PREVENTING RUMINAL ACIDOSIS IN DAIRY COWS

Karen A. Beauchemin

Lethbridge Research Centre, Agriculture and Agri-Food Canada

INTRODUCTION

Feeding dairy cows for maximum milk production increases the risk of ruminal acidosis. Diets fed to high producing dairy cows in North America are typically highly digestible in the rumen and often lack sufficient physical fiber. Rapid fermentation of carbohydrates causes an accumulation of volatile fatty acids (VFA) in the rumen, leading to a drop in ruminal pH. Lack of sufficient physical fiber in the diet reduces rumination time and the concomitant flow of salivary buffers into the rumen. A high concentration of VFA, combined with a decrease in ruminal buffering, causes prolonged periods of low ruminal pH each day, also referred to as ruminal acidosis. While feeding highly fermentable diets can lead to short-term increases in milk production, the long-term consequences of ruminal acidosis on lameness, susceptibility to metabolic diseases, and cow longevity can be devastating. While it is critical to meet the energy requirements of high producing cows, acidosis must be avoided to ensure high milk production as well as cow health. This paper discusses the factors that predispose dairy cows to acidosis and provides insight into some of the feeding and management opportunities that can be used to help reduce its occurrence.

REGULATION OF RUMINAL PH

Diets that are high in non-fiber carbohydrates (mainly starch and sugars) are rapidly fermented in the rumen, leading to rapid production of VFA. If the rate of VFA production exceeds the rate at which the rumen environment can neutralize or absorb the acids, the VFA load accumulates resulting in subacute ruminal acidosis (Fig. 1).

Prevention of ruminal acidosis therefore depends on balancing the rate of VFA production in the rumen with the rate of VFA neutralization and/or removal from the rumen. Total VFA production is determined by dry matter intake and the ruminal digestibility of the feed. Thus, high producing dairy cows fed for maximum feed intake are highly susceptible to ruminal acidosis because of the large quantity of feed fermented in the rumen.

Absorption of VFA is the main mechanism by which VFA are removed from the rumen. In the rumen, VFA occur both in the protonated form (undissociated, H-VFA) and the ionized form (dissociated, VFA–). At high pH (e.g., 7) most of the VFA are in the ionized state, whereas the opposite is true at low pH (< 6). The form of the VFA and the way they pass through the rumen epithelium contribute directly to the ruminal pH. Transfer of H-VFA into the epithelium contributes to the stabilization of intra-ruminal pH because the VFA (acid) are removed from the rumen. The H-VFA are absorbed through the rumen epithelium, with
lower pH increasing the rate of absorption. Increased VFA absorption at low pH is thought to be due to the difference in bicarbonate gradient between the rumen fluid and the epithelium. Volatile fatty acids can also be absorbed in the ionized form (Gäbel et al. 2002). In this case, VFA are absorbed in exchange for bicarbonate which can be from blood and from de novo synthesis within the epithelial cells. For every 1 mole of VFA– absorbed, 0.5 moles of bicarbonate are released. Thus, the ruminal wall is a potent source of bicarbonate. The role of epithelial bicarbonate secretion in maintaining rumen pH is very important when cattle are fed high concentrate diets because saliva production is decreased, yet VFA production is increased. Thus, absorption of VFA from the rumen (removing VFA, adding bicarbonate) is the major factor moderating ruminal pH.

Hydrogen ions are also removed from the rumen by the buffering effects of saliva, feed, and some feed degradation products (e.g., ammonia). Of these, saliva is by far the most significant buffer. Saliva contains bicarbonate and phosphate buffers that play a major role in buffering acids within the rumen.

**DEFINING ACIDOSIS**

In high producing dairy cows, ruminal pH fluctuates over the course of the day as the process of eating, rumination, ruminal digestion and VFA absorption occurs. Systems that continuously monitor ruminal pH allow researchers to characterize the changes in pH throughout the day as a function of diet and management (e.g., Penner et al. 2006). Example pH profiles measured in dairy cows fed high and low forage diets are shown in Fig. 2.

A pH threshold value is typically used to characterize bouts of ruminal acidosis. Each time the pH drops below the threshold, a bout of acidosis occurs. In our laboratory, we use a

**Figure 1.** Ruminal acidosis occurs as a result of a more rapid rate of VFA production compared with the rate of neutralization and/or removal of VFA in the rumen.
threshold of pH < 5.8 to denote total sub-acute acidosis in dairy cows because fiber digestion is reduced below this threshold (Russell and Wilson 1996). The extent of the drop in pH is also important because pH < 5.5 is detrimental to the ruminal epithelium. Continuous exposure of ruminal epithelium to pH < 5.5 rapidly results in functional and morphological alterations (Gäbel et al. 2002). Thus, prolonged periods of pH < 5.5 cause damage to the absorptive capacity of the rumen, which exacerbates the effects of acidosis. For that reason, we characterize bouts of acidosis as mild (5.8 > ruminal pH > 5.5), moderate (5.5 > ruminal pH > 5.2), and acute (ruminal pH < 5.2). For example, in Fig. 2, the cow fed the low forage diet experienced a major bout of acidosis lasting 9-10 h each day starting at about 1700 h. However, the acidosis was mild, and likely had negative effects on fiber digestion, but less significant effects on pathology of the rumen epithelium.

IMPACT OF RUMINAL ACIDOSIS

Ruminal acidosis is a major problem for the North American dairy industry (Krause and Oetzel 2006) costing between $500 million to $1 billion a year (Donovan 1997). Financial losses occur due to treatment of sick animals, reduced productivity, and lower feed efficiency. Subacute ruminal acidosis can be difficult to diagnose because the clinical signs are not unique to acidosis. Cows with ruminal acidosis can experience diarrhea, weight loss, reduced milk production, lameness and in some cases, increased susceptibility to other metabolic disorders.

Figure 2. Ruminal pH measured in two dairy cows fed either a TMR containing 60% or 45% forage (DM basis) over a 48-h period. The TMR was offered at 0600, 1500 and 1800 h each day. The dashed line indicates the acidosis threshold of pH < 5.8.
Poor Health and Increased Lameness

It is well documented that repeated bouts of ruminal acidosis damage the surface of the rumen wall (Krause and Oetzel 2006), although it is not known whether the intestinal mucosa is also affected. Damage to the absorptive tissues within the gastrointestinal tract increase the potential for bacteria, amines and the toxins produced by bacteria that lyse (lipopolysaccharides) to enter the portal circulation, causing liver abscesses and an inflammatory response (Gohzo et al. 2005). It is not clear whether the toxins are absorbed through the rumen epithelium or the intestinal mucosa.

There is increasing evidence that these toxins are implicated in laminitis. In the hoof, the horn (or exterior surface) is joined to the major bone by highly vascularized connective tissue (corium). This connective tissue acts as a shock absorber when the hoof comes into contact with the ground. During laminitis, the mechanical strength of the connective tissue within the hoof is disrupted, which allows the bone to rotate laterally or sink into the corium of the hoof. The corium can then shift laterally, expanding the white line, or upwards, causing swelling around the coronary band (Blowey 1993). Solar compression can lead to sole ulceration. The impact of acidosis on laminitis is thought to be mediated by proteinases within the connective tissue that are activated by bacterial toxins. Once activated, these proteinases degrade the connective tissue within the hoof (Mungall et al. 2001).

While there are no controlled studies that have examined the relationship between rumen pH and lameness in dairy cows, the study by Donovan et al (2004) provides some evidence for an association. While a direct relationship between ruminal acidosis and lameness was not found, cows that received diets with the greatest difference in fermentability between prepartum and postpartum periods (e.g., a low energy diet fed prepartum followed by a high energy diet postpartum) exhibited more hemorrhages and ulcers in the sole. This study supports the concept that large differences in fiber and net energy content between the close-up and early lactation diets challenges the adaptation of the rumen microbes and the rumen epithelium and can increase incidence of ruminal acidosis and lameness in dairy cows.

Poor Feed Conversion Efficiency

Ruminal acidosis decreases the digestibility of fiber in the rumen which decreases feed conversion efficiency and increases feed costs. Growth of the major cellulolytic bacteria is negatively affected at pH < 6 (e.g., Russell and Wilson 1996). In continuous culture in vitro systems, decreasing ruminal pH to within the subacute acidosis range caused 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, ADF digestibility in the total tract 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). The depression in fiber digestion caused by acidosis is sufficient to reduce productivity.

**Low Feed Intake**

Ruminal acidosis can cause erratic fluctuations in feed intake from day-to-day. The high osmolality of rumen contents during acidosis causes cows to go “off-feed”. Reduced feed intake reduces the production of VFA causing the pH to rise. The cow then resumes a high feed intake that causes excessive production of acids, and the cycle is repeated. This pattern of fluctuating intake is undesirable in terms of supplying the cow with energy precursors for milk production.

**Reduced Microbial Protein Synthesis**

Ruminal acidosis lowers the efficiency of microbial protein production in the rumen (i.e., the amount of microbial protein produced per unit of carbohydrate digested). A decrease in microbial efficiency will decrease the yield of microbial protein (g/d) unless additional fermentable carbohydrate is supplied; this additional intake of fermentable carbohydrate further increases the risk of acidosis. Decreased microbial protein synthesis increases the need for supplemental feed protein in the diet, which in most cases increases feed costs.

**FACTORS THAT CONTRIBUTE TO RUMINAL ACIDOSIS**

**Variability Amongst Cows**

The risk of acidosis is not equal for all cows, even within a herd. Ruminal pH is especially variable amongst fresh cows. Fig. 3 shows the ruminal pH profile of two fresh primiparous cows fed the same lactation diet. The “best” profile shows ruminal pH remained high throughout the day, whereas the “worst” profile shows the cow experienced severe acidosis throughout the entire day. Factors accounting for the variation among cows are many, including dry matter intake, eating rate, sorting of feed, salivation rate, absorptive capacity of the rumen, rate of passage of feed from the rumen, and other aspects of cow physiology and behavior.

**Stage of Lactation**

We studied the occurrence of ruminal acidosis pre- and post-calving in primiparous dairy cows (Penner et al. 2007). Mean ruminal pH dropped from an average of 6.32 before calving, to an average of 5.98 after calving. During early lactation, cows experienced acidosis for about 6 to 9 h/d, with the severest incidence of acidosis occurring 5 wk after calving.
(Fig. 4), indicating that fresh cows are extremely susceptible to acidosis. This susceptibility increases the need for proper transition diets that help adapt both the microbial population and the absorptive surface of the rumen epithelium.

**Previous Exposure to Acidosis**

In a recent unpublished study, we examined the impact of repeated exposure of cows to bouts of acidosis. Acidosis was induced by restricting feed to 50% of ad libitum intake for 24 h, followed by a 4 kg meal of 50% ground barley and 50% ground wheat before allocating TMR. Cows were exposed to 3 acidosis challenges, each separated by 14 d. The entire grain allotment was consumed by all cows during the 1st acidosis challenge, by 75% of the cows during the 2nd challenge, and only 38% of the cows during the 3rd challenge. Although fewer cows consumed the entire grain allotment during each subsequent challenge, the severity of acidosis increased by 3 to 6-fold for cows fed a low forage diet and by 2 to 4-fold for cows fed a high forage diet. Thus, the cows became more prone to acidosis over time even though they altered their feed intake to avoid acidosis. The severity of each subsequent bout of acidosis increased, especially when cows were fed diets low in physically effective fiber. It can be inferred from this study that once a cow experiences a bout of acidosis, she becomes more susceptible to acidosis, which can have long-term consequences on cow health and productivity.

**DIET AND FEEDING MANAGEMENT TO REDUCE ACIDOSIS**

**Adaptation of the Rumen and its Microbial Populations**

Dairy cows can tolerate a highly fermentable diet if given the opportunity to adapt by gradually increasing the amount of concentrate fed over time. Adapting ruminants to energy-rich diets causes an increase of up to 4-fold in the absorptive capacity of the rumen (Gäbel et al. 2002) mainly due to morphological and functional changes to the rumen epithelium. Slowly increasing the concentrate intake leads to increases in the size of the papillae and the number of epithelial cells (Dirksen et al. 1984). It also increases the absorptive capacity of the individual epithelial cells (Gäbel et al. 2002).

Consequently, it is usually recommended to step-up concentrate feeding during the transition period. Lactation diets contain high levels of fermentable carbohydrate and low levels of fiber to maximize energy intake, thus “priming” the rumen during the transition phase is thought to help both the rumen microflora and the rumen epithelium adapt to the change in diet. Without adaptation, the large change in diet composition that occurs at parturition exacerbates the risk of ruminal acidosis. A caveat of supplying more grain before calving is a greater decrease in dry matter intake immediately before parturition and generally higher non-esterified fatty acid levels in the blood post-partum.
Figure 3. Ruminal pH measured 5 days after calving in two cows (best and worst-case acidosis cows) fed the same lactation diet (original data from Penner et al. 2007). The dashed line indicates the sub-acute acidosis threshold of pH < 5.8.

Figure 4. The effect of day relative to parturition on total sub-acute ruminal acidosis (total time pH < 5.8), moderate to severe acidosis (time pH < 5.5), and dry mater intake in primiparous dairy cows (Penner et al. 2007).
Physically Effective NDF

Physically effective fiber (peNDF) is a term that combines the NDF content of the diet with a measure of particle size (Mertens 1997). Physically effective NDF causes the cow to spend time eating and ruminating, which promotes the flow of salivary buffers into the rumen. Dairy cows on average spend 2 to 6 h/d eating, 3 to 9 h/d ruminating, and a maximum of about 14 h/d chewing (eating + ruminating) depending upon the diet (Fig. 5). Increasing the intake of peNDF either by: 1) increasing the NDF content (i.e., including more forage), or 2) increasing the chop length of forages, increases chewing, with the greatest increase in chewing for low fiber diets. However, the role of peNDF in preventing acidosis is often over-estimated.

Increasing peNDF intake increases chewing time, which increases salivary secretion, but the increase in saliva output is not as great as is often assumed. This is because the increased flow of saliva during chewing is accompanied by a decrease in resting saliva secretion. The net increase in total salivary secretion due to 1 h/d more chewing is about 7 L (Maekawa et al. 2002). The buffering capacity supplied by the additional saliva would adequately buffer the digestion of about 0.75 kg of TMR. Thus, the net effect of this incremental saliva production on ruminal pH is relatively small. It has been estimated that salivary buffers neutralize from 15% (Gäbel et al. 2002) to 40% (Allen 1997) of the VFA produced in the rumen.

The major benefit of including peNDF in the diet is that it slows the rate of VFA production in the rumen, because long fiber particles are more slowly digested than starch and sugar. So, adding forage to the diet not only increases chewing time and saliva secretion, it also slows down the rate of VFA production. The net effect is that the VFA production is evened-out throughout the entire day, helping to avoid accumulation in the rumen. Feeding long particle fiber also shifts the site of starch digestion from the rumen to the intestine, which reduces the potential for ruminal acidosis (Yang and Beauchemin 2006). An additional benefit of long forage fiber is that it creates a floating mat in the rumen, which stimulates contractions of the rumen. Without these mixing motions the rumen can become a stagnant pool, and removal of VFA via absorption and fluid passage from the rumen would decline, thereby increasing the risk of acidosis.

Intake of Non-Fiber Carbohydrates

Intake of non-fiber carbohydrates such as sugars and grain can predispose cows to ruminal acidosis, especially when the grains are highly processed. Generally, the intent of grain processing is to optimize starch availability in the rumen while avoiding digestive disturbances. Firkins et al. (2001) summarized the published literature on the effects of processing corn for dairy cows. Ruminal digestibility was highest for high-moisture corn, followed by steam-flaking, dry grinding, and then coarse cracking or dry-rolling. While low ruminal digestion of starch is partially compensated for by post-ruminal digestion, the
compensatory digestion is not always sufficient to avoid a reduction in total tract digestion. Thus, maximizing ruminal digestion of starch maximizes total tract digestion of starch (Fig. 6, left-hand side).

As the amount of starch digested in the rumen increases, ruminal pH decreases and the risk of ruminal acidosis increases. Low ruminal pH due to increased ruminal digestion of starch decreases ruminal NDF digestibility (Fig. 6, right-hand side). The decrease in ruminal NDF digestibility negates some of the improvement in starch digestibility, but on average, the net benefit to digestion of organic matter in the total tract is positive. However, that is not always the case. The effect of increased ruminal starch digestion on total tract digestion of organic matter depends on the magnitude of the ruminal pH depression and the extent to which NDF digestion is depressed. Therefore, when formulating diets using sources of rapidly fermentable starch, it is essential to supply adequate peNDF to minimize the incidence of ruminal acidosis.

**Ration Sorting**

Dairy cows preferentially sort for the grain component of a TMR and discriminate against the longer forage components. As a consequence of this sorting behavior, cows consume diets that are higher in fermentable carbohydrate and lower in peNDF than formulated. Sorting behavior puts cows at increased risk of ruminal acidosis. Because sorting
behavior is variable among cows, some cows will be at greater risk for ruminal acidosis due to sorting than others. In general, sorting against forage is highest when diets contain long forage particles. To minimize sorting, particle uniformity of the TMR helps. Using the Penn State Particle Separator, less than 15% of the material should be retained on the top sieve. Furthermore, frequent feeding or pushing-up of the feed helps reduce sorting.

In summary, preventing ruminal acidosis requires a balance between the production of VFA and the neutralization/removal of VFA. If the rumen availability of starch is high, then diets need to be formulated for higher levels of peNDF and lower concentrations of non-fiber carbohydrates. Increased peNDF can be achieved by increasing the particle size of forage or by formulating the diet for higher NDF content. In addition to providing adequate peNDF, good feed bunk management is critical. In particular, abrupt changes in diet composition should be avoided, and consistent timing and quantity of feed delivery should be implemented.

CONCLUSION

Feeding dairy cows for maximum milk production increases the risk of acidosis, which can reduce efficiency of milk production and jeopardize cow health. Ruminal acidosis occurs when the rate of VFA production in the rumen exceeds the rate at which the rumen environment can neutralize or absorb the acids. Carefully adapting the rumen to changes in diet, supplying adequate peNDF concentration, and reducing the fermentability of the non-fiber carbohydrate fraction are the main factors to consider in preventing ruminal acidosis. Use of high quality forages helps cushion against the risk of ruminal acidosis, because a greater proportion of forage can be included in the diet without lowering its digestible energy content. Most diets are formulated for the average cow, and do not include a margin of error.
to account for the variability among cows. As diets are formulated closer to the minimum level of peNDF and with higher concentrations of non-fiber carbohydrates, a greater portion of the cows will experience ruminal acidosis. Formulating diets for the average cow may be acceptable for cows in mid and late lactation, but diets for cows in early lactation should be formulated above minimum peNDF requirements because of their higher risk for acidosis.

REFERENCES


