Subacute Ruminal Acidosis in Dairy Cattle

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Take Home Messages

- Ruminal acidosis is as much an important economic and health issue for dairy herds as it is for beef feedlots.
- Ruminal acidosis is the result of total intake of fermentable carbohydrate and cannot be predicted by low fiber density alone.
- Cows possess a number of complex mechanisms to keep their ruminal pH above the biologic threshold of about 5.5.
- Cows self-correct low ruminal pH by eating less; lower production results.
- The clinical effects of subacute ruminal acidosis are delayed from the time of the acidotic insult.
- Milk fat depression is not a consistent feature of ruminal acidosis.
- Forage particle length and grain particle size are important determinants of the risk for subacute ruminal acidosis.
- High dry matter intake and over-eating following periods of feed deprivation are often over-looked as important causes of subacute ruminal acidosis.

Introduction

Ruminal acidosis is the consequence of feeding high grain diets to ruminant animals, which are adapted to digest and metabolize predominantly forage diets. Feeding diets that are progressively higher in grain tends to increase milk production, even in diets containing up to 75% concentrates. However, short-term gains in milk production are often substantially or completely negated by long term adverse effects on cow health.

Compromises in dairy cow health due to subacute ruminal acidosis (SARA) are a concern not only for economic reasons, but also for animal welfare reasons. Lameness is probably the most important animal welfare issue today in dairy

herds, and a good portion of the lameness observed in dairy cows may be attributed to laminitis secondary to high grain feeding. Public perception of dairy production is becoming increasingly important, and lame cows do not portray a good image of our industry. Lameness (along with secondary reproductive failure and low milk production) is commonly the most important cause of premature, involuntary culling and unexplained cow deaths in a dairy herd.

Ruminal acidosis can be a direct human health concern as well. Low ruminal and intestinal pH due to high grain feeding increases the risk for shedding enterohemorrhagic *E. coli* such as 0157:H7 (Russell and Rychlik, 2001). Switching cattle to a high forage diet just prior to slaughter decreases this shedding.

Dairy production in areas with relatively inexpensive grains and with no limit to the amount of milk they can market (e.g., the US) are likely to experience the most ruminal acidosis. The economics of dairy production under these circumstances favor heavier grain feeding. However, producers, nutritionists, and veterinarians may be ignorant of the health costs of ruminal acidosis and therefore may be reluctant to decrease grain feeding.

■ Physiology of Ruminal pH

Fermentable Carbohydrate Intake.

Ruminal pH drops below physiological levels when ruminants consume excessive amounts of rapidly fermentable (non-fiber) carbohydrates. Each cow's inherent capacity to buffer and absorb acid determines how much her ruminal pH will fall after a meal containing large amounts of fermentable carbohydrates.

Dairy cattle and beef feedlot cattle are at similar risk to develop ruminal acidosis. Although dairy cattle are typically fed diets that are higher in forage and fiber compared to beef feedlot cattle, this is offset by their much higher dry matter intakes. Total consumption of non-fiber carbohydrates is similar between these two classes of livestock (Table 1). Not surprisingly, ruminal pH values measured by continuous data acquisition in feedlot steers and lactating dairy cattle were similar when the cattle consumed similar total amounts of non-fiber carbohydrates. The prevalence of ruminal acidosis in dairy herds is probably about the same as it is in beef feedlots.

Total daily intake of ruminally fermentable carbohydrates depends equally on total dry matter intake and the density of non-fiber carbohydrates in the diet. High intakes in dairy cattle are associated with lower ruminal pH. This

suggests that ruminal acidosis will become an even more common problem as genetic progress and better feeding management allow cows to eat more.

Table 1. A comparison of diet and ruminal pH in beef feedlot and lactating dairy cow studies.

Item	Steer study[a]	Lactating cow study[b]
Experimental animals	8 Holstein steers	8 Holstein cows
Stage of feeding or lactation	Compensatory gain	Early lactation
Average daily ruminal pH[c]	5.99	5.90
Forage in diet, %	26.3	52.9
Daily dry matter intake, kg	13.2	21.4
Neutral detergent fiber, %	20.0	28.9
Non-fiber carbohydrates,[d] %	58.3	36.9
Non-fiber carbohydrates, kg/day	7.73	7.91

[a] Data adapted from (Prentice, Schaefer, and Oetzel, 2000).
[b] Data adapted from (Oetzel and Nordlund, 1998).
[c] Ruminal pH was measured once per minute by indwelling ruminal electrode and averaged daily.
[d] Non-fiber carbohydrates (NFC), calculated as 100 – % crude protein - % neutral detergent fiber - % ether extract - % ash.

Ruminal pH varies considerably during the course of a day, and is particularly driven by the amount of fermentable carbohydrate in each meal. Shifts of 0.5 to 1.0 pH units within a 24-hour period are common. This represents a 5 to 10-fold change in hydrogen ion concentration in the rumen (pH is a log base 10 transformation of hydrogen ion concentration).

Feeding TMR more frequently (e.g., six times daily) will not necessarily solve the problem of post-feeding ruminal pH variation. In fact, more frequent feeding will increase dry matter intake and can lower ruminal pH more than less frequent feeding.

The enormous changes in ruminal pH after eating make it very difficult to evaluate ruminal pH, even in research settings. Continuous acquisition of ruminal pH data by indwelling electrode provides the most information possible about post-feeding variations in ruminal pH.

Intake Depression.

Because ruminal acid production from fermentation of carbohydrates varies so much from meal to meal, ruminants possess highly developed systems to maintain ruminal pH within a physiological range of about 5.5 to 7.0. As ruminal pH begins to drop following a meal, the ruminant's first response is to stop eating. Low ruminal pH may also be associated with increased osmolality of the ruminal contents, which in turn inhibits feed intake. Inflammation of the

ruminal epithelium (rumenitis) could also play a role in depressing feed intake following ruminal acidosis.

Endogenous Buffering.

Ruminant animals possess a complex system for buffering the organic acids produced by ruminal fermentation of carbohydrates. While the total effect of buffering on ruminal pH is relatively small, it can still account for the margin between health and disease in dairy cows fed large amounts of fermentable carbohydrates. Cows produce a large amount of buffers via their saliva. Saliva buffers ruminal pH because it is rich in sodium, potassium, bicarbonates, and phosphates. Unfortunately, saliva production is not triggered by declining ruminal pH, but rather is determined almost entirely by the amount of physical fiber present in the diet. Saliva is secreted during chewing activity (eating and rumination) in response to the amount of physical fiber present in the diet.

Acid Absorption.

The ability of the rumen to rapidly absorb organic acids from the rumen contributes greatly to the stability of ruminal pH. It is rarely difficult for peripheral tissues to utilize VFA already absorbed from the rumen; however, absorption of these VFA from the rumen can be an important bottleneck.

Absorption of VFA from the rumen occurs passively across the ruminal wall. This passive absorption is enhanced by finger-like papillae, which project away from the rumen wall. These papillae provide a huge surface area for VFA absorption. Ruminal papillae increase in length when cattle are fed higher grain diets; this presumably increases the surface area and absorptive capacity, which protects the animal from acid accumulation in the rumen. If the absorptive capacity of these cells is impaired (e.g., chronic rumenitis with fibrosis), then it is difficult for the animal to maintain a stable ruminal pH following a meal.

Net Ruminal pH Regulation.

Mean ruminal pH values are not dramatically affected by large dietary changes, but the lowest (nadir) pH values are greatly affected by diet. This response is in keeping with the nature of regulation of ruminal pH that has been described above. Cattle are generally able to maintain ruminal pH within physiological limits by their own regulation of intake, endogenous buffer production, microbial adaptation, and VFA absorption. However, if the amount of fermentable carbohydrate consumed results in more acid production than the system can accommodate, ruminal pH compensation fails and ruminal pH drops drastically.

As ruminal pH drops below the physiological threshold of about 5.5 (for fluid collected by rumenocentesis), cattle develop SARA. Fortunately, ruminal VFA

have a pKa of about 4.9, which means that they are rapidly shifting toward the undissociated (protonated) form at this pH. This removes a free hydrogen ion from the ruminal fluid and greatly facilitates their absorption across the ruminal epithelium, since only undissociated acids can be passively absorbed.

Lactate Production.

Unfortunately, gains in VFA absorption at ruminal pH below 5.5 can be offset by lactate production. As pH drops, *Strep. bovis* begins to ferment glucose to lactate instead of VFA. This is a dangerous situation, since lactate has a much lower pKa than VFA (3.9 vs. 4.8). At a ruminal pH of 5.0, for example, lactate is 5.2 times less dissociated than VFA. As a result, the lactate stays in the rumen and contributes to the downward spiral in ruminal pH. Fiber-digesting bacteria are severely inhibited as pH declines. Pure lactate producers such as *Lactobacillus* may begin to proliferate at these lower pHs.

Additional adaptive responses are invoked when ruminal pH drops below 5.5 and lactate production begins. Lactate-utilizing bacteria, such as *Megasphaera elsdenii* and *Selenomonas ruminantium* begin to proliferate. These beneficial bacteria convert lactate to other VFA, which are then easily protonated and absorbed. However, the turnover time of lactate utilizers is much slower than for *Strep. bovis*. Thus, this mechanism may not be invoked quickly enough to fully stabilize ruminal pH. Periods of very high ruminal pH, as during feed deprivation, may inhibit populations of lactate utilizers (which are sensitive to higher ruminal pH) and leave them more susceptible to severe ruminal acidosis. Other aspects of this delicate ruminal microbial balance may be disrupted when ruminants are deprived of feed for a time.

Besides disrupting microbial balance, feed deprivation causes cattle to overeat when feed is re-introduced. This creates a double effect in lowering ruminal pH. As a result, cycles of feed deprivation and re-feeding are more important risk factors for SARA than is diet formulation itself.

■ Pathophysiology of Ruminal Acidosis in Dairy Cattle

The ruminal epithelial cells are not protected by mucus (as abomasal cells are), so they are vulnerable to the chemical damage by acids. Thus, low ruminal pH leads to rumenitis, erosion, and ulceration of the ruminal epithelium. Rumenitis is the fundamental lesion of SARA, and it leads to delayed, chronic health problems.

Once the ruminal epithelium is inflamed, bacteria may colonize the papillae and leak into portal circulation. These bacteria may cause liver abscesses, which sometimes cause peritonitis around the site of the abscess. If the ruminal bacteria clear the liver (or if bacteria from liver infections are released into

circulation), they may colonize the lungs, heart valves, kidneys, or joints. The resulting pneumonia, endocarditis, pyelonephritis, and arthritis are all chronic inflammatory diseases that are difficult to diagnose ante-mortem. Post-mortem monitoring of these conditions in cull cows or cows that die on the dairy could be very beneficial, but has not been described.

Caudal vena cava syndrome can cause bleeding from both nostrils and sudden deaths due to massive pulmonary hemorrhage in cows that are affected with SARA. In these cases, septic emboli from liver abscesses lead to lung infections, which ultimately invade pulmonary vessels and cause their rupture.

SARA may also be associated with laminitis and subsequent hoof overgrowth, sole abscesses, and sole ulcers. These foot problems generally do not appear until weeks or months after the bout of ruminal acidosis that caused them. The mechanism by which SARA increases the risk for laminitis has not been characterized. New information from equine studies suggests that metalloproteinase enzymes produced by *Strep. bovis* in the hindgut may be the inciting cause for laminitis. If these enzymes are indeed the culprits in dairy cattle laminitis, then they may be of hindgut origin as well. This hypothesis has not been tested in cattle, but some data support the concept that laminitis is the result of abnormal hindgut fermentation of carbohydrates instead of ruminal acidosis *per se*.

■ Milk Fat Depression and Ruminal Acidosis

The relationship between milk fat test and ruminal pH is very weak. Cows and herds with severe SARA may have normal milk fat tests. Thus, it is vitally important not to exclude a diagnosis of SARA just because the herd has a normal milk fat test.

New research on the mechanism of milk fat depression supports this general concept. We now know that milk fat depression is not caused by a relative shortage of ruminal acetate for milk fat synthesis in the udder. Since SARA does shift ruminal VFA production away from acetate towards propionate, the incorrect assumption followed that milk fat depression was a necessary consequence of low ruminal pH.

In fact, milk fat depression is caused by incomplete saturation (biohydrogenation) of fatty acids within the rumen. Incompletely saturated fatty acids (particularly certain *trans* forms) are potent inhibitors of milk fat synthesis in the mammary gland. Feeding high levels of unsaturated dietary fatty acids is one means of increasing *trans* fatty acid uptake from the rumen. Ruminal acidosis may also increase *trans* fatty acid uptake, perhaps by inhibiting certain bacteria, which are normally responsible for fatty acid biohydrogenation in the rumen.

■ Nutritional Management Issues in Subacute Ruminal Acidosis

Once a diagnosis of SARA has been established in a herd, then the cause of the acidosis must be determined before appropriate preventive measures can be instituted. Causes of ruminal acidosis can be grouped into three categories: excessive intake of rapidly fermentable carbohydrates, inadequate ruminal buffering, and inadequate ruminal adaptation to a highly fermentable diet.

Excessive Intake of Rapidly Fermentable Carbohydrates.

This is the most obvious cause of ruminal acidosis in dairy cattle. Because of their relatively high dry matter intakes, dairy cattle cannot tolerate diets as proportionately high in concentrates as beef feedlot diets. An important goal of effective dairy cow nutrition is to feed as much concentrate as possible, in order to maximize production, without causing ruminal acidosis. This is a difficult and challenging task because the indications of feeding excessive amounts of fermentable carbohydrates (decreased dry matter intake and milk production) are very similar to the results from feeding excessive fiber (again, decreased dry matter intake and milk production). An important distinction is that even slightly over-feeding fermentable carbohydrates causes chronic health problems, while slightly under-feeding fermentable carbohydrates does not compromise cow health.

Evaluating the diet for chemical fiber density is an important first step in determining the cause of SARA in a dairy herd. This requires a careful evaluation of the ration actually being consumed by the cows. A cursory evaluation of the “paper” ration formulated by the herd nutritionist is usually of little value. Ascertaining the ration actually consumed by the cows requires a careful investigation of how feed is delivered to the cows, accurate weights of the feed delivered, and updated nutrient analyses of the feeds delivered (particularly the dry matter content of the fermented feed ingredients). Careful bunk sampling of total mixed rations (TMR) may uncover unknown errors in feed composition or feed delivery.

Dairy herds that use component feeding in early lactation often bring cows up on grain faster than their actual rise in dry matter intake. This puts cows at great risk for SARA, since they cannot eat enough forage to compensate for the extra grain consumed. Careful modeling of early lactation diets in such herds often reveals drastic fiber deficiencies around one to three weeks post-calving. As a general rule, cows should receive no more than 3.5 to 5.5 kg of dry matter from grain in the first week after calving. Grain feeding should then increase by about .10 to .25 kg/cow/day until peak grain feeding is reached at six to eight weeks post-calving.

The physical form of feed ingredients can be just as important as their chemical composition in determining how rapidly and completely they are fermented in the rumen. Grains that are finely ground, steam-flaked, extruded, and/or very wet will ferment more rapidly and completely in the rumen than unprocessed or dry grains, even if their chemical composition is identical. Similarly, starch from wheat or barley is more rapidly and completely fermented in the rumen that starch from corn. Corn silage that is very wet, finely chopped, or kernel-processed also poses a greater risk for SARA than drier, coarsely chopped, or unprocessed corn silage.

Particle size analysis of grains is a useful adjunct test when assessing the risk for SARA in a dairy herd. Very finely ground grains, especially if they are moist, will increase their rate of fermentation in the rumen and increase the risk for SARA.

Dairy cattle groups are commonly fed for *ad libitum* intake (typically a 5% daily feed refusal) in order to maximize potential dry matter intake and milk yield. However, slightly limiting intake in dairy cattle at high risk for SARA would in theory reduce their risk of periodic over-consumption and SARA. Feed efficiency might also be improved. This approach has been successfully used in beef feedlots. However, dairy cow groups are much more dynamic than feedlot groups. This makes it considerably more challenging for dairy cattle feeders to slightly limit intakes without letting the feed bunks be without palatable feed more than about four hours a day. It can be done, but only with adequate bunk space and excellent feed bunk management. Perhaps *ad libitum* feeding with a 5% daily feed refusal is the best option for most dairy herds. This would especially apply to the pre- and post-fresh cow groups because they have rapid cow turnover and because individual cows have rapidly changing dry matter intakes during these time periods.

Inadequate Ruminal Buffering.

Ruminant animals have a highly developed system for buffering the organic acids produced by ruminal fermentation of carbohydrates. While the total effect of buffering on ruminal pH is relatively small, it can still account for the margin between health and disease in dairy cows fed large amounts of fermentable carbohydrates.

Dietary buffering is the inherent buffering capacity of the diet and is largely explained by dietary cation-anion difference (DCAD). Diets high in Na and K relative to Cl and S have higher DCAD concentrations, tend to support higher ruminal pH, and increase dry matter intake and milk yield. Optimal DCAD for early lactation diets is about +400 mEq/kg of (Na +K) – (Cl + S). Mid-lactation cows have an optimal DCAD of about +275 to +400 mEq/kg. Formulating diets with a high DCAD typically requires the addition of buffers such as sodium bicarbonate or potassium carbonate. Alfalfa forages tend to have a higher

DCAD than corn silage, although this depends considerably on the mineral composition of the soil on which they were grown. Concentrate feeds typically have low or negative DCAD, which adds to their already high potential to cause ruminal acidosis because of their high fermentable carbohydrate content.

Endogenous buffers are produced by the cow and secreted into the rumen via the saliva. The amount of physical fiber in the diet determines the extent of buffer production by the salivary glands. Coarse, fibrous feeds contain more effective fiber and stimulate more saliva production during eating than do finely ground feeds or fresh pasture. Coarse, fibrous feeds also make up the mat layer of the rumen, which is the stimulus for rumination. Fiber particles longer than about 3.5 cm contribute the most to mat layer formation. Rumination promotes much chewing activity and therefore the secretion of large amounts of saliva into the rumen. Ruminal pH increases noticeably during bouts of rumination.

Endogenous buffering can be estimated by observing the number of cows ruminating (a goal is at least 40% of cows ruminating at any given time) and by measuring the particle length of the TMR actually consumed by the cows using the Penn State Forage Particle Separator. Diets with less than 7% long particles put cows at increased risk for SARA, particularly if these diets are also borderline or low in chemical fiber content. Increasing chemical fiber content of the diet may compensate for short particle length.

Diets with excessive (over about 15%) long forage particles can paradoxically increase the risk for SARA. This happens when the long particles are unpalatable and sortable. Sorting of the long particles occurs soon after feed delivery, causing the cows to consume a diet that is low in physically effective fiber after feeding. The diet consumed later in the feeding period is then excessively high in physically effective fiber and low in energy. Socially dominant cows are particularly susceptible to SARA in this scenario, since they are likely to consume more of the fine TMR particles soon after feed delivery. Cows lower on the peck order then consume a very low energy diet. Thus, cows on both ends of the social spectrum become thin and produce poorly. Limiting bunk space to less than 75 cm per cow exacerbates the effect of TMR sorting in a group of cows. Sorting of long particles during the feed-out period can be evaluated by conducting sequential analysis of the TMR bunk samples at differing times after feeding.

Inadequate Adaptation to Highly Fermentable, High Carbohydrate Diets.

Cows in early lactation may be particularly susceptible to SARA if they are poorly prepared for the lactation diet they will receive. Ruminal adaptation to diets high in fermentable carbohydrates apparently has two key aspects – microbial adaptation (particularly the lactate-utilizing bacteria, which grow more slowly than the lactate-producing bacteria) and ruminal papillae length (longer papillae promote greater VFA absorption and thus resist ruminal pH decline).

The known principles of ruminal adaptation suggest that increasing grain feeding toward the end of the dry period should decrease the risk for SARA in early lactation cows. However, a recent field study in TMR-fed herds found no effect of dry period feeding on early lactation ruminal pH. Ruminal pH was unexpectedly lower in cows at 106 average days in milk compared to cows at 15 average days in milk (Garrett and others, 1997). These results suggest that high dry matter intake is a more important risk factor for SARA than ruminal adaptation problems in dairy herds. Also, a controlled study in component-fed cows found no positive effect of increased grain feeding during the dry period on early lactation ruminal pH or dry matter intake (Andersen, Sehested, and Ingvartsen, 1999). These results suggest that the practical impacts of ruminal adaptation may be small in dairy herds, and particularly when cows are fed a TMR after calving.

■ Prevention of Subacute Ruminal Acidosis in Dairy Herds

The basic principles of preventing SARA in dairy herds have been discussed above and include limiting the intake of rapidly fermentable carbohydrates, providing adequate ruminal buffering, and allowing for ruminal adaptation to high grain diets. However, I expect SARA to remain an important dairy cow problem even when these principles are understood and applied, because the line between optimal milk production and over-feeding grain is exceedingly fine. In many dairy herd situations, milk production can appear to be temporarily increased by over-feeding grain and causing SARA; however, the long-term health and economic consequences of this approach are devastating.

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