

JOURNAL OF ANIMAL SCIENCE

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J Anim Sci 1998. 76:275-286.

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Acidosis in Cattle: A Review¹

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ABSTRACT: Acute and chronic acidosis, conditions that follow ingestion of excessive amounts of readily fermented carbohydrate, are prominent production problems for ruminants fed diets rich in concentrate. Often occurring during adaptation to concentrate-rich diets in feedyards, chronic acidosis may continue during the feeding period. With *acute* acidosis, ruminal acidity and osmolality increase markedly as acids and glucose accumulate; these can damage the ruminal and intestinal wall, decrease blood pH, and cause dehydration that proves fatal. Laminitis, polioencephalomalacia, and liver abscesses often accompany acidosis. Even after animals recover from a bout of acidosis, nutrient absorption may be retarded. With *chronic* acidosis, feed intake typically is reduced but variable, and performance is depressed, probably due to hypertonicity of digesta. Acidosis control measures

include feed additives that inhibit microbial strains that produce lactate, that stimulate activity of lactate-using bacteria or starch-engulfing ruminal protozoa, and that reduce meal size. Inoculation with microbial strains capable of preventing glucose or lactate accumulation or metabolizing lactate at a low pH should help prevent acidosis. Feeding higher amounts of dietary roughage, processing grains less thoroughly, and limiting the quantity of feed should reduce the incidence of acidosis, but these practices often depress performance and economic efficiency. Continued research concerning grain processing, dietary cation-anion balance, narrow-spectrum antibiotics, glucose or lactate utilizing microbes, and feeding management (limit or program feeding) should yield new methods for reducing the incidence of acute and chronic acidosis.

Key Words: Acidosis, Grain, Engorgement, Rumen, Osmotic Pressure

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J. Anim. Sci. 1998. 76:275–286

Introduction

By definition, acidosis is a decrease in the alkali (base excess) in body fluids relative to the acid (hydrogen ion) content (Stedman, 1982). Because pH of body fluids is buffered by bicarbonate, the pH of body fluids may or may not be depressed during acidosis, depending on the degree to which bicarbonate compensation is possible. Central nervous system function can be disturbed by low bicarbonate concentrations even if blood pH is not depressed. Although clinical diagnosis of acidosis requires blood pH to fall below 7.35, other clinical signs such as ruminal pH, anorexia, variable feed intake, diarrhea, and lethargy are the routine diagnostic indications of

acidosis of feedlot cattle. The etiology of ruminal and systemic acidosis has been described in excellent reviews by Elam (1976), Huber (1976), Slyter (1976), Britton and Stock (1987), Huntington (1988), Elanco (1993), and Harmon (1996). Highlights are outlined below together with specific hazard control points where alterations might help prevent or alleviate acidosis.

Acidosis of Herbivores

Anaerobic microbes in the rumen and cecum ferment carbohydrates to VFA and lactate. Ruminal production of more than 55 mol of VFA daily has been measured in steers fed feedlot diets (Sharp et al., 1982). Herbivores absorb these organic acids from the rumen and(or) cecum for metabolism by tissues. When carbohydrate supply is increased abruptly (i.e., following grain engorgement or during adaptation to high-concentrate diets), the supply of total acid and the prevalence of lactate in the mixture increase. Normally, lactate is present in the digestive tract at only low concentrations, but when carbohydrate supply is increased abruptly, lactate can accumulate;

¹Presented at a symposium titled "Bud Britton Memorial Symposium on Metabolic Disorders of Feedlot Cattle," July 1996, following the ASAS 88th Annu. Mtg., Rapid City, SD. Financial support was provided by Elanco Animal Health. Approved for publication by the Director, Oklahoma Agric. Exp. Sta. This research was supported under Project H-2123.

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Received September 3, 1996.

Accepted March 27, 1997.

ruminal concentrations occasionally reach 100 mM. Dunlop and Hammond (1965) coined the term "D-lactic acidosis" to encompass this metabolic disturbance variously described as overeating, acute impaction, grain engorgement, founder, and grain overload. Today, the term "acidosis" is used collectively for digestive disturbances of the rumen and intestines. However, acidosis of ruminants often is separated into several forms, including acute, chronic (or subclinical), and subliminal types. Animals exhibit acute acidosis as an overt illness following consumption of readily fermented carbohydrates in amounts sufficient to reduce ingesta pH. With chronic acidosis, feed intake and performance are reduced, but animals may not appear sick. Clinical diagnosis of acidosis depends on measurements of ruminal or blood acidity, with ruminal pH of 5.6 and 5.2 often being used as benchmarks for chronic and acute acidosis, respectively (Cooper and Klopfenstein, 1996). Britton et al. (1991) has used variation in feed intake between days as an index of subclinical or chronic acidosis based on the concept that an increased variability from day to day in feed intake by individual animals is associated with feeding acidotic diets (Britton and Stock, 1987).

Etiology of Acidosis

The relevant steps involved with acid production in and output from the rumen are illustrated in Figure 1. For discussion purposes, reactions have been numbered.

Starch Concentration and Conversion to Glucose (Items 1 and 2)

Acidosis is most prevalent following engorgement of large amounts of starch or other rapidly fermented carbohydrate. Excessive intake of readily fermented starch often occurs when animals are first being adapted to a high-concentrate (feedlot) diet and(or) when animals are switching from bulk fill to chemostatic intake regulation. Acidosis also can occur when grazing animals are fed a large amount of a starch-rich supplement.

Rate of cleavage of starch to glucose varies with grain source, grain processing, and starch type. Certain grain sources (i.e., wheat) and grain varieties with more readily extracted starch, as preferred by distilleries, presumably are hydrolyzed to glucose more rapidly than other sources or varieties. Starch granules embedded in protein in the "horny endosperm" of milo and corn have less surface exposed for microbial attack. Heat and pressure treatment explodes starch granules into sheets of starch that are fermented very rapidly. Heat and pressure processing, particle size reduction, and high-moisture storage of grain increase starch availability and the propensity for acidosis (Johnson et al., 1974; Britton and Stock,

1987; Reinhardt et al., 1993). Glucose is liberated from starch granules by specific strains of microbes that attach to the grain particles. Several methods have been developed to quantify flake quality (e.g., test weight, birefringence, gas production rate during incubation with yeast or ruminal contents, and glucose or maltose release during incubation with amyloglucosidase or amylase). These should reflect the extent of exposure of starch and(or) its rate of fermentation. For maximum energetic efficiency, a *high extent* of fermentation is desired. But for acidosis prevention, a *slow rate* of fermentation is preferred. Unfortunately, among grain sources and processing methods, rate and extent of digestion typically are correlated in a positive direction.

Traditionally, glucose has not been considered to be an important metabolic intermediate in the rumen because ruminal concentrations normally are extremely low. However, in incubations by Slyter (1976) and engorgement studies by Horn et al. (1979), glucose concentrations in the rumen often exceeded 160 mg/dL, a concentration greater than that found in blood. In one of our acidosis studies, ruminal glucose exceeded 1,400 mg/dL. Glucose is liberated from starch by amylase, but whether this elevated concentration is simply a result of more rapid hydrolysis or of a reduction in the rate of glucose utilization by ruminal microbes is not clear.

Presence of free glucose in the rumen can have at least three adverse effects. First, ruminal bacteria that normally are not competitive can grow very rapidly when provided with high amounts of glucose. *Streptococcus bovis*, an inefficient microbe that thrives only when free glucose is available, was proposed by Hungate (1968) as the major culprit in lactic acidosis. However, concentrations of this organism in the rumen of cattle fed high-concentrate diets are very low (Leedle, 1993). Other bacteria, those directly involved with starch fermentation, may be more important sources of lactate. Indeed, lactate often accumulates faster *in vitro* from starch than from glucose. Second, other opportunistic microbes, including coliforms and amino acid decarboxylating microbes, may thrive in the rumen of cattle fed concentrate diets (Slyter and Rumsey, 1991; Leedle, 1993) and produce or, during lysis, release endotoxins or amides (e.g., histamine; Huber, 1976; Brent, 1976) when glucose is readily available. Third, free glucose released from starch increases the osmolality of ruminal contents. An increased osmolality exacerbates accumulation of acid within the rumen by inhibiting VFA absorption.

Limiting the Supply of Starch and Glucose (Items 1 and 2)

Two common management practices that help to prevent acidosis are diluting the diet with roughage or modulating intake of starch. Dietary roughage decreases eating rate and meal size. Increasing the

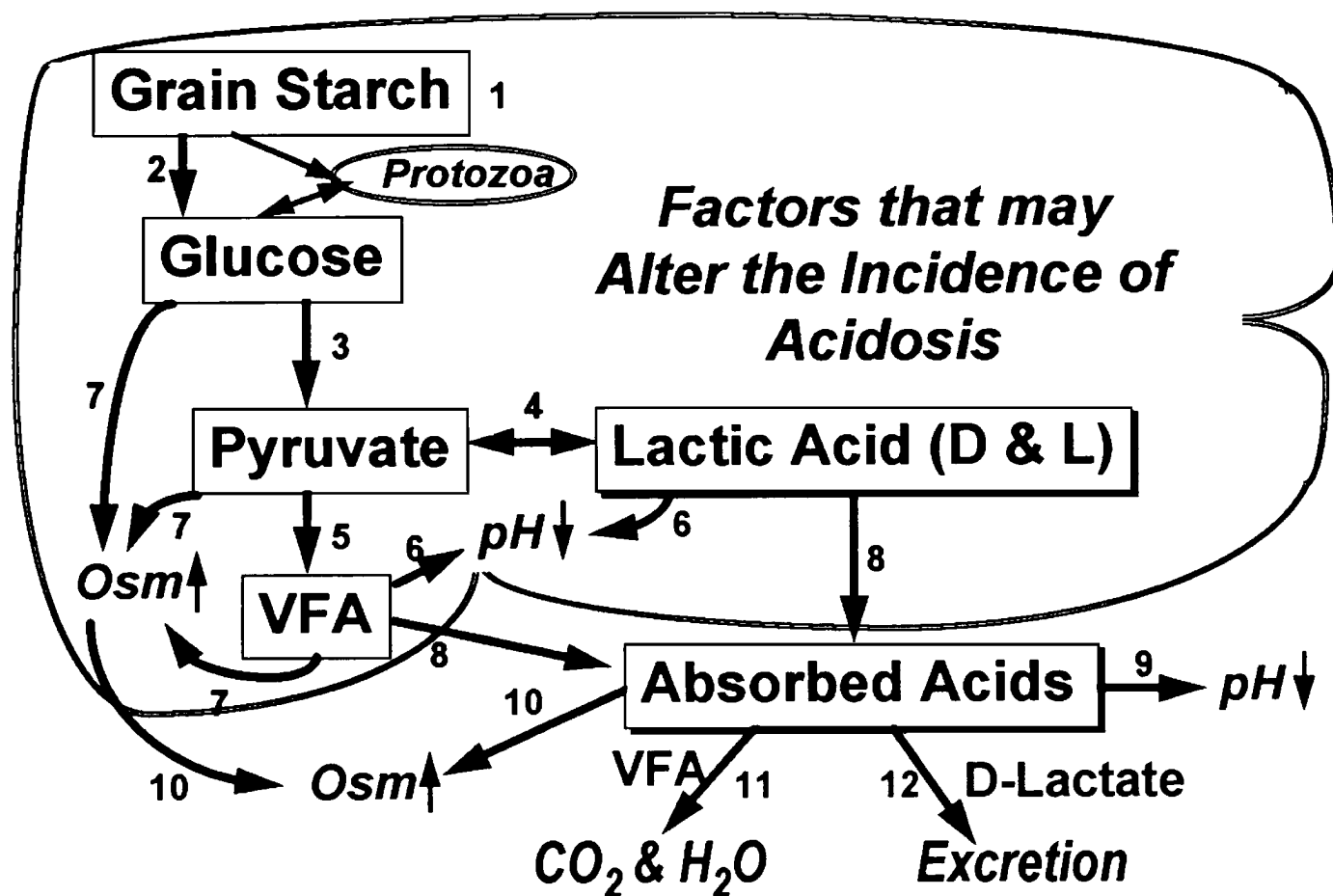


Figure 1. Key reactions in acidosis of ruminants. Individual numbered reactions are discussed in the text.

concentration of dry roughage increases chewing time and saliva production. Although an increased extent of mastication will decrease size of grain particles entering the rumen and thereby increase its rate of fermentation, an increased input of buffers from saliva from a longer chewing time or rumination neutralizes and dilutes ruminal acids. Starch content of the diet also can be reduced by substituting starch-extracted concentrates (e.g., distilling or brewing co-products and middlings) for cereal grains. Total diet intake also can be restricted by using a limited maximum intake feeding scheme as described by Preston (1995).

For experimental purposes, researchers often induce acute acidosis by withholding feed for 12 to 24 h and then feeding (or ruminally dosing) 150% of the normal day's feed allotment. This shows how an increased meal size can precipitate acidosis and has led to the suggestion that daily variation in feed intake among days within an animal will increase the potential for acidosis. Regularity of intake also has been implicated as a sign of "subclinical" acidosis. Fulton et al. (1979) observed that following a bout of acidosis, feed intake by animals typically is low; they suggested that a cyclic feed intake pattern reflected repeated bouts of acidosis. When animals are fed

individually, such fluctuations in intake are detected readily. However, when 20 or more animals are fed together, daily fluctuations in intake (or feed delivered) may not be detected unless all animals experience acidosis at the same time, as can happen following diet changes or mishaps in processing or mixing.

Effects of feed intake regularity on acidosis have been examined in trials from New Mexico, California, and Nebraska (Galyean et al., 1993; Zinn, 1994; Cooper and Klopfenstein, 1996). In these trials, feed supply for the pen or the animal was purposely altered or meals were skipped. Although altering the daily supply of feed has adversely altered feed efficiency slightly, and performance was reduced in the New Mexico trial, animal health was not affected drastically. Stock and Britton (1993), Stock et al. (1995b), and Cooper and Klopfenstein (1996) indicated that monensin and monensin-tylosin combinations reduced daily variation in feed intake by feedlot steers. Including monensin in the diet has reduced the incidence of digestive deaths in pens of feedlot cattle (Parrott, 1993; Vogel, 1996), presumably due to inhibition of certain lactate-producing bacteria and

reduced daily variation in feed intake (Cooper and Klopfenstein, 1996).

Meal frequency may be as important as total feed intake as a cause of acidosis. For example, cattle with implants typically have greater feed intakes. Weather changes and processing cattle to provide implants or inoculations often disrupt feeding patterns and may result in overconsumption and acidosis. Proper timing of processing so that cattle are not deprived of feed may be useful; intake restriction following working or weather changes also may be beneficial. Estrogenic implants have been shown to increase meal frequency, which in turn may decrease the potential for acidosis. Effects of meal frequency also may explain why more timid animals and certain breeds experience acidosis more frequently. But if meal frequency is important, the incidence of acidosis would be expected to be higher when cattle are limit- or program-fed. To date, acidosis incidence has not been reported to be increased by limit feeding, perhaps because the total quantity of feed supplied is not excessive. However, when excessive amounts of feed are provided, either mistakenly or during the switch from limit feeding to free-choice intake, acidosis might be expected.

The roles of ruminal protozoa in acidosis are not clear. By engulfing starch particles and storing glucose as polysaccharide, protozoa delay starch fermentation by bacteria, help to retard acid production, and stabilize ruminal fermentation (Slyter, 1976; Nagaraja et al., 1990). In view of the large amounts of starch consumed by ruminants, the quantitative significance of starch consumption by protozoa seems questionable. However, the population of ruminal bacteria normally decreases when protozoa are present; this decrease also could delay fermentation. Protozoal numbers in the rumen typically decline when high-concentrate diets are fed, probably because long dietary fiber provides a fibrous mat in the rumen to which protozoa attach and remain long enough to replicate. Free fatty acids and detergents reduce protozoal numbers, as well, and a low pH may cause defaunation. However, in addition to stabilizing normal fermentation, protozoal presence in the rumen can be deleterious. Because they have much higher amylase activity per unit of protein than bacteria (Mendoza and Britton, 1991), protozoa, when rupturing due to changes in acid or osmolality associated with acidosis, release large amounts of amylase that in turn accelerates glucose production from starch and increases the likelihood of acidosis.

Protozoal stimulants or inhibitors, as indicated in Figure 1, may have an impact on propensity and seriousness of acidosis. Because protozoal numbers are reduced by high-concentrate diets and removed by unsaturated fatty acids, high concentrations of dietary fat often lead to ruminal instability. Huffman et al. (1992) suggested that by coating the grain and reducing its rate of fermentation, supplemental fat should reduce the incidence of acidosis. However, in

vivo challenge studies with corn and wheat detected no effect of fat level on time that pH fell below 6.0, suggesting that fat was ineffective in preventing subacute acidosis (Krehbiel et al., 1995b).

Including lactobacillus cultures in the diet may prolong ruminal retention of protozoa (Van Koeveering et al., 1994), attenuate fermentation and production of ruminal lactate, and help maintain a higher ruminal pH (Cooper and Klopfenstein, 1996). Williams et al. (1991) observed that the mean and peak L-lactate concentration in ruminal fluid of steers fed a barley-hay diet was lower and ruminal pH was higher when the diet was supplemented with a yeast culture. Because yeasts fail to compete and grow in the rumen, frequent dosing is necessary to maintain activity. In engorgement studies, a yeast culture did not alter the fermentation pattern (Godfrey et al., 1992), but specific yeast cultures, through stimulating growth of lactate utilizing bacterial strains in the rumen (Dawson, 1995), may help moderate ruminal pH and avoid acidosis.

Glycolysis (Reaction 3)

Anaerobic microbes typically thrive when free glucose is available. Yet, the fact that free glucose concentrations in the rumen are high during acidosis indicates that glycolysis may be partially blocked. In our ruminal fluid incubation studies and those of others (A. Z. Leedle, personal communication), less than half of the glucose incubated with ruminal contents (1% wt/vol) disappeared within 6 h; this supports the concept that free glucose is not being catabolized readily for reasons yet unknown.

Control of Glycolysis (Reaction 3)

Rate of glycolysis can be limited by inhibiting hexokinase, phosphofructokinase (both of which use ATP), and pyruvate kinase (that yields ATP); lack of oxidized NAD (that is regenerated with lactate production) also can limit glycolysis. Certain metabolic inhibitors, including iodoacetate, fluoride, and metabisulfite, by retarding glycolysis, have been proposed to reduce ruminal acidosis.

Volatile Fatty Acid Production and Lactate Production and Utilization (Reactions 4 and 5)

Bacteria in the rumen often are classified as "lactate producers" or "lactate users." Balance between these two groups determines whether lactate accumulates. End products of bacterial strains may change depending on substrate availability and culture conditions (Russell and Hino, 1985). Most lactate-using microbes are sensitive to low pH, whereas most lactate producers are not. Under anaerobic conditions, pyruvate is converted to lactate to regenerate the NAD used in glycolysis. Under "normal" conditions, lactate does not accumulate in the rumen at concentrations

above 5 μM . In contrast, ruminal concentrations exceeding 40 mM are indicative of severe acidosis. Ruminal and silage microbes produce two forms of lactate, the D+ and L form. The L form, identical to that produced from glucose by exercising muscle, can be readily metabolized by liver and heart tissue. In contrast, D+ lactate, typically 30 to 38% of the total lactate found in the rumen, is not produced by mammalian tissues. Accumulation of free lactate in silage serves to halt fermentation and stabilize the mass. In addition to D-lactate and VFA being involved with acidosis, other microbial products including ethanol, methanol, histamine, tyramine, and endotoxins often are detectable during acidosis and can exert systemic effects (Koers et al., 1976; Slyter, 1976).

Conversion of pyruvate to VFA involves multiple steps and generates approximately half the ATP for microbial growth in the rumen; the other half is derived from conversion of glucose to pyruvate. Normally, VFA do not accumulate at sufficient concentrations in the rumen to reduce pH drastically. However, when the rate of acid production exceeds the rate of acid absorption, due either to rapid production, inhibited absorption, or reduced dilution, VFA accumulate to higher concentrations. In some studies, ruminal pH falls below 5.0 even without lactate being present. This has led to the suggestion that total acid load, not lactate alone, is responsible for acidosis (Britton and Stock, 1987), particularly with chronic acidosis.

Control of Lactate Production and Utilization (Reactions 4 and 5)

Streptococcus bovis and lactobacilli, which produce lactate, the coliforms, which seem responsible for anaphylactic shock and sudden death, and the amino acid degrading microbes associated with tyramine and histamine production all may contribute to ruminal acidosis; these organisms might be controlled with antibiotics or bacteriophages. Inoculation with lactate-using microbes that can tolerate a low pH also should be useful for preventing acid accumulation. Repeated inoculation with *Megasphaera elsdenii* (Kung and Hession, 1995), *Lactobacillus acidophilus* (in metabolism trials, e.g., Huffman et al., 1993; Van Koeveering et al., 1994; but not feeding trials, e.g., Klopfenstein et al., 1995; Stock et al., 1995a), and the three species present in Activated Rumen Microbes (ARM) that Grace, Inc. tested extensively may enhance lactate utilization. Unfortunately, survival in the face of vigorous competition from other microbial species complicates long-term alteration of the ruminal microflora. Yet, individual animals differ and may remain consistently different in ruminal metabolism. During a 6-mo ad libitum feeding period, lactate production by ruminal contents during incubation with cornstarch ranked 10 steers similarly, suggesting that the mixture of microbial species within an animal

remains stable even though some animals remained more prone to acidosis (F. N. Owens, unpublished data).

To decrease lactate concentrations, stimulation of microbes that use lactate (e.g., *Selenomonas ruminantium*) seems beneficial. Dosing cultures or animals with certain dicarboxylic acids, particularly fumarate and malate, has decreased lactate production and increased pH in vitro and following grain engorgement, perhaps through enhanced lactate utilization by *S. ruminantium* (Martin and Streeter, 1995). Feasibility of malate supplementation during diet adaptation to increase lactate utilization needs further testing. An interesting alternative to microbial control is to precondition microbes to handle lactate, either by including lactate in the diet or feeding ensiled feeds that contain lactate. Adding lactate to the adaptation diet for sheep increased the rate of disappearance of lactate in vitro (Huntington and Britton, 1978), suggesting that feeding diets that contain lactate before an acidotic challenge may be beneficial.

Feeding ionophores has reduced lactate production in vitro and in vivo (Newbold and Wallace, 1988; Bauer et al., 1992; Syntex, 1994); effects may be either through inhibition of lactate-producing bacteria or reduced meal size. For inhibiting lactobacilli, Tetronasin seems more potent than monensin. Selective inhibition of *S. bovis* by thiopeptin indicates that controlling the microbial population can prevent acidosis. When cattle were fed virginiamycin they could be switched uneventfully from a forage diet to a 100% wheat diet within 24 h (Zorrilla-Rios et al., 1991). Virginiamycin may protect animals from ruminal and postruminal acidosis (Godfrey et al., 1992) by inhibiting lactate-producing bacteria (Rogers et al., 1995).

In addition to lactate, substances toxic to ruminal microbes are produced by certain strains of ruminal bacteria grown with excess carbohydrate but insufficient nitrogen (Russell, 1993). Under "glucose toxicity" conditions and accumulation of methylglyoxal, viability of bacteria declines drastically (Russell, unpublished data). With lysis of microbial cells, endotoxins also may be released. Presence of such antimicrobial compounds might readily explain the stagnancy of ruminal contents immediately following a bout of acidosis. Antibacterial activity of ruminal fluid during acidosis deserves further research attention. Toxicants might be neutralized through inoculation with toxin-catabolizing or toxin-tolerant strains of microbes, and conditions conducive to toxin production, such as a deficiency of specific nitrogenous compounds, might be corrected by diet modification.

Depression of Ruminal pH (Reaction 6)

Ruminal pH represents the consortium of relative concentrations of bases, acids, and buffers. The primary ruminal base is ammonia. The two primary

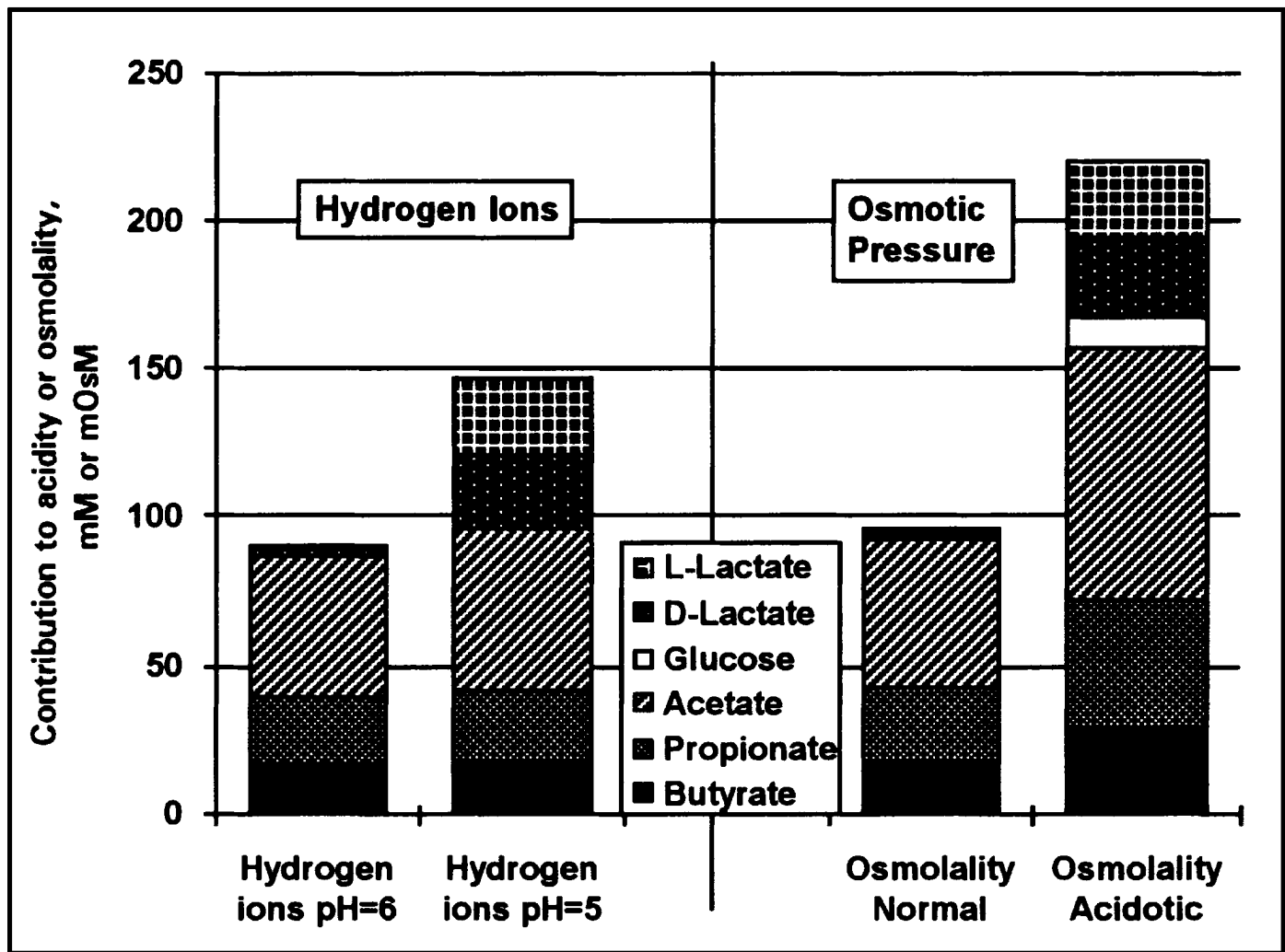


Figure 2. Relative contributions of various organic compounds to ruminal acidity and osmolality under normal or acidotic conditions. Heights of bars indicate relative contributions to hydrogen ion and osmolality from (bottom to top) butyrate, propionate, acetate, glucose, D-lactate, and L-lactate.

buffers under neutral pH conditions are bicarbonate and phosphate. In addition, VFA and lactate act as buffers when pH falls below 5, as discussed by Counotte et al. (1979). The relative contributions of various organic compounds to ruminal acidity and to ruminal osmolality under normal and acidotic conditions are presented in Figure 2 based on ruminal concentrations and calculated extent of ionization based on data published by Fulton et al. (1979) combined with glucose measurements from Horn et al. (1979). As indicated in Figure 2, when pH decreases to 5.0 during acidosis, ionization of acids increases slightly, but the added lactate is primarily responsible for the increased hydrogen ion concentration. Lactate depresses pH more drastically than similar amounts of other ruminal acids because its pK (the pH point of maximum buffering) is considerably lower (3.8 vs 4.8). As shown in Figure 2, with an acidotic pH, osmotic pressure is increased by greater ionization of

acids and presence of free glucose. Compared with normal concentrations, the change during acidosis is much greater in osmolality than in the hydrogen ion concentration.

Absorption from the rumen normally prevents acid accumulation; however, high osmolality of ruminal contents reduces the rate of acid absorption (Tabaru et al., 1990). This exacerbates acidity and osmolality. Furthermore, acidity enhances activity of lactate dehydrogenase, increasing conversion of pyruvate to lactate and complicating recovery from acidosis. Combined with the fact that a low pH enhances activity of pyruvate hydrogenase and favors pyruvate conversion to lactate (Russell and Hino, 1985), a severe drop in ruminal pH is difficult to reverse.

Control of Ruminal pH (Reaction 6)

Increasing ruminal input of bases or buffers (e.g., bicarbonate from the diet or from saliva) or feeds that

yield bases or buffers (e.g., ammonia from degraded protein or nonprotein N) will help prevent a depression in ruminal pH. Absorption of VFA, by removing un-ionized acid and by the exchange of ionized VFA for bicarbonate during the absorption process (Stevens, 1970), aids in maintaining pH near neutrality. Consequently, a reduced rate of VFA absorption causes ruminal pH to drop for two reasons: ruminal VFA accumulate and bicarbonate input from the blood stream is decreased. Approximately half the bicarbonate entering the rumen comes from saliva, during eating and rumination; the other half enters the rumen in exchange for ionized acids being absorbed. In addition, carbon dioxide produced during fermentation typically saturates ruminal fluid and exchanges with the bicarbonate pool of the rumen. With concentrate diets and reduced input of saliva, a higher proportion of bicarbonate must be derived from the blood. This reduces the base excess of blood; if inadequately compensated by respiratory and renal mechanism, this causes metabolic acidosis.

Ruminal Osmolality (Reaction 7)

Osmotic pressure pulls or pushes water through membranes depending on relative concentrations of dissolved materials. Ruminal osmolality normally ranges from 240 to 265 mOsm/L with roughage diets and 280 to 300 mOsm/L with concentrate diets (Garza et al., 1989). Minerals, VFA, lactate, and glucose are the primary solutes in ruminal fluid. In blood, dissolved protein contributes substantially to osmotic pressure that normally ranges from 285 to 310 mOsm. With the acidotic conditions of engorgement studies, we have measured ruminal osmolality as high as 515 mOsm. When ruminal osmolality is markedly greater than blood osmolality, water from blood is drawn rapidly inward through the rumen wall. Rapid influx to neutralize osmotic pressure swells the ruminal papillae and can pull patches of the ruminal epithelium into the rumen by stripping the internal surface layers of the rumen wall from the underlying layers, as illustrated vividly in histological studies by Eadie and Mann (1970). Damage to the wall of the rumen or small intestine due to high osmotic pressure, detected later as sites of abscesses, is a result of this rapid influx of water. When sepsis occurs, ruminal microbes responsible for liver abscesses freely enter the blood stream. Subsequently, repaired tissues of the digestive tract will be thickened (hyperkeratosis or parakeratosis); this may inhibit rate of VFA absorption for months or years after the damage has occurred (Krehbiel et al., 1995a). Passage of VFA postruminally for absorption is possible, but abomasal presence of VFA hinders acidification and protein and mineral digestion; this may reduce postruminal starch digestion. Consequently, a single bout of non-fatal acidosis may have prolonged effects; this may explain

why subsequent performance is suboptimal for many of the animals denoted as "realizers" in feedlots.

An elevation in osmotic pressure in the rumen is sensed by the wall of the reticulorumen to inhibit feed intake (Carter and Grovum, 1990). In addition, osmotic pressures above 350 mOsm inhibit bacterial digestion of fiber and starch, causing ruminal contents to become stagnant. High osmolality (> 300 mOsm) combined with distention of the abomasum, through inhibition of outflow, complicates removal of fluid and acid from the rumen (Scott, 1975). During a 22-d trial adapting three heifers to a high-concentrate diet, we observed that ruminal osmolality averaged 339 ± 35 mOsmol and peaked over 420 mOsm on several days. Although ruminal hypertonicity usually but not always reduces the frequency of ruminal contractions (Carter and Grovum, 1990), inhibited gut motility or hypertonicity at the abomasum may halt flow and exacerbate ruminal acidification; altered motility or tonicity also may cause feed intake to fluctuate with chronic acidosis.

Control of Osmolality (Reaction 7)

High osmolality from elevated concentrations of glucose and acids cannot be readily prevented. However, other contributors to osmotic pressure, such as ammonia and soluble minerals (i.e., sodium, potassium, chloride) from the diet or water can be altered. Salt at 5% of the diet increased ruminal osmolality to 344 mOsm. Reducing intake of minerals and salt might reduce ruminal osmolality slightly. Increasing input of saliva, at approximately 255 mOsm, also reduces ruminal osmotic pressure. High moisture diets or increasing intake of drinking water probably do not reduce ruminal osmolality because fermented diets often have high osmolality, and drinking water intake may partially flush past the rumen (Garza and Owens, 1989). Because high intakes of salt or minerals in feed can exacerbate the situation, anorexia during acidosis should be beneficial.

Acid Absorption (Reaction 8)

Lactate and VFA are absorbed passively through the rumen and intestinal epithelium. Rate of absorption is greater when concentrations are high, pH is low, and osmolality is normal (Tabaru et al., 1990). The lower the pH, the higher the percentage of each organic acid in the non-dissociated (acid) form and the greater the absorption rate. During absorption, butyrate is partly metabolized as an energy source for the rumen wall and glucose is partly converted to D-lactate. Lactate also is produced in and absorbed from the intestines (Godfrey et al., 1992), so total lactate load for the liver may greatly exceed the lactate absorption from the rumen.

Control of Acid Absorption (Reaction 8)

Whether it is desirable to increase acid absorption from the rumen to avoid ruminal acid overload and osmolality or to decrease absorption and temporarily avoid systemic acidosis depends on the potential severity of the ruminal or systemic damage. On a chronic basis, acid absorption rate is reduced most easily by increasing ruminal pH. Higher bicarbonate input from the diet or saliva will help increase ruminal pH and an increased pH stimulates utilization of lactate in the rumen. Higher ruminal ammonia concentrations from higher dietary protein or urea concentrations often are associated with a higher ruminal pH and ruminal buffering capacity (Haaland et al., 1982) even though the ammonia released could neutralize only 10 to 15% of VFA produced. Increasing the cation:anion balance also may be useful to increase ruminal pH. However, in research with acute acidosis induced by a grain overload, including various buffers with the diet has not produced a consistent benefit. This probably relates to the long time lag (6 to 10 h) between a meal and minimum ruminal pH. When combined with outflow of soluble materials from the rumen, buffers are diluted too much to have any residual effect. Protein or nonprotein N sources that release ammonia gradually may be beneficial, certainly more beneficial than urea or bypass protein sources. Enhanced saliva flow, achieved through enhancing chewing and rumination time by including long roughage in the diet or by addition of specific chemicals to the diet (e.g., slafamine and pilocarpine) should reduce the incidence of acidosis.

Blood pH (Reaction 9)

Blood acidosis, which is similar in ruminants and nonruminants, can result from either excessive production or insufficient removal of acid. With respiratory inadequacy, as can occur during respiratory disease, carbon dioxide accumulates in blood; this depresses blood pH unless renal retention of bicarbonate compensates sufficiently. In contrast, with metabolic abnormalities, excess acid production or absorption decreases pH and bicarbonate in body fluids due to accumulation of acids or loss of fixed base from the body (as in diarrhea or renal disease). In nonruminants, lactic acidosis is a special type of metabolic acidosis resulting from decreased tissue perfusion, drug reaction, or inhibition of pyruvate conversion to acetyl coenzyme A that can occur in deficiencies of specific cofactors (i.e., thiamin or lipoic acid). With arsenite or mercury binding of sulfhydryl groups of lipoic acid, pyruvate and lactate accumulate. Nutritionally deprived alcoholics that are thiamin-deficient when administered glucose rapidly accumulate pyruvate, and the lactic acidemia that results frequently is lethal. In humans, acidosis can occur with high-protein diets with inhibited metabolism of

either propionic acid (from a B₁₂ deficiency) or isovaleric acid (impaired isovaleryl dehydrogenase) as described by Murray et al. (1993).

Blood pH depends on the relative concentrations of bases, acids, and buffers in solution. Low blood concentrations make ammonia irrelevant to blood pH, except that the liver by urea synthesis may alter acid-base status of the animal. Phosphate plays a minor role due to its relatively low and constant concentration. This leaves bicarbonate as the primary blood buffer (Counotte et al., 1979). A base-excess normally is present in blood, but an acid load can decrease this base-excess and can overcome the buffering capacity of bicarbonate. Acids of concern include those absorbed from the digestive tract plus L-lactate produced by muscular activity. Acid absorption or production is of concern only at the site of absorption or when acids accumulate (i.e., when rate of entry exceeds the rate of metabolism). Of the VFA, only acetate normally reaches the peripheral blood stream; much of the butyrate is converted to beta-hydroxybutyrate during absorption through the rumen wall and all of the propionate is converted to glucose by the liver. Presumably, VFA should not accumulate in blood plasma at sufficient concentrations to depress blood pH, but exactly how blood VFA concentrations change under acidotic conditions has not been determined. However, metabolism of the ruminal wall and the liver may be compromised during acidosis. Further complicating the situation, the liver is faced with L-lactate from tissue metabolism plus the D- and L-lactate absorbed from the digestive tract. Indeed, when ruminal glucose concentrations are high, as seen with acidosis, glucose being absorbed is partially converted to L-lactate by the digestive tract (Seal and Parker, 1994); if excessive, capacity of the liver to catabolize lactate may be overloaded (Naylor et al., 1984). Individual animals with a larger (wheat-pasture cattle) or more adapted liver have greater capacity for metabolizing lactate and thereby may be less likely to experience blood acidosis.

Blood usually is saturated with bicarbonate. Blood acid-base balance is regulated by the respiratory system (blowing off carbon dioxide), muscular activity (lactate production by muscles), and kidney function (excretion of acid or ammonium salts), as described by Harmon (1996). Balance of absorbed ions can alter acid-base status. Normally determined by measuring concentrations in feed and designated as the dietary cation-anion balance (**DCAB**), cations (sodium, potassium, and perhaps ammonia for ruminants) increase the base load, whereas anions (chloride, and probably sulfate for ruminants) increase the acid load. Cattle fed cereal grain diets typically are acidotic and secrete acid urine partially neutralized by ammonia and phosphorus. Infused acid increases excretion of ammonium ions but not phosphorus, indicating that phosphorus excretion is inconsequential to acidosis and may reflect decreased recycling via saliva to the

rumen. An increased dietary electrolyte balance has been found to increase ruminal pH of growing steers and to linearly or quadratically increase arterial blood pH (Ross et al., 1994a,b).

Control of Blood pH (Reaction 9)

Increasing the base excess, through altering the cation-anion balance of the diet, should aid in preventing acidosis. Substitution of buffers (e.g., carbonates and bicarbonates) for other dietary ions often proves beneficial, probably more from the added cation than from carbonate or bicarbonate that are lost as carbon dioxide from the rumen, especially when pH is low. This reduces the impact of added buffer on blood pH or base excess. In contrast, higher levels of urea or ruminally degraded protein, through metabolism by the liver, may enhance base status. To avoid elevating ruminal osmolality, cations should be readily absorbed (Na, K) rather than maintained in the rumen (Ca, Mg) in order to increase blood base status although excesses may increase blood osmolality and reduce feed intake. Manipulation of DCAB may be helpful for increasing feed intake and preventing blood acidosis (Ross et al., 1994a,b; Block, 1994). Because ammonium salts are excreted to counterbalance an acid load, additional sources of ammonia (i.e., degraded protein or NPN) may be needed and beneficial in controlling chronic acidosis. Urinary ammonia, which is derived from glutamine, increases the hepatic demand for ammonia and energy for glutamine synthesis.

Blood Osmolality (Reaction 10)

Blood osmolality increases during acidosis for two reasons. First, high ruminal osmotic pressure pulls fluid from plasma into the rumen; this concentrates blood components, increasing both blood osmolality, packed cell volume, and drinking of water, symptoms typical of dehydration. Second, when rates of absorption of ruminal acids or glucose exceed their rates of metabolism or excretion, these compounds can accumulate in blood and directly increase osmolality. Hoof damage and laminitis typical of acidosis have been attributed to elevated histamine concentrations and blood vessel damage due to uncontrolled elevations in blood pressure inside the hoof (Vermunt and Greenough, 1994). Whether blood osmolality plays a role in laminitis is not clear. Dark lines around the hoof and rough surface bands readily reveal an historical record of the animal's nutritional status, stress, and acidosis.

Increased osmolality has numerous physiological effects. Hypertonicity is detected at several sites. It is sensed by osmoreceptors in the rumen to inhibit feed intake, in the portal system or liver to inhibit rumination, and in the brain to inhibit salivation (Carter and Grovum, 1990). The short-term feed

intake depressions noted with "subclinical" acidosis may reflect fluctuating blood osmolality. In contrast, with clinical or acute acidosis, the decrease in blood pH is a life-threatening situation. Hence, acute and chronic acidosis, although working from a similar base of ruminal acidity, probably should be considered as separate disorders.

Control of Blood Osmolality (Reaction 10)

Increased salivation, achieved through higher intakes of coarse particles from roughage or whole corn, and higher intake of water should serve to buffer and dilute ruminal contents to reduce ruminal osmolality. Reduced ruminal osmolality will avoid the influx of fluid from blood that causes an elevation in blood osmolality. Similarly, supply of cofactors involved with VFA metabolism (B₁₂, thiamin, and lipoic acid) must be adequate to prevent accumulation of these acids.

Acid Metabolism and Excretion (Reactions 11 and 12)

Metabolism of VFA to glucose for storage or to carbon dioxide to yield energy typically proceeds rapidly so that blood concentrations remain low. Traditionally, acidosis was attributed strictly to lactate because lactate is 10 times stronger an acid than VFA. Huber (1976) reviewing earlier studies indicated that D-lactate was not metabolized by tissues as rapidly or extensively as L-lactate; he suggested that it was removed from blood only by excretion through the kidney. In contrast, more recent studies indicate that D-lactate is metabolized by ruminant tissues (Giesecke and Stangassinger, 1980; Harmon et al., 1983), although rate of metabolism under acidotic conditions have not yet been measured. Presence of butyrate reduces conversion of L-lactate to glucose by calf hepatocytes (Reynolds et al., 1992), so interactions among absorbed acids may alter rate of metabolism.

Enhancing Acid Metabolism and Excretion (Reactions 11 and 12)

Kidney clearance of fluid can be increased by elevating dietary concentrations of salt or digestible protein. Although VFA should be resorbed by the kidney, D-lactate is partially excreted. Deficiencies of cofactors and adaptation to metabolize VFA and lactate may increase the maximum rate of metabolism of these acid end-products. Specific cofactors of interest include vitamin B₁₂, lipoic acid, and thiamin. The latter could explain why polioencephalomalacia occasionally is linked to acidosis. In contrast, Brent (1976) indicated that ruminal thiaminase developed after acidosis and suggested that bacterial thiamin destruction was enhanced by acidosis. Concentrations of VFA and lactate in plasma of animals dying from acidosis need to be measured to determine whether

Table 1. Subclinical acidosis checklist, scale of risk, and apparent impact under various conditions

Factor	Danger scale		Impact		
	Low	High	Lab	In vivo	Feedlot
Management					
Cattle disposition	Tame	Flighty	?	?	Moderate
Meal size	Small	Huge	Strong	Strong	Strong
Feed access	Limited	Unlimited	Strong	Strong	Limited
Diet composition					
Concentrate level	0%	100%	Strong	Strong	Strong
Grain	Corn, Milo	Wheat	Strong	Strong	Strong
Grain processing	Whole	Steam-flaked	Strong	Strong	Strong
Feed type	Unfermented	Fermented	Weak	?	?
DCAB ^a	Acid	Basic	Weak	?	?
Additives					
Ionophores	Present	Absent	Strong	Strong	Strong
Bicarbonate	Present	None	Weak	Weak	Weak
Fat	Up to 8%	None	Weak	Negative	None
Probiotics	Lactobacilli	None	Moderate	Some	?
Protozoal stimulants	Present	Absent	Strong	Strong	?
Protein level	High	Low	Weak	Weak	?
Thiamin	Supplemented	None	Weak	Weak	?
Virginiamycin	Present	Absent	Strong	Strong	Moderate
Malate/Fumarate	Present	Absent	Strong	Moderate	Weak

^aCation-anion balance.

rates of acid metabolism are limited. Naylor et al. (1984) indicated that hepatic metabolism of lactate may be limited. Only with absorption or liver malfunction would one expect butyrate or propionate to accumulate in peripheral blood. Relative concentrations of acetate and D and L lactate could reveal whether catabolic rates are being exceeded.

Overview

Factors that might be modified to reduce the incidence of subclinical and clinical acidosis are listed in Table 1 together with a relative danger scaling concerning the expected effect on the incidence of acidosis. Effectiveness of modifying certain factors has been studied only in ruminal fluid incubation studies, whereas other factors have been tested with cannulated animals or have been observed in feedlot; relative degrees of testing and effectiveness under these three scenarios are estimated. Inhibiting lactate-producing microbes and enhancing activity of lactate-using microbes and new procedures in feed management (limit or program feeding) may help reduce the incidence of acute and subacute acidosis.

Implications

Acute and chronic acidosis continues to plague feedlot ruminants. Steps involved with accumulation of ruminal acids and elevated osmolality of ruminal contents and blood are outlined and control procedures are proposed. Promising control measures include

specific feed additives that inhibit the lactate-producing bacterial strains, that stimulate activity of lactate-using bacteria or starch-engulfing ruminal protozoa, and that reduce meal size. Ruminal inoculation with microbial strains capable of preventing glucose or lactate accumulation or of metabolizing lactate at a low pH may help. Continued research concerning grain processing, dietary cation-anion balance, narrow-spectrum antibiotics, glucose- or lactate-utilizing microbes, salivary flow stimulants, and feeding management should yield new methods for reducing the incidence of acute and chronic acidosis.

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