of abscesses from 0 (none), A-, A to A+ (severely abscessed). Animals with A+ liver abscesses began to show reduced intake as early as 15 days into the feeding period (Figure 1) which continued throughout the feeding period (Figure 2). Animals with A+ liver scores exhibited reduced gain (11.5%), feed intake (3.9%) and feed efficiency (8.8%).

**Figure 1.**

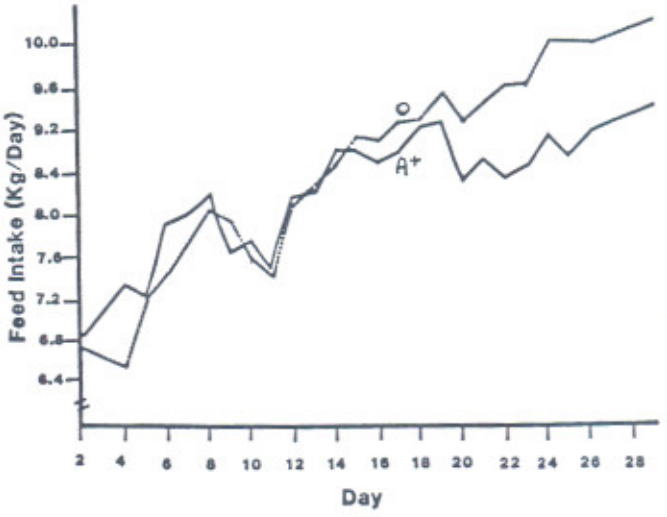
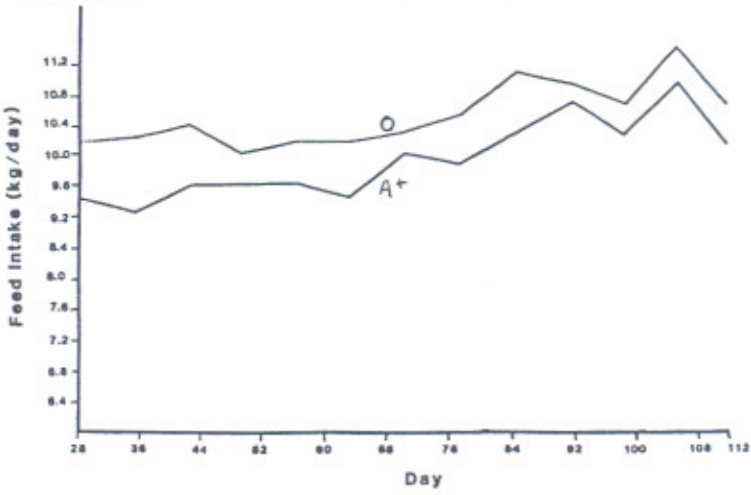


Figure 2.




ACUTE AND SUBACUTE

In our estimation acidosis is not one disease, but rather a continuum of degrees of acidosis. We have characterized it into acute and subacute for simplicity. These categories represent both extremes. The actual point where subacute becomes acute is difficult to determine and probably not very important. What is important is the realization that the extremes of acidosis are manifested differently in the animal. Acute acidosis has been reviewed and summarized by Huber (1976) and Slyter (1976). In acute acidosis, the problem can be so severe that the animal may be sick to the point of death or may have impairment of some physiological function, usually absorption, which causes reduced performance for a period of time. Acute acidosis can be minimized with proper management. Subacute acidosis, is more subtle and difficult to assess; however, the effects are just as real. The major response seen in subacute acidosis is a reduction in intake with concomitant reduction in performance.

Harmon et al. (1985) contrasted differences in ruminal concentrations and absorption of organic acids produced in the rumen of animals acutely and subacutely acidotic. Acute acidosis was induced by infusing glucose intraruminally to steers at the rate of 12 mg/kg of body weight. The subacute acidosis treatment was a rapid shift from a forage diet to a 70% concentrate diet (61.7% dry rolled corn) with no intermediate concentrate levels. Intake, in the subacute group, was 13.6 kg for the first 24 h and only 1.6 kg during the next 24 h indicating digestive upset. Rumen pH declined to 5.8 in the subacute group and 4.2 (18 h post dose) in the acute treatment. Concentrations of L-lactate rose dramatically in the rumen fluid of the glucose induced acidosis group reaching a peak of about 80 millimolar at 20 hours post dose. Very small increases in L-lactate were observed in the rumen fluid of the subacutely acidotic animals. Similar patterns for both groups were observed for D-lactate accumulation which reached a peak in rumen fluid

at about 24 to 26 hours post dosing at a concentration of about 50 millimolar. Rumen fluid lactate levels reflect the rates of microbial synthesis and degradation. Microbial degradation of lactate increases dramatically as dietary starch is introduced into the diets (Huntington and Britton, 1978, 1979; Mackie and Gilchrist, 1979; Kunkle et al., 1976). The large lactate levels in the acute acidosis group is probably a result of greatly increased synthesis which overwhelms degradative capacity and pH declines. In the subacute treatment microbial lactate degradation was sufficient to keep lactate levels moderate. Net portal absorption of organic acids showed that the lactate isomers accounted for about 60% of the organic acids absorbed in the acute treatment and only 13% in the subacute treatment. Absorption of all organic acids was reduced in the acute treatment (about 50% of the subacute) even though rumen concentrations of organic acids were not different. The reduced absorption was probably related to epithelial damage as blood flow to the gut was not affected by treatment. These data emphasize that acute acidosis and subacute acidosis really are different problems and that the body has to deal with different metabolic substrates coming into the system in each circumstance.

We measured the metabolic capacity of cattle to handle the substrates in acute acidosis by estimating oxidation and gluconeogenesis from D and L lactate (Harmon et al., 1984). Rates of metabolism of D-lactate were dependent on substrate concentrations and were similar to L-lactate metabolism at low (.1-1.0 mm) substrate levels in kidney and liver slices in vitro. From these estimates of oxidation and gluconeogenesis, we calculated the total potential utilization of D-lactate in a 450 kg steer at either 1 millimolar or 10 millimolar concentrations of D-lactate in the plasma. Utilization of D-lactate at 1 millimolar concentration of plasma was 8.5 millimoles per hour whereas at 10 millimolar concentration, the utilization was estimated to be about 46 millimoles per hour. These studies demonstrate that D-lactate metabolism is vigorous in cattle. In subacute acidosis, D-lactate is rarely found in quantities that are high enough to exceed the metabolic capacity of the animal.

Ruminal lactate still is a potent inhibitor of intake (Baile and Forbes, 1974) although the nature and site of the receptors remain unresolved at this time. Koers and coworkers (1976) reported two trials infusing lactic acid intraruminally in sheep fed high grain diets (Table 1). Two levels of lactic acid were infused: .17 moles in the first experiment and .33 moles in the second experiment. If we assume a 5 liter rumen volume for sheep, the concentrations of lactate would have been 34 mM

Table 1. Effect of lactic acid infusion on feed intake of sheep.

| Infusion | Daily intake |
|---|---|
| | -kg- |
| Trial 1 | |
| $H_2O$ | 1.60 |
| Lactic acid (.17 moles) | 1.70 |
| Trial 2 | |
| $H_2O$ | 1.73 |
| Lactic acid (.33 moles) | 1.03 |

and 66 mM, respectively. Only the infusion of .33 moles reduced intake in these experiments. This level of rumen lactate is found in animals suffering from acute acidosis, but not in the subacute type. Therefore, rumen lactate concentrations are probably not a major factor regulating intake in subacute acidosis, but rumen pH may be.

## SUBACUTE ACIDOSIS AND INTAKE

The most important animal response seen with subacute acidosis is reduced intake. A corollary to that statement is anything that interrupts normal consumption patterns can precipitate acidosis. Environment impacts an animals desire to eat. Impending storms cause animals to eat more before the storm arrives which keeps cattle feeders with one eye on the weather map. Factors, such as heat, cold or mud can impact intake patterns and cause acidosis. Other factors like proper feedlot design and bunk management are important factors in maintaining normal intake patterns in cattle.

Diet composition can have an impact on subacute acidosis and intake patterns. Type and amount of grain, grain processing, feed additives, roughage level and type of roughage influence intake patterns and subacute acidosis. Figure 3 depicts the relative rate of starch digestion in the rumen for grains commonly fed in the U.S. Wheat and barley have the fastest rates of starch digestion of the grains whereas dry whole corn and dry rolled milo are generally the slowest. This figure was made without absolute rates because variation within grains and processing may alter the rate of fermentation and, therefore, the order of ranking. Grains with the fastest rates of starch digestion generally cause the most acidosis. Another factor to consider is that slower fermenting grains will also alter the site of digestion from the rumen to the lower tract. Both changes in acidosis and site of digestion could have effects on efficiency of utilization of the grains fed. Stock et al. (1987) have used mixtures of fast and slow fermenting grains to test these hypotheses. Table 2 shows a three trial summary of

Figure 3.

*Grains Categorized By Rate of Breakdown In The Rumen*

Table 2. Effect of feeding mixtures of high moisture corn and dry rolled grain sorghum - three trial summary.

| Item | High moisture corn:dry rolled grain sorghum 100:0 | 75:25 | 50:50 | 0:100 | SE[a] |
|---|---|---|---|---|---|
| | First 28 d | | | | |
| Daily feed[b], kg | 9.40 | 9.15 | 9.35 | 9.42 | .09 |
| Daily gain[c], kg | 1.47 | 1.53 | 1.51 | 1.41 | .05 |
| Gain/feed[b] | .158 | .170 | .164 | .154 | .005 |
| | Entire feeding period | | | | |
| Daily feed[de], kg | 9.32 | 9.31 | 9.53 | 10.07 | .12 |
| Daily gain[f], kg | 1.32 | 1.36 | 1.36 | 1.29 | .03 |
| Gain/feed[de] | .142 | .149 | .143 | .129 | .003 |

[a]SE = common standard error of treatment means.
[b]Quadratic effect (P<.05).
[c]Quadratic effect (P<.14).
[d]Linear effect (P<.01).
[e]Quadratic effect (P<.01).
[f]Quadratic effect (P<.10).

cattle fed high moisture corn in various combinations with milo. The effect of the grain mixtures was apparent within the first 28 days of the feeding period. We feel that acidosis was minimized by including the sorghum at all levels of the mixtures. The inclusion of 25% sorghum exhibited a positive associative effect on gain/feed in the total trial. Starch digestion in duodenally and ileally fistulated cattle revealed that more starch was being digested in the small intestine and total starch digestion decreased as milo made up a greater percentage of the grain fed (Table 3).

Table 3. Starch intake, flow and digestibility.

| Item | High moisture corn:dry rolled grain sorghum 100:0 | 67:33 | 33:67 | 0:100 | SE[a] |
|---|---|---|---|---|---|
| Diet: | 1 | 2 | 3 | 4 | |
| Intake[b], g/d | 3,893 | 3,981 | 4,302 | 4,439 | 62 |
| Digesta flow: | | | | | |
| Duodenal[bc], g/d | 407 | 596 | 1,300 | 2,293 | 192 |
| Ileal[bc], g/d | 156 | 280 | 456 | 768 | 47 |
| Digestion: | | | | | |
| Rumen[bc], % of intake | 89.9 | 85.0 | 68.8 | 45.7 | 4.8 |
| Small intestine[b], % of intake | 6.3 | 7.9 | 20.9 | 37.3 | 5.0 |
| Small intestine, % entering intestine | 67.8 | 53.7 | 45.5 | 54.3 | 20.9 |
| Total tract[bc] | 95.3 | 93.5 | 92.4 | 87.0 | .9 |

[a]Standard error of treatment means.
[b]Linear (P<.01).
[c]Quadratic (P<.10).

INTAKE PATTERNS

Intake patterns are important barometers of subacute acidosis. Fulton and coworkers (1979a) studied intake patterns of cattle being adjusted to high energy diets by increasing concentrate level from 35, 55, 75 to 90% concentrate. Each level was fed for 5 days and then cattle were switched to the next increment level. The grains used in this experiment were dry rolled corn (slow fermenting starch) and hard red winter wheat (fast fermenting starch). Intake patterns (average of days within level) of the corn fed cattle appeared to be smooth and indicative of cattle adjusting normally to a high concentrate diet (Figure 4). Intake of the cattle fed the wheat diets appeared level as intake did not increase. Looking at those data on a day within concentrate level basis revealed that there was a fair amount of variation in intake patterns across each concentrate level in both groups (Figure 5). Cattle would eat and then back off feed for a period of time. Flucuations in intake were evident in both groups, but were not as great for the corn fed cattle until the 90% concentrate level at which time intake decreased sharply. These intake fluctuations were quite pronounced in the wheat fed animals. The wheat fed cattle would eat too much, experience acidosis and reduce intake dramatically for a couple of days. These cattle ate when they overcame the acidosis, but would overeat again, get sick and intake dropped. The wheat fed cattle never really became adjusted to the high energy diets. These data emphasize that looking at average intakes can be misleading. Pen feed sheets are really average intakes of all the cattle in the pen and there may be cattle experiencing wide daily intake fluctuations indicative of acidosis even though pen means are not changing.

Intake patterns of these cattle over a 24-hour period within each concentrate level changed drastically (Figure 6). Cattle fed the corn diet consumed a meal when the diet was offered. Meal size decreased as concentrate level increased, but nevertheless a meal was still consumed. The wheat fed animals ate a meal at the 35% concentrate level, but at

**Figure 4.**

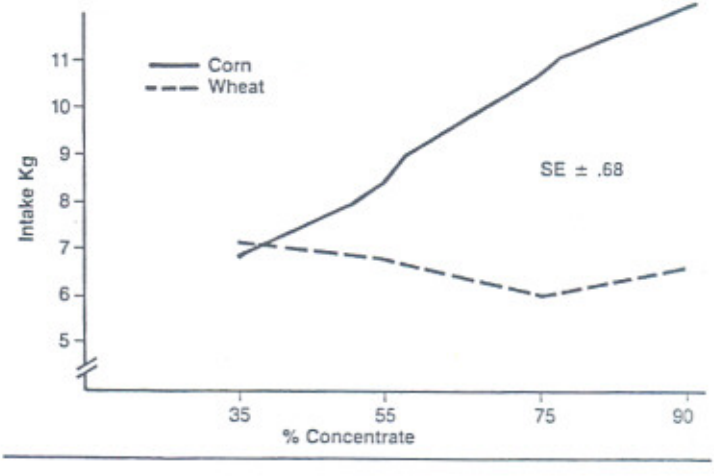


Figure 5.

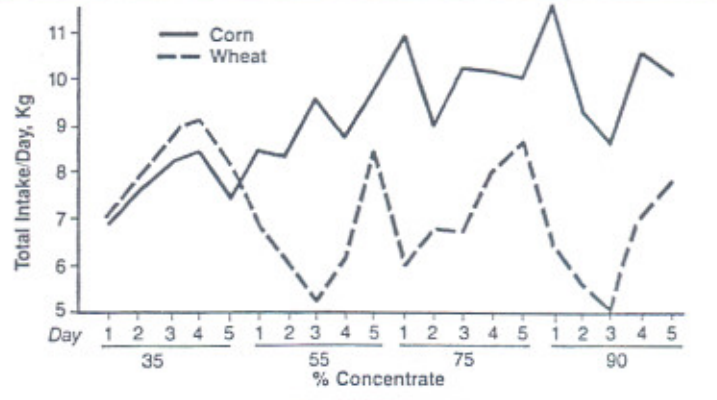
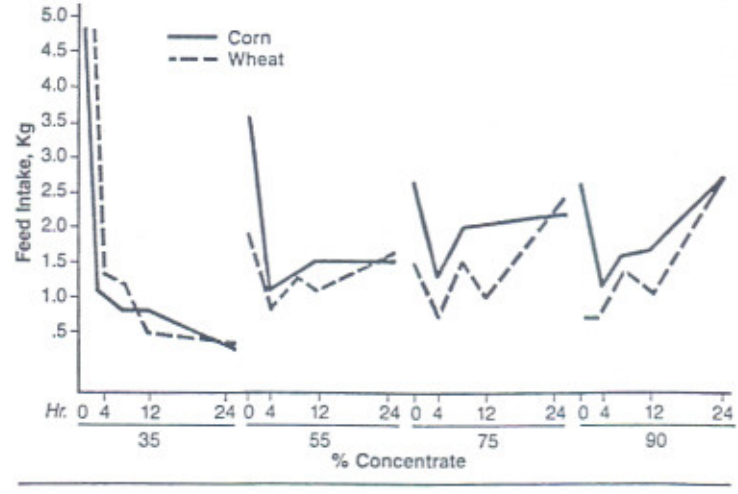



Figure 6.




the higher concentrate levels meals consumed when feed was offered were either very small or absent. The wheat group partitioned feed intake over a longer period of time and ate more during the last 12 hours of the day than they did during the first 12 hours. These cattle became nibblers rather than meal eaters which is indicative of subacute acidosis. Rumen pH averaged across concentrate level (Figure 7) was decreased in the wheat group. Notice the rumen pH in the corn fed cattle averaged about 5.6 until the 90% concentrate level. Rumen pH throughout the day (Figure 8) started lower and remained lower for the wheat fed cattle even though they consumed less feed. The faster fermenting starch in the wheat was converted to acid too quickly for the

Figure 7.

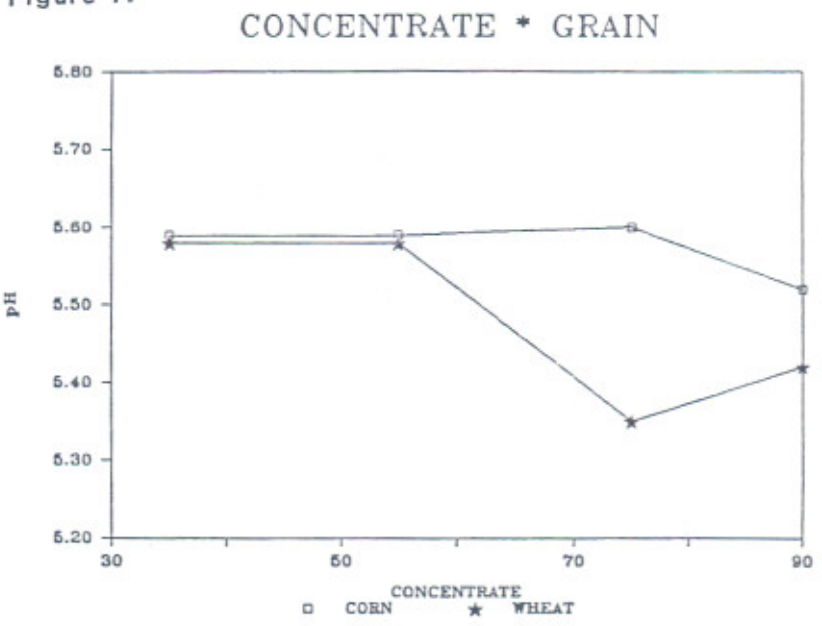
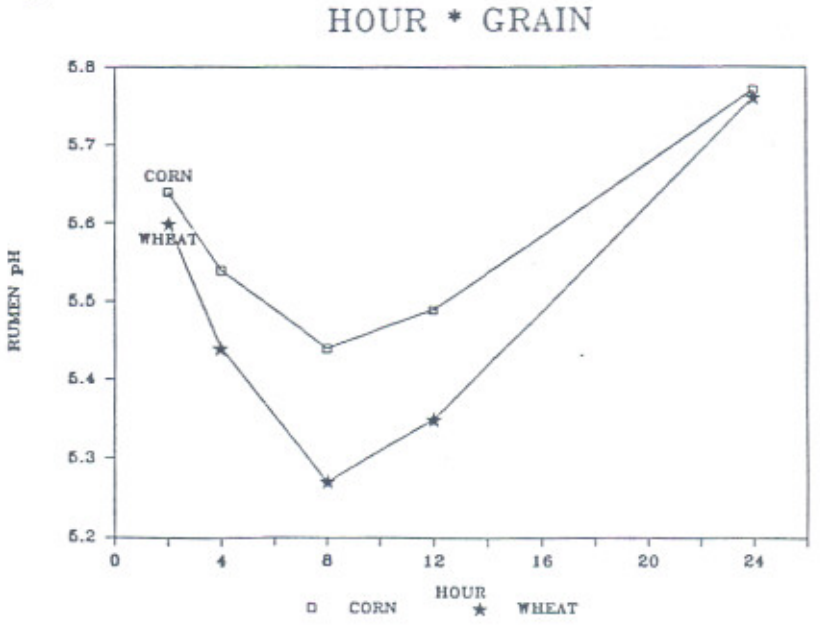



Figure 8.

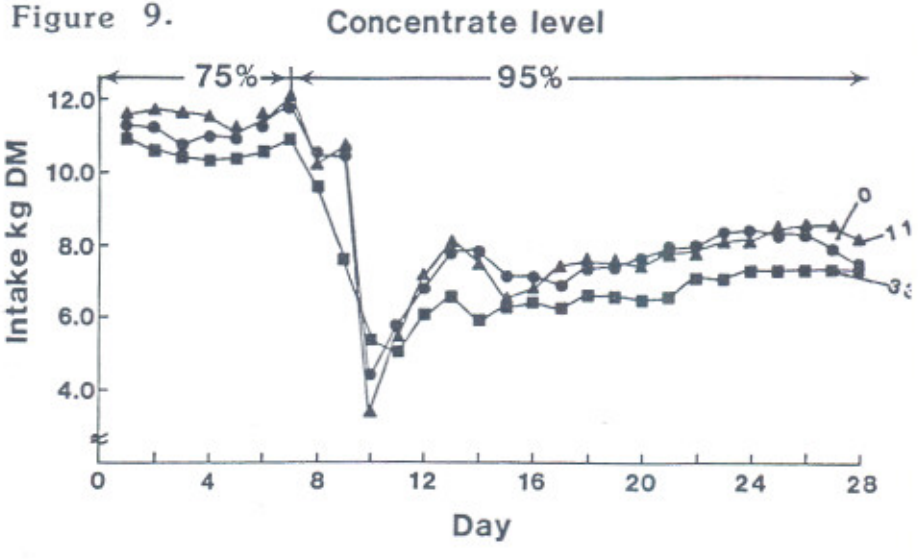


animal to cope. As we evaluated ruminal pH of the wheat fed animals the question arose about what rumen pH was when cattle would begin eating again? Observation of the pH data suggested that the cattle ate when ruminal pH was 5.6. In another experiment by Fulton and coworkers (1979b), ruminal pH was adjusted by infusing sodium and potasium hydroxides intraruminally to maintain pH above 5.6 in animals fed wheat diets. Intake patterns of those cattle fed wheat with rumen pH maintained above 5.6 were similar to the corn fed cattle. The wheat fed cattle did not experience large intake fluctuations. It seems critical to maintain ruminal pH above 5.6 to aid in controlling subacute acidosis.

Another factor that can affect the way feeds and acidosis interact is the use of feed additives. Two antibiotic ionophores on the market today, monensin and lasalocid, have also been shown to be effective in controlling ruminal lactate production in vitro (Dennis et al., 1981) and elevating ruminal pH while reducing ruminal concentrations of lactate and streptococcus bovis during acute acidosis in vivo (Nagaraja et al., 1982). Recently conducted work with monensin and subacute acidosis (Burrin and Britton, 1986) in ruminally fistulated steers. These steers were switched from a roughage diet to a 75% high moisture corn diet containing one of three levels of monensin (0, 150 or 300 mg per day) in one day and ruminal measurements taken for 48 h. Steers experienced subacute acidosis as evidenced by ruminal pH declining to about 5.4 and the presence of diarrhea. Monensin resulted in higher pH and propionate with lower acetate and butyrate concentrations. Total VFA were reduced in the monensin treatment. Ruminal pH was more highly correlated to ruminal VFA (r=-.69) than ruminal lactate (r=-.14).

Feedlot studies with cattle abruptly switched to high concentrate diets containing different levels of monensin (Burrin et al., 1984) show that monensin does not prevent cattle going off feed (Figure 9) when cattle were switched to the 95% concentrate diet. All treatments (0, 11 or 33 ppm monsenin) exhibited similar intake patterns. Closer examination of

Figure 9.



the data revealed that mean variance in daily intake of the cattle fed 33 ppm monsenin was lower than the other treatments (Table 4). After 28 days all cattle were fed the 33 ppm treatment. Cattle started on 0 or 11 ppm ate more total feed than the 33 ppm treatment, but had similar gains and feed efficiency.

Table 4. Influence of monensin level on mean variance in daily intake at various intervals -- trial 1

| | Monensin level, ppm | | | |
|---|---|---|---|---|
| Intervals | 0 | 11 | 33 | Mean |
| Day 1-7 | .85 | .74 | .88 | .82 |
| Day 7-10 | 18.65[a] | 13.19[ab] | 10.05[b] | 14.04 |
| Day 10-16 | 5.20[a] | 4.07[a] | 2.49[b] | 3.94 |
| Day 16-28 | 1.15[a] | 1.17[a] | .79[b] | 1.04 |
| Day 1-28 | 6.26[a] | 5.68[b] | 5.23[b] | 5.73 |

[a,b]Means within row with different superscript differ P<.05.

The data generated suggest that feed additives like lasalocid and monensin can help alleviate the problems of both acute and subacute acidosis, but do not totally remove the problem.

## SUMMARY

Acidosis, rate of starch digestion and intake are closely associated in feedlot cattle. There is a fine line between maximum performance and acidosis. These animals evolved on diets greatly different than what is being fed in feedlots and do not have the proper regulatory mechanisms to prevent acidosis. We think of feedlot cattle being fed for maximum performance as walking a tightrope without a net. Our job as scientists and nutritionists is provide that net so that acidosis can be minimized. Monitoring intake of cattle fed high grain diets is one way to keep our finger on the pulse of the problem. Making ration decisions to maintain proper rumen conditions will help us put that net of safety under the cattle.

## REFERENCES CITED

Baile, C. A. and M. J. Forbes. 1974. Control of feed intake and regulation of enery balance in ruminants. Physiol. Rev. 54:160.

Brent, B. E. 1976. Relationship of acidosis to other feedlot ailments. J. Anim. Sci. 43:930.

Brink, D. R., J. C. Parrott, S. R. Lowry and R. A. Stock. 1985. Steer performance and factors which affect severity ofliver abscesses. National ASAS:698 p. 496.

Burrin, Doug, Rick Stock and Bud Britton. 1984. Monensin level in cattle step-up rations. Nebraska Beef Cattle Report p 10, MP 47.

Burrin, D. G. and R. A. Britton. 1986. Response to monensin in cattle during subacute acidosis. J. Anim. Sci. 63:888.

Fulton, W. R., T. J. Klopfenstein and R. A. Britton. 1979a. Adaptation to high concentrate diets by beef cattle. I. Adaptation to corn and wheat diets. J. Anim. Sci. 49:775.

Dennis, S. M., T. G. Nagaraja and E. E. Bartley. 1981. Effect of lasalocid or monensin on lactate production from in vitro rumen fermentation of various carbohydrates. J. Dairy Sci. 64:2350.

Fulton, W. R., T. J. Klopfenstein and R. A. Britton. 1979b. Adaptation to high concentrate diets by beef cattle. II. Effect of ruminal pH altering on rumen fermentation and voluntary intake of wheat diets. J. Anim. Sci. 49:785.

Harmon, D. L., R. A. Britton and R. L. Prior. 1985. Net portal absorption of lactate and volatile fatty acids in steers experiencing glucose induced acidosis or fed a 70% concentrate diet ad libitum. J. Anim. Sci. 60:560.

Harmon, D. L., R. A. Britton and R. L. Prior. 1984. In vitro rates of oxidation and gluconeogenesis from L (+) and D (-) lactate of bovine tissues. Comp. Biochem. Physiol. 77B:365.

Huber, T. L. 1976. Physiological effects of acidosis on feedlot cattle. J. Anim. Sci. 43:902.

Huntington, G. B. and R. A. Britton. 1978. Effect of dietary lactic acid content and energy level on rumen lactate metabolism in sheep. J. Anim. Sci. 47:241.

Kunkle, W. E., A. W. Fetter and R. L. Preston. 1976. Effect of diet on in vitro and in vivo rumen lactate disappearance rate in sheep. J. Anim. Sci. 42:1256.

Mackie, R. I. and Frances M. C. Gilchrist. 1979. Changes in lactate producing and lactate utilizing bacteria in relation to pH in the rumen of sheep during stepwise adaptation to a high concentrate diet. Appl. Environ. Microbiol. 38:422.

Nagaraja, T. G.,T. B. Avery, E. E. Bartley, S. K. Roof and A. D. Dayton. 1982. Effect of lasalocid, monensin or thiopeptin on lactic acidosis in cattle. J. Anim. Sci. 54:649.

Slyter, Leonard L. 1976. Influence of acidosis on rumen function. J. Anim. Sci. 43:910.

Stock, R. A., D. R. Brink, R. A. Britton, F. K. Goededen, M. H. Sindt, K. K. Kreikemeier, M. L. Bauer and K. K. Smith. 1987. Feeding combinations of high miosture corn and dry rolled grain sorghum to finishing cattle. J. Anim. Sci. (in press).

**Discussion of Paper by Dr. Britton; Chaired by Dr. Holloway**

DeHaan: You have shown that increasing the molar concentration of lactate reduces feed intake. Is this a hormonally mediated response?

Britton: No, there are lactate receptors in the rumen which reduce intake. Baile and Forbes (1984 Physiological Reviews) reported the presence of potent

ruminal receptors for lactate that have not yet been characterized so far as I know.

Brethour: You mentioned nothing about histamine production. This may occur at a low pH. Is it important?

Britton: I omitted histamine because I don't think it is very important. Histamine production in the rumen during acidosis, in our studies, appears to be low. We were often unable to detect histamine in an acidotic rumen. Only with prolonged acidosis did we find histamine in the rumen. Some of the early work that referred to histamine had measured levels of 20 micrograms per ml in animals with prolonged acidosis. Two other factors are important. First, acetylation of histamine is a primary mechanism of detoxifying histamine in the rumen. Rumen microbes carry out this reaction readily. Secondly, with acidosis, ruminal pH is low enough to fully protonate histamine which reduces absorption. Does all this mean that histamine is unimportant in acidosis? Not necessarily. The histamine response seen in the animal is not from absorbed histamine but more probably is from absorbed acids or endotoxins released from lysed gram negative bacteria.

Horn: Does a low rumen pH have postruminal effects? Could the total acid load flowing to the small intestine cause release of gastro-intestinal hormones like secretin which in turn will have powerful effects on ruminal and abomasal motility to decrease feed intake?

Britton: We feel that is sequel may be true, but it hasn't been tested under acidosis conditions. The relationship of those hormones to motility are clear, and a relationship of pH receptors in the rumen to rumen motility are clear. Other factors may be involved. Stress is involved in acute acidosis. With stress, one has the "fight or flight" syndrome. Blood flow is diverted away from the gut which in turn reduces acid absorption. So rumen pH continues to decline more than it should. With sub-acute acidosis, we do not have any blood flow measurements to date.

Fields: Did you measure ruminal pH in animals fed various level of roughage? Are they lower with 5 vs 15% roughage?

Britton: No, we have not measured ruminal pH with all concentrate diets. It probably is quite low.