DIGESTIVE DISTURBANCES: ACIDOSIS, LAMINITIS, AND BLOAT

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INTRODUCTION

In modern North American beef and dairy production systems, cattle typically are fed relatively high concentrate diets to achieve maximum production. Usually grains are processed to increase their digestibility in the rumen and in the total digestive tract; this increases feed conversion efficiency and helps reduce feed costs. However, the potential benefits of increased ruminal starch digestibility must be balanced against the increased risk of digestive disorders in cattle. While it is critical to meet the energy requirements of high producing ruminants, digestive disturbances must be avoided to ensure that meat and milk are produced from healthy animals in an efficient and cost-effective manner.

ACIDOSIS

Acute Acidosis

Acute ruminal acidosis is characterized by an extended period of time that pH in the rumen remains very low (usually less than 5.2; Figure 1). The depression in ruminal pH usually is due to an abrupt increase in the intake of rapidly fermentable carbohydrates; this results in an accumulation of volatile fatty acids (VFA) and lactic acid in the rumen. The excessive build-up of short chained fatty acids in ruminal fluid increases the osmolality of rumen contents which in turn inhibits feed intake, salivation, and the onset of rumination following meals (Carter and Grovum, 1990). A prolonged period of acute ruminal acidosis leads to systemic or metabolic acidosis (Owens et al., 1998). High osmotic pressure in the rumen pulls water from the blood into the gastrointestinal tract causing diarrhea. Loss of water from the blood increases blood osmolality and packed cell volume leading to dehydration of the animal. Furthermore, rate of absorption of VFA from the rumen is enhanced at low pH. When acid absorption exceeds metabolism, these compounds can accumulate in blood and increase blood osmolality further.

Clinical signs of acute acidosis include complete anorexia, abdominal pain, rapid beating of the heart, abnormally fast breathing, diarrhea, lethargy, staggering, recumbency and death (Krause and Oetzel, 2006). Cattle that survive the systemic effects of acute acidosis often become “poor doers” or “realizers” due in part to damage of the gastrointestinal tract. Prolonged periods of low ruminal pH reduce the absorptive capacity of the ruminal epithelium by causing abnormalities of ruminal papillae and ruminitis (McGavin and Morrill, 1976; McManus et al., 1977). The absorptive capability of the ruminal epithelium can be limited for up to six months after a bout of acidosis (Krehbiel et al., 1995). Lesions of the ruminal epithelium also have been implicated in systemic bacterial infiltration that can lead to liver abscesses (Nagaraja and Chengappa, 1998).

Fortunately, the prevalence of acute acidosis usually is low in commercial feedlots and dairies. For feedlot cattle, Smith (1998) reported that 3 to 7% of sick cattle (those placed in sick pens) were treated for digestive disorders and that about one-third of feedlot mortalities (which usually total < 2%) were due to digestive disorders. The prevalence of acute acidosis usually is even lower in commercial dairies. Gröhn and Bruss (1990) reported the incidence of acute ruminal acidosis was only 0.3% throughout lactation among 61,000 dairy cows; incidence was greatest during the three months after calving. The risk of acute acidosis is low in adapted animals; gradual changes in diet composition and quantity of diet delivered allow the rumen environment added time to cope with rapidly fermented diets. Experimentally, acute acidosis can be induced by withholding feed for a period of time (usually 12 to 24 h) followed by over-feeding additional concentrates (Krause et al., 2005) or by delaying feeding followed by overfeeding (Erickson et al., 2003). Hence, the risk of acute acidosis appears greatest during the period of transition from high forage to high grain diets and when feed delivery is inconsistent, conditions that promote the excess consumption of rapidly fermented diets.

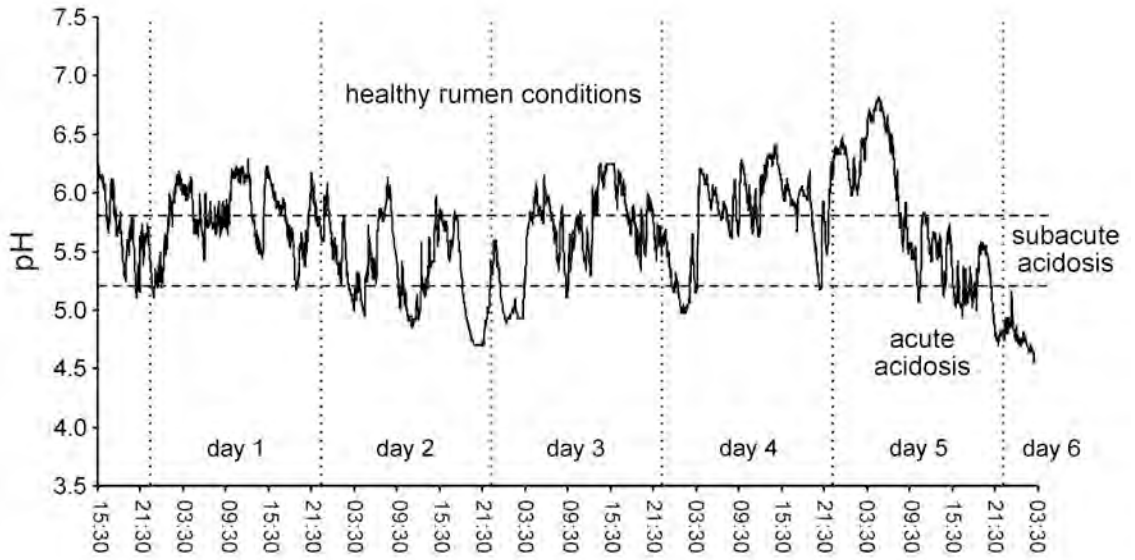

Figure 1. Ruminal pH profile in a dairy cow. Subacute acidosis was defined as pH < 5.8 and acute acidosis as pH < 5.2. The prolonged period of subacute acidosis that occurred on day 5 developed into acute acidosis on day 6 (Beauchemin, unpublished data).

Subacute Acidosis

Unlike acute acidosis, subacute ruminal acidosis (SARA) is prevalent in modern commercial feedlot and dairy production systems in North America. Its high prevalence has been correlated with the use of diets that contain substantial quantities of processed grains. It is difficult to identify animals suffering from subacute acidosis because clinical signs are not unique to acidosis. Cattle with subacute acidosis can experience diarrhea, weight loss, reduced milk production, and increased susceptibility to other metabolic disorders. Krause and Oetzel (2006) used rumenocentesis (i.e., percutaneous needle aspiration of fluid from the caudal ventral rumen) 6 to 10 h after feeding total mixed rations and 2 to 4 h after feeding component diets in 55 dairy herds to determine the prevalence of subacute acidosis in commercial dairies. They reported that during the first 140 days of lactation, 12 to 40% of the cows had a ruminal pH below 5.5. Prevalence of acidosis in the feedlot industry is not known, but considering that feedlot cattle diets contain even more grain, its prevalence is likely even higher than for the dairy industry.

An episode or bout of subacute ruminal acidosis occurs when pH of the rumen drops into a suboptimal zone for a period of time. Hence, the definition of subacute ruminal acidosis incorporates both a pH threshold and a duration. To characterize bouts of subacute ruminal acidosis, the pH threshold value typically used is 5.6 to 5.8 (Figure 1). In our laboratory, we use a ruminal pH < 5.8 to denote acidosis in dairy cows because the reduced fiber digestion below a pH of 5.8 has negative effects on milk production. The threshold pH of 5.6 typically is used to denote subacute acidosis of feedlot cattle because negative impacts of acidosis on feedlot cattle are related more to its effects on intake, nutrient metabolism, and animal health than on fiber digestion.

Automated systems have been developed recently to continuously monitor ruminal pH over an extended period of time (Dado and Allen, 1993; Penner et al., 2006); this makes it possible to characterize subacute ruminal acidosis in terms of bout duration. We use 4 h as the minimum duration (continuous time period when pH remains below the threshold value) to define a single bout of acidosis (Paton et al., 2006); shorter durations with a low pH are less detrimental to bacterial growth. The total duration of time that pH remains below the acidosis threshold value in a 24 h time period is an additional method for characterizing subacute ruminal acidosis (Penner et al., 2007).

RUMEN MICROBIAL DYNAMICS DURING ACIDOSIS

Ingestion of carbohydrates provides substrate for microbial growth in the rumen; this increases the total number of bacteria and VFA production. When

production rate of VFA exceeds the capacity of the system to neutralize or absorb these acids, ruminal pH declines (Allen, 1997). Ruminal pH of feedlot cattle and dairy cows varies considerably during a day; the drop in pH following meals is substantial when the diet contains a high proportion of rapidly fermented carbohydrates (Figure 1). With subacute acidosis, ruminal pH usually recovers to pre-feeding levels as the acids are absorbed from the rumen and as the buffering capacity of the rumen increases due to salivation. However, subacute ruminal acidosis can develop into acute ruminal acidosis in some cases as shown in Figure 1. For acute ruminal acidosis, immediate intervention is critical.

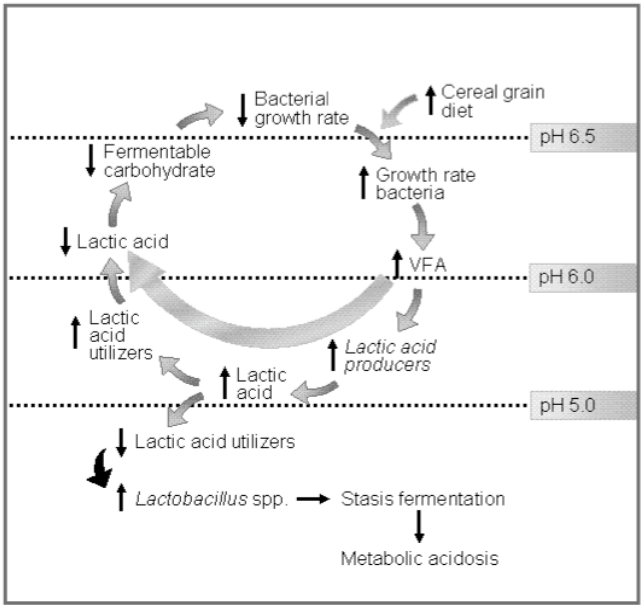

Figure 2. Sequence of events leading to subacute and acute acidosis (adapted from McAllister et al., 1996).

In the absence of acidosis, ruminal glucose concentrations remain low because glucose is transformed rapidly into VFA by rumen microorganisms. Lactic acid concentrations also are low because competition for substrate normally moderates the growth of lactic-acid producing bacteria, such as *Streptococcus bovis* and *Lactobacillus* spp. (Figure 2). Furthermore, growth of bacteria that use lactic acid, e.g., *Selenomonas* spp., *Megasphaera elsdenii*, and *Propionibacterium* spp., ensures that any lactic acid produced is rapidly metabolized; this prevents lactic acid from accumulating in the rumen. However, with high grain diets or a sudden change in diet composition or supply, the microbial populations become unstable and this allows rapid growth of lactic acid producers such as *S. bovis*. Because most lactate-utilizing bacteria are not acid tolerant, the balance between lactate-producing bacteria and lactate-utilizing bacteria is disrupted. During subacute acidosis transient spikes in lactic acid concentration in ruminal fluid appear, but eventually the balance between lactic acid production and utilization is achieved. In contrast, during acute acidosis, lactate often accumulates in ruminal fluid (> 40 m*M*), although acute acidosis can occur without lactic acid being present. Low ruminal pH also activates lactate dehydrogenase, the enzyme involved in converting pyruvate to lactate; this exacerbates the accumulation of lactic acid in the rumen. Furthermore, feeding a large amount of starch also can increase ruminal concentrations of free glucose; this increases the competitiveness of lactate-producing bacteria such as *S. bovis* in the rumen (Owens et al. 1998). Lactic acid is a very potent acid (10-times stronger than VFA), and this property contributes further to the decline in ruminal pH.

Excess carbohydrates in the rumen can lead to the production of toxins by some ruminal bacteria. For example, an excess of glucose causes *Prevotella ruminicola* to produce methylglyoxal, a substance that

is toxic to rumen bacteria (Russell 1998). As a result, rumen fluid of acidotic animals can appear stagnant. Furthermore, cellulolytic bacteria and protozoa are inhibited by a pH below 6.0; instead, acid tolerant bacterial species such as *S. bovis* and lactobacilli become dominant when pH is maintained below 6.0 for a prolonged time. If the pH drops further, *S. bovis* is inhibited; when pH drops below 4.7, only acid tolerant species such as lactobacilli are maintained. The many interconnected factors that cause ruminal pH to decline also make it very difficult to reverse a severe drop in ruminal pH that occurs during acute acidosis.

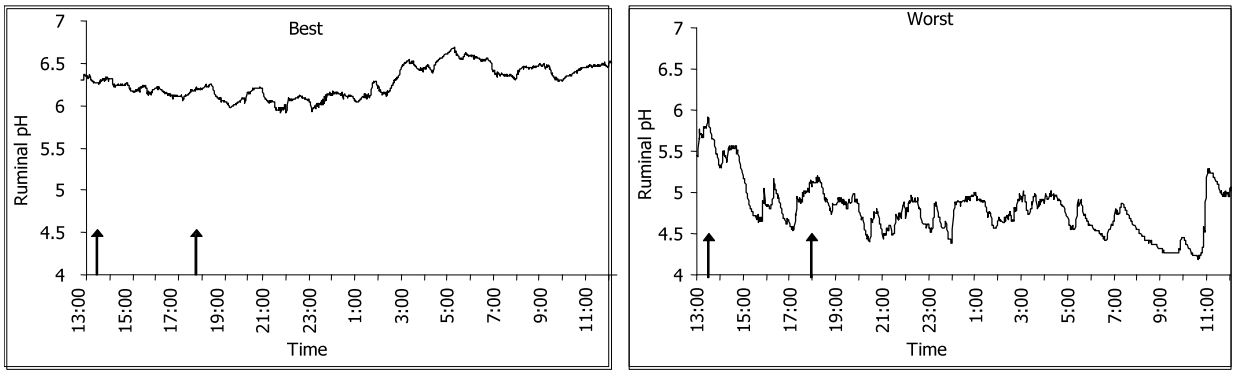

Figure 3. Ruminal pH measured 5 days after calving in two dairy cows (best and worst-case acidosis cows) fed the same lactation diet (Penner, Beauchemin and Mutsvangwa, unpublished data). Arrows indicating feeding of the total mixed ration.

VARIABILITY IN ACIDOSIS AMONG ANIMALS

The risk for acidosis is not equal for all animals. Individual dairy cows exhibit a tremendous amount of variation in the degree of acidosis. Figure 3 shows ruminal pH profiles on day 5 post-partum for two dairy cows fed the same diet. For the cow with the "best" profile, ruminal pH remained above 6, whereas for the cow with the "worst" profile, acute acidosis continued for the entire day.

Similar variability exists for beef cattle during the feedlot finishing phase (Figure 4). Factors accounting for this variation among animals are not well documented, but presumably they are related to differences in feed intake, eating rate, sorting of feed, salivation rate, rate of passage of feed from the rumen, and other aspects of physiology and behavior. The goals in beef and dairy production are to minimize the number of cattle that experience ruminal acidosis, and to reduce the duration and intensity of each episode of acidosis that an individual animal experiences.

IMPACT OF SUBACUTE ACIDOSIS

Fiber Digestibility

Subacute ruminal acidosis decreases the digestibility of fiber in the rumen; this decreases feed conversion efficiency and increases feed costs. Numerous in vitro studies using pure cultures of rumen microorganisms have shown that growth rate and ability of the major cellulolytic bacteria (*Ruminococcus albus, R. flavefaciens, and Fibrobacter succinogenes*) to degrade cellulose is negatively affected at pH below 6 (e.g., Russell and Wilson, 1996). Furthermore, ruminal cellulolytic protozoa and fungi also are sensitive to low pH. The effects of low pH on mixed cultures of rumen microorganisms have been studied using continuous culture in vitro systems. Decreasing ruminal pH to within the subacute acidosis range causes a 2 to 3 percentage unit decrease in NDF digestibility per 0.1 unit decrease in pH (Calsamiglia et al., 2002; Yang et al. 2002). In dairy cows, total tract acid detergent fiber digestibility decreased by 3.6 percentage units per 0.1 unit decrease in mean daily ruminal pH (Erdman 1998), while in dairy cows and feedlot cattle a decrease in mean ruminal pH from 6.4 to 5.7 lowered total tract NDF digestibility by 1.3 percentage units per 0.1 unit decrease in ruminal pH (Beauchemin, unpublished data).

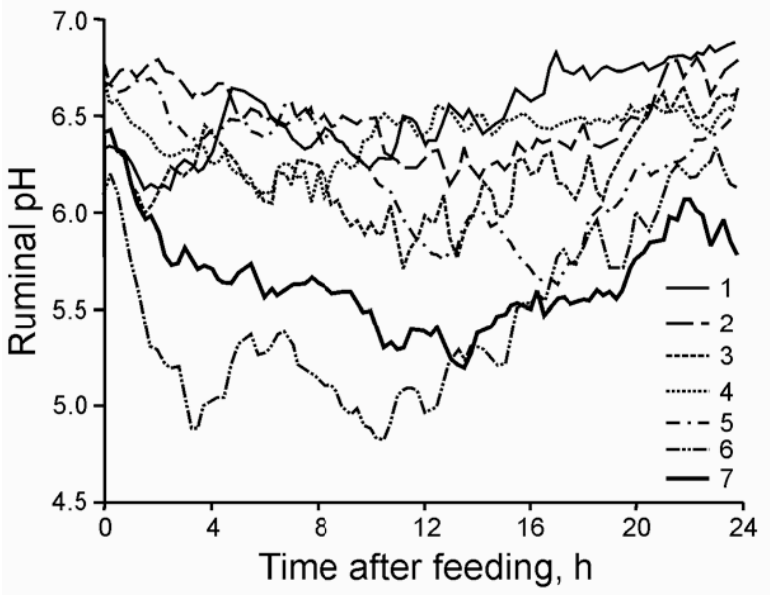

Figure 4. Changes in ruminal pH following ad libitum feeding of a high-grain finishing diet fed to feedlot steers (Schwartzkopf-Genswein et al. 2003). Each line represents an individual steer.

A constant low pH obviously has a negative effect on ruminal fiber digestion, but the effect of pH fluctuations on fiber digestion is less clear. Short (30 min), infrequent drops in pH failed to reduce NDF digestibility, unlike repeated 4-h periods of acidosis (Calsamiglia et al. 2002). Although subacute acidosis reduces fiber digestion in cattle, microbial populations apparently recover between bouts of acidosis when pH rises. In the case of dairy cows, the depression in fiber digestion associated with subacute acidosis is sufficient to reduce productivity. Oba and Allen (1999) proposed that a one percentage unit decrease in NDF digestibility was associated with a 0.25 kg/d decrease in 4% FCM yield and 0.17 kg/d decrease in dry matter intake. The significance of intermittent acidosis on fiber digestion by feedlot cattle in terms of production efficiency is less drastic because feedlot diets are typically low in fiber.

Feed Intake Variability

Subacute ruminal acidosis can lead to reduced feed intake and erratic eating patterns. For feedlot cattle, Brown et al. (2000) observed a high correlation coefficient ($r = 0.84$) between the lowest daily ruminal pH and feed intake on the subsequent day. When ruminal pH is low, the animal's feed intake drops; this limits further production of fermentation acids and restores pH to more optimum conditions. Once the pH is restored, the animal then resumes consuming feed that again may lead to excessive production of acids and this cycle can be repeated. Such an effect is illustrated in Figure 5, which shows the pH profile for an adapted feedlot steer fed a 92% concentrate diet containing barley. Variation in day-to-day intake is undesirable in terms of maximizing mean feed intake and providing a constant supply of nutrients for growth or lactation.

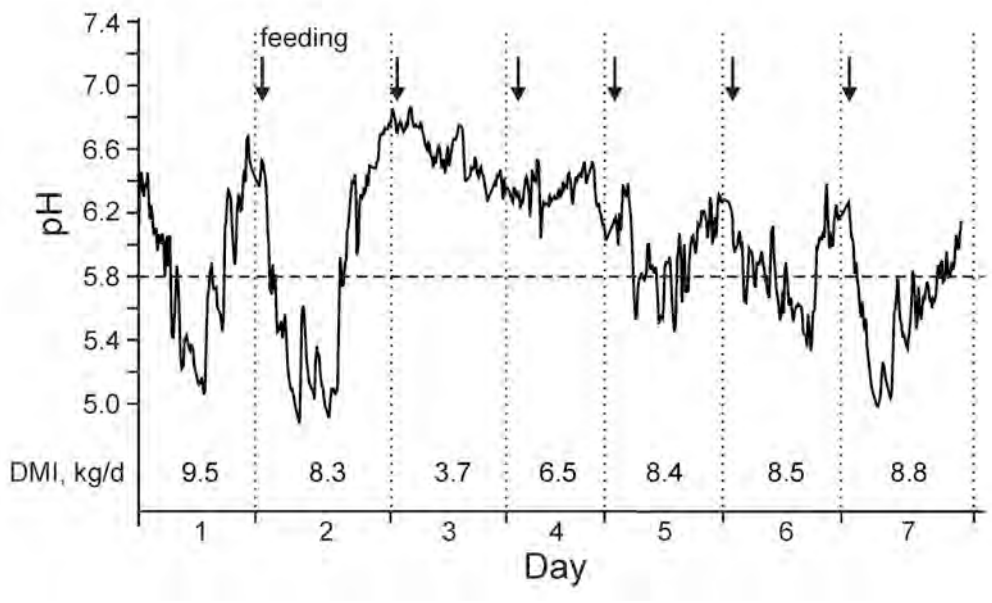

Figure 5. Ruminal pH and dry matter intake (DMI) for an adapted feedlot steer fed once daily (feeding indicated by arrows) measured for 7 days (Schwartzkopf-Genswein et al. 2003).

LAMENESS

Lameness is a major health and welfare concern for the North American cattle industry, particularly for the dairy industry (Hendry et al., 1997). Ruminal acidosis can cause lameness in cattle due to laminitis and associated hoof lesions (Cook et al., 2004; Nordlund et al., 2004). Laminitis is a generic term that refers to inflammation of the connective tissue located in the hoof. Severe cases of laminitis are characterized by deformed claws such as concave hoof walls, irregular hoof shape, and hoof overgrowth (Blowey, 1993). Laminitis can lead to white line hemorrhages, sole ulcers, and the formation of ridges on the hoof wall.

Several theories have been proposed to explain the link between acidosis and laminitis. According to one theory, a reduction in systemic pH during acidosis activates a vasoactive mechanism that increases total blood flow to the hoof (Nocek, 1997). Alternatively, histamine may be absorbed through rumen epithelium damaged during acidosis. Histamine is an inflammatory agent and vasoactive substance, and as such, may increase blood pressure and damage blood vessel walls causing inflammation and haemorrhaging within the hoof. It is well established that grain feeding increases the formation of histamine in the rumen (Garner et al. 2002).

Recent studies have produced compelling evidence for a different link between acidosis and laminitis based on the effects of bacterial toxins rather than vasoactive substances. Acute acidosis and repeated bouts of subacute acidosis damage the surface of the rumen wall and possibly the intestine; this allows bacteria and bacterial toxins to enter the portal circulation, causing liver abscesses and an inflammatory response (Gozho et al., 2005). The exterior surface of the hoof (the horn) is joined to the major bone in the hoof (the pedal bone) by highly vascularized connective tissue (corium) that acts as a shock absorber when the hoof contacts the ground (Figure 6). The corium is attached to the horn through folds of tissue, called laminae (within the wall) or papillae (within the sole; Hendry et al., 1997). The impact of acidosis on laminitis may be mediated by matrix metalloproteinases in a manner similar to the effects of parturition.

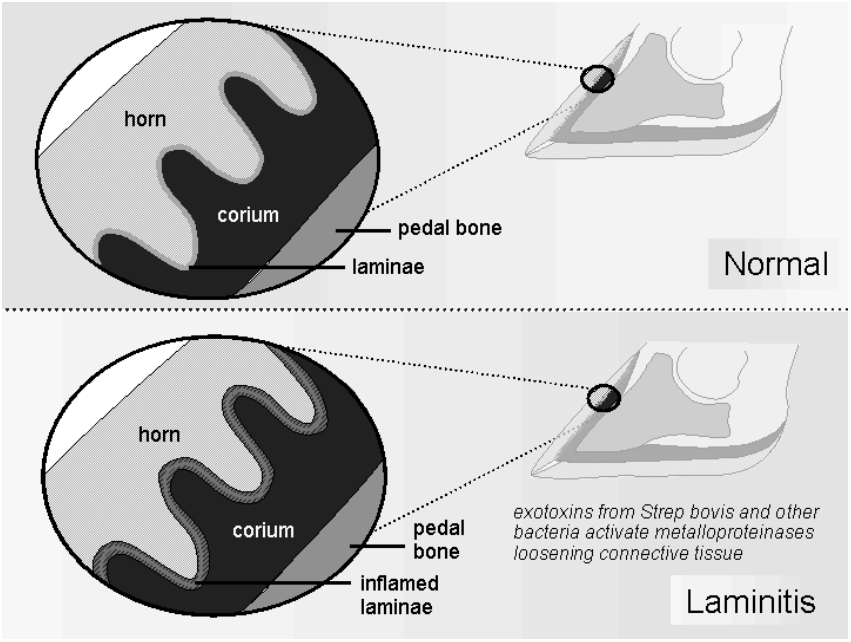

Figure 6. Schematic diagram of the hoof and the inflammation that occurs during laminitis.

At parturition, hormonal changes affect connective tissue metabolism within the hoof by elongating collagen fibers and loosening the connective tissues (Tarlton et al., 2002). In the case of laminitis, metalloproteinases are thought to be activated by exotoxins (proteases) released by bacteria. Once activated, these metalloproteinases degrade key components of the corium (Mungall et al., 2001). Studies in horses have shown that several gram-positive (*S. bovis*) and gram-negative bacteria produce exotoxins capable of activating the resident metalloproteinases within the lamellar structure of the hoof.

Dairy heifers subjected to an acute acidosis challenge developed signs of lameness within 24 h (Thoefner et al., 2005). Examination of the hoofs post-mortem detected weakening at the dermo-epidermal junction caused by stretching of the laminae and detachment of the basement membrane. However, acidosis in cattle does not always result in laminitis (Momcilovic et al., 2000; Donovan et al., 2004); thus, other factors appear to alter the susceptibility of cattle to acidosis induced laminitis. For example, an animal's environment may exacerbate or temper the effects of acidosis on laminitis (Cook et al., 2004).

GRAIN BLOAT

Grain-related bloat, or frothy-bloat, occurs primarily in feedlot cattle and often is associated with acidosis. However, prevalence of bloat in commercial feedyards usually is low due to the widespread use of ionophores combined with careful feeding management (Smith, 1998). The risk of feedlot bloat increases when rapid changes occur in diet composition or in feed delivery that increases the supply of rapidly fermented carbohydrate (Schwartzkopf-Genswein et al., 2004). A sudden increase in fermented carbohydrates in the rumen leads to rapid microbial growth rates and subsequent cell lysis. Extracellular bacterial mucopolysaccharides (slime) and stored carbohydrates released during microbial cell lysis increase the viscosity of ruminal fluid trapping gas and forming the stable foam that leads to bloat (Cheng et al., 1998).

Bloat results from the accumulation of gas in the rumen. Normally, gas produced during fermentation of feed rises through the rumen contents and forms a gas pocket in the dorsal sac. During frothy bloat, the gas, trapped within the liquid and particulate contents of the rumen, continues to accumulate. Continued accumulation of gas within the rumen increases the pressure within the rumen, eventually causing death by asphyxiation as the rumen exerts pressure on the diaphragm and lungs (Dougherty, 1956).

PREVENTING DIGESTIVE DISTURBANCES

Adaptation of the Rumen Environment

In general, the risk factors for digestive disorders include extensive grain processing, a low concentration of forage in the diet, and abrupt changes in diet supply or composition. The key to minimizing acidosis, bloat and laminitis is to provide ample time for the rumen environment to adapt to dietary changes (Schwartzkopf-Genswein et al., 2003). This adaptation phase allows the ruminal epithelium and the rumen microbial populations to adapt to changes in substrate supply.

Absorption of VFA from the rumen occurs passively through papillae (finger-like projections) located on the rumen wall. The rumen papillae gradually lengthen when cattle are exposed to a grain-based diet (Dirksen et al., 1985). This effect appears to be mediated by a stimulatory effect of VFA, especially butyrate (Sakata and Tamate, 1978), on papillae growth. This increased surface area and absorptive capacity of the rumen helps protect the cow from accumulating VFA in the rumen, the main driver of ruminal pH depression.

Gradual transitions of animals from a high forage to a concentrate diet helps to avoid the instability of microbial populations observed in cases of acute acidosis. Similarly, chemical buffers like bicarbonate may be added to the diet as a means of stabilizing ruminal pH and preventing subacute acidosis. Other dietary additives that have positive effects upon ruminal pH include ionophores, lactic acid fermenting bacteria, and yeast. Ionophores also help to prevent bloat because they reduce the variation in feed intake and inhibit the growth of gram-positive bacteria (Bergen and Bates, 1984), including *S. bovis* and *Lactobacillus*, two of the major lactic-acid and muco-polysaccharide-producing species found in the rumen.

Balancing Starch Availability with Physically Effective Fiber Content

Maintaining a balance between physically effective fiber content of the diet and starch availability is a key factor for stabilizing ruminal pH. Starch availability in the rumen depends on the source of grain and its processing. As the amount of starch digested in the rumen increases, ruminal pH decreases and the risk of ruminal acidosis increases. For example, cattle fed steam-rolled barley have a lower ruminal pH than cattle fed steam-rolled corn because ruminal starch digestion is greater for barley than corn (Yang et al., 1997). Similarly, ruminal pH is lower for cattle fed high moisture corn than for those fed dry cracked corn diets (Figure 7; Krause et al., 2002).

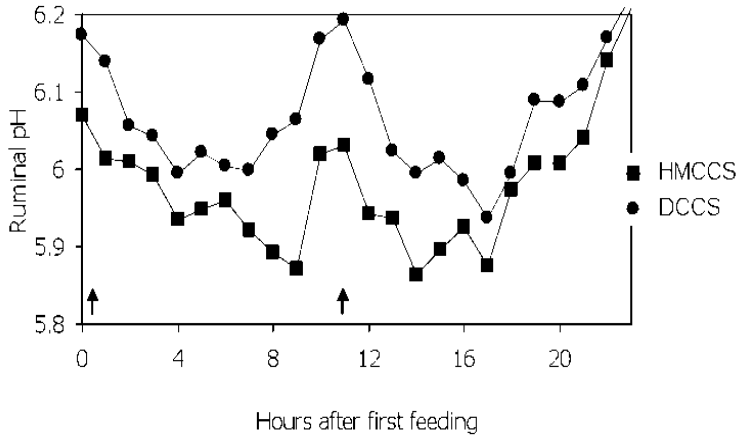

Figure 7. Ruminal pH of dairy cows fed high moisture corn (HMC) versus cracked shelled corn (DC). The forage was coarsely chopped (CS) corn silage (Krause et al., 2002).

Preventing ruminal acidosis requires a balance between the production of VFA and the neutralization/removal of VFA (which also reduces rumen osmolality). When the rate of ruminal fermentation of starch is high, diets need to be formulated to supply sufficient forage and forage needs to be of an adequate particle length. Longer forage particles promote chewing and saliva secretion that help

to buffer the acids resulting from feed digestion. In addition, long forage particles create a floating mat in the rumen that stimulates reticuloruminal contractions. Without such mixing motions, the rumen can become a stagnant pool and removal of VFA via absorption and fluid passage from the rumen declines. This condition increases the risk of acidosis and bloat. Similarly, anecdotal evidence indicates that acidosis occurs in feedlots when water supply is interrupted, a condition that would decrease ruminal dilution and outflow. Fiber is more slowly digested than starch and sugar, so including fiber in the diet slows the rate of carbohydrate digestion in the rumen. Decreasing the rate of carbohydrate digestion reduces the rate of VFA production, thereby preventing abrupt drops in ruminal pH. Feeding long particle fiber also can shift the site of starch digestion from the rumen to the intestine and this can reduce the potential for ruminal acidosis (Yang and Beauchemin, 2006). The multitude of mechanisms whereby optimizing physically effective fiber in the diet helps to modulate ruminal pH makes this approach a very effective method of acidosis prevention.

IMPLICATIONS

Processing grains to enhance ruminal digestion increases the risk of digestive disorders in cattle fed such grains. The key to attaining production responses from diets with high rates of ruminal starch digestion is through formulating diets and employing cattle management practices that avoid acidosis. Diet formulation typically is based on the average cow and often does not include a safety factor that considers the high variability among individual animals. Diets containing processed gains that are formulated to provide a minimum level of physically effective fiber will result in some degree of acidosis, and can have negative effects on production efficiency and animal health. The overall goal is to minimize the number of cattle that experience acidosis and, for individual animals that experience acidosis, to reduce the duration and intensity of the condition.

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QUESTIONS AND ANSWERS

Q: Karen, is there a learned behavior regarding feed aversion of cattle that experience acidosis similar to the feed aversions that Fred Provenza has studied for years?

A: I don't know, but Provenza's work is intriguing. He has clearly shown learning behavior for calves with their mothers. Perhaps there is some learned behavior associated with acidosis. If an animal is exposed to a bout of acidosis, that animal may become more reluctant in the future to eat as rapidly or fall into the acidosis "trap." We have a study on-going at the moment to look at this very issue. The graph I showed was from 8 dairy cows that were subjected to an acidosis challenge where we monitored how long it took for rumen pH to recover after a challenge. We repeated the acidosis challenge three consecutive times using the same cows and are in the process of looking at the data. A learned behavior could work to our favor. If animals can be trained to be less susceptible to acidosis, we could employ that as part of a management strategy. By using a very controlled exposure to acidosis, perhaps we could make animals more acidosis "fool-proof" in the future.

Update from Karen: Analysis of the data from that study is now complete. When cattle were subjected to repeated acidosis challenges (two weeks apart), the severity of each acidosis bout actually worsened even though the cows became increasingly reluctant to consume the grain offered each challenge. Thus, avoidance of grain intake did not minimize the severity of acidosis. These results lend support to the theory that animals will alter their feed consumption to correct ruminal imbalances, but our study also shows that this change in behavior does not necessarily reduce the incidence of acidosis. In fact, the study shows that once cows experience a bout of acidosis, cows are more prone to subsequent bouts of acidosis and each subsequent bout of acidosis is increasingly severe.