

Minimizing the Risk for Rumen Acidosis

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Abstract

Ruminal acidosis is a common disease in high yielding dairy cows, during which the rumen pH is depressed for several hours per day. There is no general agreement of the definition of the disease, and its diagnosis based on rumen pH measurement is invasive and inaccurate. As a result, the disease often goes unnoticed, and its impact is not well recognized. Surveys suggest a prevalence of between 19 and 26% in early and mid-lactation dairy cows in North America. Many symptoms have been attributed to subacute rumen acidosis (**SARA**), including feed intake depression, milk fat depression, reduced fiber digestion, inflammation of the rumen, systemic inflammation, dehydration, diarrhea, laminitis, liver abscesses, and increases in the concentrations of bacterial toxins in digesta throughout the digestive tract. These symptoms are not always obvious. The mechanisms of how, and even if, the rumen pH depression causes these symptoms are not always well understood. Cows may vary in their susceptibility to ruminal acidosis. Preventing ruminal acidosis requires formulating diets that contain enough coarse (physically effective) fiber and do not contain excessive amounts of rapidly-fermenting non-fiber carbohydrates. In addition, these diets must be ingested by the cows in the way they were formulated, which involves preventing mixing errors, excessive mixing, and sorting by cows. The inclusion of buffers, e.g. sodium bicarbonate,

and yeast in the diet can prevent rumen acidosis, as does adaptation to high grain diets during the last part of the dry period. Feeding strategies, such as frequent feeding, continuous availability of feed, and sufficient bunk space, spread out meals throughout the day, and reduces the risk of ruminal acidosis.

Introduction

Milk production yields of dairy cows in North America have increased substantially during the last few decades. This has greatly increased the energy and nutrient requirements of these cows (NRC, 2001). As a result, more nutrient-dense diets need to be fed, which had been mainly achieved by feeding more grain and less forages. Feeding these diets can lead to a build up of organic acids in the rumen and a reduction in rumen buffering (Kleen et al., 2003; Stone, 2004; Plaizier et al., 2008). These changes will reduce the rumen pH. When the rumen pH is depressed for prolonged periods each day, ruminal acidosis occurs (Kleen et al., 2003; Stone, 2004; Plaizier et al., 2008). This nutritional disorder has major effects on the health and production of dairy cows that are not always realized. This manuscript will review the definitions, diagnosis, prevalence, causes, symptoms, and effects of ruminal acidosis, as well as give recommendations for its prevention.

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Definition of Rumen Acidosis

Rumen acidosis is a metabolic disorder in ruminants during which the rumen pH is moderately depressed for several hours per day. There is no general agreement about the magnitude and the duration of this depression, and on how the pH depression differs between acute and SARA. Definitions of rumen acidosis are based on the pH of rumen fluid (Kleen et al., 2003; Duffield et al., 2004; Plaizier et al., 2008). Rumen fluid can be sampled with a stomach tube, by rumenocentesis, or through cannula of rumen-fistulated cows (Garrett et al., 1999; Duffield et al., 2004; Plaizier et al., 2008).

Rumen acidosis can be acute or subacute. The difference between these forms of the disorder are that during acute ruminal acidosis, the pH depression is more severe, the concentration of lactic acid in the rumen digesta is higher, and the clinical signs more prominent (Underwood, 1992; Kleen et al., 2003; Nagaraja and Titgemeyer, 2007). A rumen pH depression below pH 5.2 and concentrations of lactic acid in rumen fluid in excess of 5 mM are regarded as signs of acute ruminal acidosis (Krause and Oetzel, 2006; Nagaraja and Titgemeyer, 2007; Plaizier et al., 2008). Acute rumen acidosis is common in feedlots, whereas SARA is common on dairy farms (Krause and Oetzel, 2006; Nagaraja and Titgemeyer, 2007; Plaizier et al., 2008). This manuscript will, therefore, focus on SARA, rather than on the acute form of rumen acidosis.

Different rumen pH thresholds for SARA are used. These thresholds differ among the techniques used for the rumen fluid collection and the location in the rumen from where the fluid is sampled, as these have large effects on the pH of the sample (Garrett et al., 1999; Duffield et al., 2004; Krause and Oetzel, 2006). For field studies using rumenocentesis,

Garrett et al. (1999) proposed a pH threshold of 5.5. Duffield et al. (2004) recommended pH thresholds of 5.5, 5.8, and 5.9 for rumen fluid samples collected by rumenocentesis, through a rumen cannula from the ventral sac, and using a stomach tube, respectively. Duffield et al. (2004) recommended pH thresholds of 5.5, 5.8, and 5.9 for the diagnosis of SARA when rumen fluid samples collected by rumenocentesis, through a rumen cannula from the ventral sac, and using a stomach tube, respectively. As the duration that the rumen pH is below a critical value is important for the physiological impact of the rumen pH depression, our group uses a threshold on 180 min/day below pH 5.6 for the diagnosis of SARA in cows with indwelling pH probes in the rumen (Plaizier et al., 2008). This threshold was chosen, as only rumen pH depressions that exceeded this value were associated with increases in the concentrations of bacterial endotoxins in rumen fluid and markers of inflammation in blood (Gozho et al., 2005).

Diagnosis of Rumen Acidosis

As rumen acidosis is defined as a depression of the rumen pH, the measurement of this pH is the most obvious technique for the diagnosis of this metabolic disorder. This technique requires the collection of rumen fluid. Rumenocentesis, a stomach (oral) tube, and a rumen cannula have all been used for this collection (Garrett et al., 1999; Duffield et al., 2004; Plaizier et al., 2008). The rumenocentesis technique consists of a rumen puncture during which rumen fluid is collected close to the wall of the ventral sac (Garrett et al., 1999; Kleen et al., 2003; Duffield et al., 2004). Problems with these techniques are that they are invasive, can cause health problems, and that the pH of rumen fluid varies among sites in the rumen and within a site of the rumen during a 24 hr period (Enemark et al., 2004; Krause and Oetzel, 2006; Plaizier et al., 2008). The invasiveness of rumenocentesis

is probably responsible for the limited use of this technique for the diagnosis of rumen acidosis on dairy farms. Due to the differences in the site of the rumen fluid collection among the techniques, the pH values of the collected samples differ among these techniques. Due to the diurnal variation in the rumen pH, the timing of the rumen fluid collection is critical for diagnostic purposes. The highest accuracy of the diagnosis is expected when the rumen pH is at its lowest, i.e., at its nadir. Our group, therefore, collects rumen fluid samples for the measurement of rumen pH at 6 hr after feed delivery (Plaizier et al., 2008). However, others have reported that the rumen pH nadir did not occur at this time, but occurred at times varying from 2 to 12 hr after feed delivery (Keunen et al., 2002; Duffield et al., 2004; Krause and Oetzel, 2006).

In a research setting where rumen-cannulated cows are available, in-dwelling rumen pH probes have been used to monitor rumen pH (Duffield et al., 2004; Penner et al., 2006; Plaizier et al., 2008). These probes are placed through the cannula and secured at one site in the rumen, generally in the ventral sac, and allow continuous measurement of rumen pH. For rumen fluid samples that are collected through cannula, or when in-dwelling rumen pH probes are used, rumen pH thresholds of 5.5, 5.6, 5.8, and 6.0 have been proposed for the diagnosis of SARA (Krause and Oetzel, 2006; Penner et al., 2006; Plaizier et al., 2008). In-dwelling rumen pH probes that can be placed in the reticulo-rumen through the esophagus and that can be used on non-cannulated cows also have become commercially available (Gasteiner et al., 2009; Mottram, 2010). Issues with the use of these probes have included short battery life, drift in pH measurement, uncertainties about their location in the reticulo-rumen, and their effects on cow health (Gasteiner et al., 2009; Mottram, 2010). It appears that these issues are being overcome. Several users of these probes have

reported that, in most cows, their location is in the reticulum. The pH of digesta in the reticulum is higher than that in the rumen and this has to be taken into account in the interpretation of pH data from these probes.

Mainly due to the invasiveness of the rumen fluid collection and problems with obtaining representative rumen fluid samples, alternative measures have been proposed for the diagnosis of rumen acidosis. These include milk fat, blood pH, partial pressures of CO₂ and O₂ in blood; fecal pH; urine pH; blood concentrations of glucose, lactate, and acute phase proteins; packed cell volume of blood; renal net acid base excretion; and rumen contraction frequency (Enemark et al., 2004; Ganesela et al. 2010; Li et al., 2012b). Most of these measures can be affected by the rumen pH, and could therefore, be indirect indicators of rumen pH, but most of these measures also are affected by many other factors also, which makes their use in the diagnosis of rumen acidosis inaccurate. This has been proven in several on-farm studies and studies in which SARA was experimentally induced, which showed that none of these measures were consistently related to rumen pH (Enemark et al., 2004; Plaizier et al., 2008; Li et al., 2012b).

Experimentally-induced SARA by feeding excessively high grain diets has been associated with an increase in the content of bacterial lipopolysaccharide endotoxin (LPS) of feces (Plaizier et al., 2012). This suggests that the fecal LPS content may be a diagnostic tool for SARA. To test this, the relationship between the fecal LPS content of individual cows and risk factors for SARA were determined on commercial dairy farms (Li et al., 2010). This study showed large variations in fecal LPS content among farms and among cows within farm, but relationships among fecal LPS and these risk factors were not obvious. Hence, at this

time, the measurement of LPS in feces cannot yet be recommended as an alternative for the measurement of rumen pH for the diagnosis of SARA.

Prevalence of Rumen Acidosis

The prevalence of a disease is the proportion of a population found to have this condition. More specifically, the point prevalence is the proportion of a population that has the condition at a specific point in time. The prevalence of a disease differs from that of the incidence of that disease, as the latter is the risk of developing some new condition within a specified period of time. Several surveys have been conducted to determine the prevalence of SARA. These surveys have been conducted using rumenocentesis and a threshold pH of 5.5 for the diagnosis of SARA.

Two surveys were conducted at dairy farms in Wisconsin. The first survey found prevalences of 19 and 26% in early lactation and mid-lactation cows, respectively (Garrett et al., 1999). The second survey found that the prevalence of SARA in early and peak lactation cows was 20.1% (Oetzel et al., 1999). A survey on 18 dairy herds in The Netherlands that included 197 lactating cows found an overall SARA prevalence of 13.8% across all stages of lactation (Kleen et al., 2009). In this survey, the prevalence on individual farms ranged from 0 to 13%. Kleen et al. (2013) included 315 cows on 26 farms in Northern Germany for their survey and determined a SARA prevalence of 20% with a great variation in the SARA prevalence among farms. Morgante et al. (2007) surveyed 12 dairy farms in Italy and included 10 cows in each herd between 5 and 60 days in milk (**DIM**). They found that in 3 out of the 10 herds the prevalence of SARA was greater than 33%. The results of these surveys agree on how common SARA is in high yielding dairy cows; and that its prevalence varies among herds.

Causes of Rumen Acidosis

The rumen pH will drop when organic acids that are produced during fermentation by rumen microbes accumulate and rumen buffering is not sufficient to prevent the increase in acidity that this accumulation may cause (Kleen et al., 2003; Krause et al., 2006; Plaizier et al. 2008). These organic acids include short-chain fatty acids (**SCFA**), otherwise known as volatile fatty acids, and lactic acid. Lactic acid is about 10 times more acidic than the most common SCFA, like acetic acid, propionic acid, and butyric acid. Hence, a change in the ratio between lactic acid and SCFA has a major effect on the rumen acidity, and the rumen pH will drop greatly when lactic acid accumulates. The substrates for fermentation in the rumen are carbohydrates and nitrogen containing compounds, such as proteins, peptides, and amino acids. Amounts of rapidly fermentable carbohydrates result in the production of more organic acids in the rumen than equal amounts of structural carbohydrates, such as cellulose and hemicellulose. As a result, increasing the proportion of grain and decreasing the proportion of forages in the diet increases the production of SCFA, and possibly lactic acid, in the rumen (Kleen et al., 2003; Krause and Oetzel, 2006; Plaizier et al., 2008). Increasing the digestibility of the forage portion of the diet, i.e., by reducing its particle size or by feeding more highly degradable grass and pasture, will also increase the production of these acids in the rumen (Mertens, 1997; O'Grady et al., 2008).

The source and processing of the grain portion of the diet also affects the rate of production of acids during fermentation. This production rate is higher for barley grain than for corn grain, and the more the grain is processed, e.g. steam flaked vs. cracked corn, the more rapidly rumen degradable it becomes (Herrera-Saldana et al., 1990; Huntington, 1997).

Inorganic buffers, including bicarbonate, from the saliva buffer the rumen, as they contribute to the neutralization of the acids produced during fermentation. Generally, the longer a cow chews, the more saliva it produces, but this effect is not linear (Mertens, 1997; Maekawa et al., 2002; Yang and Beauchemin, 2006.). The amount of time that the cow chews is affected by the content of coarse fiber in the diet. This amount can be quantified by measuring the physically effective fiber (**peNDF**) content of the diet. Taking account only of the total NDF in a diet is not sufficient, as not all NDF stimulates chewing equally. The peNDF has been defined as the ability of a feed to stimulate chewing and saliva buffering in the rumen (Mertens, 1997). The main factor that determines the dietary peNDF content is the particle size distribution, as well as the NDF content of the diet, but other factors, such as particle fragility, can also play a role. Several techniques for the measurement of this particle size distribution have been proposed, and of these, the Penn State Particle Separator is the most commonly used. Studies on the relationship between peNDF and rumen buffering have not always shown a high correlation (Maekawa et al., 2002; Yang and Beauchemin, 2006; Plaizier et al., 2008). The highest correlation between these measures is observed when both rumen pH and dietary peNDF content are in the low end of their scales (Plaizier et al., 2008). In addition to stimulating saliva production, forages also contribute to rumen buffering through their intrinsic buffering capacity. This capacity is related to the protein content of the feed, which explains why alfalfa forages have a higher buffering capacity than corn silage (McBurney and Chase, 1983).

Next to their rate of production, the rate of clearance of the SCFA that are produced during fermentation determines if they accumulate in the rumen. These acids are mainly cleared from the rumen through absorption by the papillae

(Stone, 2004). Dirksen et al. (1984) concluded that the capacity for this absorption is related to the absorptive surface area and that this capacity is twice as high when high grain diets are fed compared to when very high-forage diets are fed. As a result, the capacity of the rumen papillae to absorb these acids varies throughout lactation. Based on these findings, Stone (2004) recommended to gradually increase the grain content of the diet during a 5-week period around calving. However, Stone (2004) also suggested that common dry-cow diets contain less forage than the high-forage diets used by Dirksen et al. (1984) and that when these common diets are fed, the reduction in SCFA absorption during the dry period will be less than that found by Dirksen et al. (1984) and the duration of the step-up grain feeding to gradually provide this capacity can be less than 5 weeks. In any case, many management recommendations have been made based on the findings of Dirksen et al. (1984). It is, therefore, of great importance to confirm these findings. Penner et al. (2011) concluded that not only the absorptive area of the rumen epithelia respond to increased grain feeding, but the capacity of epithelial cells for the uptake of SCFA also are altered, and several molecular adaptations of these cells occur. Cows vary in the adaptations of their rumen epithelia to high grain feeding, and this variation may be partially caused by differences in genetics (Penner et al., 2011). This may help explain why cows differ in their susceptibility to rumen acidosis and may offer opportunities for the selection of cows that are less susceptible for rumen acidosis.

Symptoms of Rumen Acidosis

Many symptoms have been attributed to SARA, including feed intake depression, milk fat depression, reduced fiber digestion, rumenitis, systemic inflammation, dehydration, diarrhea, laminitis, liver abscesses, and increases in the concentrations of LPS in digesta throughout

the digestive tract (Kleen et al, 2003; Krause and Oetzel, 2006; Plaizier et al., 2008). Many of these relationships were demonstrated in observational studies in which causal relationships between SARA and these symptoms are difficult to prove or in studies in which SARA was experimentally induced. These inductions of SARA were conducted by feeding excessively high grain diets (Khafipour et al., 2009a; Li et al. 2012a), by a combination of feed restriction and high grain diets (Krause and Oetzel, 2006; Dohme et al., 2007), or by feeding pellets of ground forages (Khafipour et al., 2009b; Li et al. 2012b). Such feeding practices are unlikely to occur on commercial dairy farms. Hence, the question arises of how representative the experimentally induced SARA are for SARA that occurs on these commercial farms. Attributing symptoms to SARA is further complicated by the variety of techniques and definitions that are used for the diagnosis of this disorder.

Milk fat depression is the symptom most commonly associated with rumen acidosis (Kleen et al, 2003; Krause and Oetzel, 2006; Plaizier et al., 2008). This association is most likely caused by the incomplete biohydrogenation of dietary fatty acids that occurs during an excessive drop in rumen pH (Bauman and Griinari, 2003). This change in biohydrogenation increases the production of certain isomers of unsaturated fatty acids that reduce milk fat synthesis. The milk fat depression resulting from ruminal acidosis is likely one of the costs of this disorder to dairy farmers in North America.

Inducing ruminal acidosis by feeding excessively high grain diets has consistently increased markers of inflammation in blood (Plaizier et al., 2012). A low rumen pH may cause inflammation of the rumen wall, i.e. rumenitis (Kleen et al., 2003; Plaizier et al., 2012). However, a low rumen pH has also been associated with a reduced barrier function of

the rumen epithelium and an increase in the content of bacterial endotoxins (LPS) in rumen digesta (Plaizier et al., 2012). This combination may cause passage of LPS and other toxins, and pathogens from the rumen into the interior circulation and, thereby, resulting in an immune response. In most studies, this immune response has been characterized by an increase in acute phase proteins in blood but not by fever and other severe signs of inflammation (Plaizier et al., 2012). It is not yet clear the level of impact of the low level inflammation during ruminal acidosis. However, during stresses, such as ruminal acidosis and subsequent inflammation, nutrients are diverted away from production (Elsasser et al., 1997). Hence, the cost of this inflammation is not negligible.

In contrast to inducing ruminal acidosis by excessively high grain feeding, inducing this disorder by feeding pelleted forages does not result in inflammation (Khafipour et al., 2009b, Plaizier et al., 2012). This shows that a rumen pH depression alone does not cause inflammation of the rumen epithelium or damage the barrier function of the rumen epithelium (Plaizier et al., 2012). It has, therefore, been suggested that a combination of a low pH and high LPS and starch contents of digesta in the large intestine is responsible for the systemic inflammation during grain-induced ruminal acidosis.

Effects on Microbial Communities in the Digestive Tract

As inducing ruminal acidosis substantially alters the conditions in the rumen (Krause and Oetzel, 2005; Dohme et al., 2006; Plaizier et al., 2012), it can be expected that the induction greatly affects microbial communities in the rumen greatly. Among the rumen conditions that are affected by these inductions are rumen pH, the concentrations of SCFA, ammonia, and various substrates for microbial

fermentation, as well as the osmolality of rumen digesta (Krause and Oetzel, 2006; Dohme et al., 2007; Plaizier et al., 2012). It has also been demonstrated that the induction of ruminal acidosis also can alter these conditions in the large intestine (Li et al., 2012a; Plaizier et al., 2012). Due to the complexity of these changes, it is difficult to isolate which of these changes affect rumen communities the most.

Rumen microbes vary in their tolerance to a low pH, and as a result, rumen acidosis is expected to reduce populations of cellulolytic and gram-negative bacteria, reduce populations of protozoa, and increase populations of gram-positive cocci and rods (Nagaraja et al., 1978; Wells and Russell, 1996; Goad et al., 1998.). Khafipour et al. (2009c) observed that grain-induced ruminal acidosis reduced the population of gram-negative *Bacteroidetes* bacteria, including *Prevotella albensis*, *Prevotella brevis*, and *Prevotella ruminicola* in rumen fluid and that the severity of the rumen acidosis determined the reduction of these bacteria. The severity of the rumen acidosis in this study was determined not only based on the rumen pH depressions but also on the increases in bacterial endotoxins and the immune response that it caused. Severe ruminal acidosis also increased the populations of *Streptococcus bovis* and *Escherichia coli* in rumen fluid; whereas, its milder form increased the population of the lactate utilizer *Megasphaera elsdenii*. Khafipour et al. (2011a) concluded that ruminal acidosis does not only increase the population of *Escherichia coli* but also increases the presence of virulence genes in these bacteria.

Li et al. (2011) and Khafipour et al. (2011b) compared the effects of moderate ruminal acidosis induced by high grain feeding and by feeding pelleted forages. They found that, despite similar rumen pH depressions, only the grain-induced ruminal acidosis reduced the

richness and diversity of bacterial species in the rumen, large intestine, and feces. In contrast with the study from Khafipour et al. (2009c), both acidosis inductions increased *Prevotella bryantii* and *Selenomonas ruminantium*, but decreased *Streptococcus bovis* in rumen fluid. In addition, both of these inductions increased *Prevotella ruminicola*; whereas, only the grain-induced acidosis increased *Lactobacillus spp.* and *Escherichia coli*, and decreased *Streptococcus bovis* in the large intestine. Similarly, Li et al. (2013a, 2013b) showed that moderate grain-induced ruminal acidosis increased *Lactobacillus spp.*, *Megasphaera elsdenii*, and *Prevotella albensis*, and decreased *Prevotella brevis* and *Treponema bryantii*. This induction also increased *Lactobacillus spp.*, *Megasphaera elsdenii*, *Treponema bryantii*, and *Escherichia coli*. Comparing these studies show that the effects of ruminal acidosis on bacterial communities are substantial but differ greatly among experiments. This may be due to the differences in rumen conditions other than rumen pH and differences in conditions in the large intestine among experiments (Plaizier et al., 2008; Plaizier et al., 2012). As long as the ruminal acidosis is not too severe, microbial richness and diversity are not greatly affected, and the accumulation of lactate is prevented by increasing the populations of lactic acid utilizing bacteria and by preventing increases in lactate producing bacteria. Under these conditions, shifts in bacterial communities may reflect shifts in the availability of substrates for fermentation, rather than indicate undesirable conditions for the bacterial species whose populations are reduced. The severity of the rumen acidosis is only, in part, determined by the diets used for its induction, as cows vary greatly in their response to the SARA challenges (Plaizier et al., 2008; Plaizier et al., 2012). The factors that determine the susceptibility of cows to rumen acidosis are not yet well understood but are at least in part of genetic origin (Penner et al., 2011). These studies

did not confirm that ruminal acidosis reduces cellulolytic bacteria. In order to confirm this, analysis of bacterial communities in solid rumen digesta, next to the analyses of liquid digesta that were conducted in these studies, will be required, as most cellulolytic bacteria are in the solid digesta fraction (Wells and Russell, 1996; Goad et al., 1998). The bacterial populations that adhere to the rumen epithelium (epimural bacteria) differ greatly from those in solid and liquid rumen digesta and also are affected by changes in diet composition and induction of ruminal acidosis (Petri et al., 2013). Changes in the communities of epimural bacteria may have major effects on the rumen epithelium, including their barrier function and absorptive capacity. As changes in epimural bacterial communities are difficult to predict from changes in bacterial communities in solid and liquid rumen digesta (Petri et al. (2013), more research on the effects of ruminal acidosis on epimural bacteria is needed to better understand the etiology of this disease.

Prevention of Rumen Acidosis

Dietary fiber and non-fiber carbohydrates

Key in the prevention of rumen acidosis is to formulate diets that contain sufficient buffering capacity and peNDF; do not contain excessive amounts of non-fiber carbohydrates; and ensure that cows consume the diets the way they have been formulated. Therefore, in order to prevent SARA, diets must contain sufficient coarse fiber to stimulate chewing because this contributes to rumen buffering by stimulating the production of saliva which contains buffers. Diets are commonly balanced for total NDF, but this may not always ensure sufficient rumen buffering since not all NDF contributes equally to chewing and saliva production. For example, the NDF in long hay contributes much more to saliva production than the NDF in barley

grain. To circumvent this problem, the concept of peNDF has been developed. The peNDF of a feed is defined as the fiber that contributes to chewing, saliva production, and rumen buffering and is largely affected by feed particle size. The peNDF of forages is mainly determined by the chop length. Forages that are chopped too finely will put cows at risk for SARA. The peNDF content of the diet can be increased either by including more forage to increase both the dietary NDF and peNDF contents, or by increasing the chop length of forages (Beauchemin and Penner, 2009). The rationale is that either way of increasing the peNDF content of the diet increases chewing time and salivary secretion.

NRC (2001) recommends that when diets mainly contain alfalfa silage, corn silage, and ground corn, these diets should contain at least 25% of DM as NDF, as long as 19% of the DM is NDF from forages. NRC (2001) further recommends that when diets contain grains that are more rumen degradable than corn, such as barley, include forages with small particle sizes, and are not fed as TMR, they need to contain more than 25% of DM as NDF. Beauchemin (1991), therefore, recommended a minimum NDF content of 34% of DM for barley-based diets. NRC (2001) also recommends that the maximum dietary content of non-fiber carbohydrates can be 44% of DM, as long as the minimum dietary NDF and forage NDF contents are provided and corn is the grain source in the diet. However, NRC (2001) also indicates that due to possible errors with mixing and feed delivery, it is highly recommended to use a safety margin for the maximal dietary non-fiber carbohydrate content.

Currently, several methods to determine peNDF are used. If the peNDF is determined as the proportion of the diet retained by the 0.75” and 0.31” screens of the Penn State

Particle Separator (**PSPS**), then a minimum dietary peNDF content of 19 to 20% may be sufficient (Plaizier et al., 2008). Other research groups have, however, come up with slightly different minimum peNDF requirements. It is clear that these requirements depend on how much concentrate is included in the diet and which forages and grains are used. Yang and Beauchemin (2009) concluded that, due to inconsistent research results, firm conclusions on the minimum dietary peNDF content can still not be drawn. Heinrichs (2013) recommended that the 0.75", 0.31", and 0.16" screens of the PSPS should contain 2 to 8, 30 to 50, and 10 to 20% of TMR, and that 30 to 40% of these rations should be retained by the bottom pan of the PSPS. Heinrichs (2013) also provided recommendations of particle size distributions of corn silage and haylage. Although the impact of deviating from these recommendations is not immediately clear, it is highly recommended to regularly check diets for lactating cows with the PSPS.

Feed delivery and prevention of sorting

Diets that cows consume frequently differ from those that have been formulated due to errors in mixing, excessive mixing, and feed sorting by cows. Errors in mixing can occur when the components of the diet are not weighed in accurately and if the DM contents of forages are very different than assumed. Other nutritional management strategies to prevent SARA include presenting the feed in a form that promotes the consumption of a more consistent ration, such as the use of TMR instead of component feeding systems, as well as increased feed access through frequent feeding throughout the day. Several experiments conducted by our research group and in Wisconsin have shown that cows select against large feed particles in favor of smaller feed particles (Leonardi and Armentano, 2003). This means that even if the diet contains

sufficient peNDF, the portion that is consumed might not. Cows are better able to sort very dry diets compared to wetter diets. Hence, feeding diets that are not excessively dry will reduce sorting. Some dairy farmers add water to the TMR to reduce DM content in order to reduce sorting. Adding water is meant to bind ration particles together and make it difficult to sorting against the larger forage particles.

However, adding water to TMR has yielded mixed results in studies reported in the literature. Some studies showed that adding water to reduce a TMR DM from 80 to 64% DM reduced sorting for the smallest particles (Leonardi et al., 2005). In other studies reducing TMR DM concentration from 58 to 48% resulting in more sorting and lower DMI (Miller-Cushon and DeVries, 2009). Adding water to the rations can potentially lower DM intake because of the filling effect of higher moisture rations. Adding liquid feed, such as molasses, instead of water to a dry TMR has been shown to prevent sorting by binding ration particles together and making it difficult for cows to sort against the larger forage particles (DeVries and Gill, 2012).

Additionally, sorting can be minimized by avoiding excessive amounts of long material in the TMR. It has been suggested that added hay or straw should not be longer than 2.5 to 5 cm (1.0 to 2.0 in) (Hall, 2002; Shaver, 2002). Such feeding behavior may increase during heat stress as the cows attempt to reduce metabolic heat production by selecting away from forage toward concentrate (McDowell, 1972). Therefore when conditions of temperature and relative humidity are likely to lead to heat stress, it is recommended that dietary peNDF content be increased and non-fiber carbohydrates content decreased (Stone, 2004).

If herds are at risk of ruminal acidosis, increasing the frequency of feed delivery should

be considered. The provision of fresh feed will stimulate eating (DeVries et al., 2003a). As a result, supplying feed more than once daily will spread out the meals of cows during the day. This will reduce diurnal variation in rumen pH, and therefore, reduce the risk of rumen acidosis. Other conditions that reduce rumination and the distribution of meals throughout the day, such as insufficient and competition for bunk space, limited access to feed, and long milk parlor holding times, will also increase the risk of SARA (Stone, 2004). DeVries et al. (2003b) suggested that a feeding space of 24”/cow in a freestall, will not allow all cows to access feed simultaneously, and the change in feeding pattern that this causes increases the risk for rumen acidosis.

Dietary buffers and yeast

Exogenous dietary buffers, such as sodium bicarbonate, can be added to high grain diets at rates ranging from 0.75 to 1% of DM to complement the intrinsic buffering from the forage components of the diet (Krause, 2008). These buffers have been found to be beneficial in stimulating DMI in corn-silage based diets only but not with alfalfa or grass silage based diets (Erdman, 1988; Staples and Lough, 1989). This is probably because of the relatively higher intrinsic buffering capacity of alfalfa and grass silage compared to corn silage. Force-feeding buffers is not practical and would be expensive as all animals, including those that do not require buffers, would be fed. Alternatively, free choice feeding of buffers does not appear to alleviate SARA since cows that are affected by SARA do not appear to ‘correct’ this imbalance in rumen environment by increasing intake of sodium bicarbonate (Keunen et al., 2003). In addition to the added buffers, the diet also has intrinsic buffering capacity that is an inherent property of the feed ingredients used in diet formulation. Intrinsic buffering is largely explained by dietary cation-anion difference (DCAD) (i.e., (Na + K)

– (Cl + S)). Diets with high net positive DCAD values tend to support higher rumen pH and increase DMI and milk yield (Sanchez et al., 1994; Block and Sanchez, 2000) with optimum early and mid-lactation DCAD values of about +400 mEq/kg (+182mEq/lb) and +275 to +400 mEq/kg +125 to +182 mEq/lb), respectively. Alfalfa forages tend to have a higher DCAD than corn silage, and concentrate feeds generally have lower or negative DCAD, which adds to their high potential to cause SARA because of the high fermentable carbohydrate content (Oetzel, 2007).

In vitro studies suggested that *Saccharomyces cerevisiae* yeast interact with rumen bacteria to reduce lactate accumulation (Chaucheyras et al., 1996), which implies that yeast supplementation may be beneficial to the cows suffering from ruminal acidosis. However, Moya et al., (2009) did not observe that a *Saccharomyces cerevisiae* fermentation product (SCFP, Original XPC, Diamond V) reduced the effect of a grain-based ruminal acidosis challenge on rumen fermentation. In contrast, Li et al. (2013a) reported that SCFP stabilized ruminal pH.

Adaptation to high grain diets

A rapid increase in the concentrate inclusion in the diet also will put cows at risk for SARA, as the rumen microbes and the cows (especially the rumen wall) need to adapt to the change in diet. Lead feeding, the practice of increasing the proportion of concentrate in the TMR during the last few weeks prior to calving; has become common practice in North America. Even though increasing the amount of concentrate fed during the close-up dry period will reduce SARA in fresh cows, it is possible that errors in which too much concentrate is fed can also increase the risk for SARA. However, the risks of this practice resulting in

overconsumption of concentrate that would cause SARA are minimal because of low DMI during the transition period (Bertics et al., 1992). Instead, insufficient concentrate feeding during the transition period may increase the risk of SARA through failure to increase the SCFA absorptive capacity of the ruminal papillae, as well as due to failure to adapt the rumen microbial population to high concentrate diets (Dirksen et al., 1984). Hence, it is recommended to start feeding close-up dry cows more grain starting 3 weeks before calving, as this will adapt the rumen microbes and the rumen papillae to the high concentrate diet that will be fed after calving. Close-up dry cows should receive diets with 8 to 10 lbs/day of grain by calving time (NRC, 2001).

Other Factors

Another important point that can be overlooked is ration preparation. There is a possibility for there to be significant variation in forage DM and nutrient composition which may create a potential for a feeder to deliver rations with different composition with each load. This variation can be minimized through regular sampling and laboratory analysis and adjustment of the formulation to match changes in forage quality. Thus, forage samples should be taken and analyzed with wet chemical procedures. Even with feeding systems where samples are collected and tested regularly, variation in DM and chemical constituents have been shown to exist (Stone, 2004). Regular checking of the DM of the forages and the diets is, therefore, important precaution. A quick DM test using a microwave oven can be used for this purpose. This would reduce errors in the mixing of diets, which can occur when the DM of the forages is different from the DM used in the diet formulation.

Excessive mixing can reduce the size of coarse feed particles, and thereby, reduce the

peNDF content of the diet. It is, therefore, highly recommended to determine what the minimum time is needed to obtain a good feed mix and use this time for future feed preparations.

Additionally, the rumen pH depression may be exacerbated by the type of grain fed (corn vs barley), harvest /storage method (dry vs high moisture), and degree of processing (rolled, ground, or steam-flaked) (Nocek and Tamminga, 1991; Shaver, 2005). These factors often affect both the amount and degree of starch degradability in the rumen. The challenge that animal nutritionists face is finding nutritional interventions that might prevent SARA without limiting grain feeding. Some of the approaches that have been suggested include supplementation with specific yeast strains that enhances lactate utilization in the rumen under certain specific conditions (Dawson, 1995), and preconditioning rumen microbes to lactate by adding it to the diet to enhance the ability of the microbes to adapt to sudden increase in lactate (Owens et al., 1998). Alternatively, direct-fed microbials also might be added to the rumen of cows (Nocek et al., 1999). Additionally, feeding ionophores reduce ruminal lactate production, possibly through the inhibitory effects of ionophores on lactate-producing bacteria (Owens et al., 1998).

Conclusions

Diagnosis of ruminal acidosis remains difficult, but many management strategies are available that can reduce the risk of this disease. Nutritional management both in terms of DM and chemical composition remain the major way to control SARA in cattle. However, environmental factors, such as insufficient bunk space and social dominance, also can affect the feeding patterns and predispose some animals to SARA. Some of the basic principles of reducing SARA include providing sufficient peNDF

while limiting the intake of readily fermentable carbohydrates and ensuring that cows are adequately adapted to high grain diets. Thus, the importance of regular monitoring of feeds used in diet formulation, as well as ensuring that the diet that is formulated closely matches what the cows consumes, cannot be over-emphasized.

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