Clinical Aspects of Ruminal Acidosis in Dairy Cattle

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Introduction

Acute and subacute ruminal acidosis are well recognized as important diseases in beef feedlots.[6,14,32,37] Acute ruminal acidosis has been long recognized in dairy cattle, although deaths due to acute ruminal acidosis are apparently less frequent in dairy cattle than in beef feedlot cattle.[35] Only recently has subacute ruminal acidosis (SARA) been described for dairy cattle.[26]

Although dairy cattle are typically fed diets higher in forage and fiber compared to beef feedlot cattle, total consumption of rapidly fermentable (non-fiber) carbohydrates is similar between these two livestock classes because lactating dairy cows have very high feed intakes. This principle is illustrated by the data presented in Table 1. 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 SARA in dairy herds is probably about the same as it is in beef feedlots.

The objectives of this paper are to review the pathophysiology, clinical signs, diagnostic methods and prevention of ruminal acidosis in dairy herds. The subacute form of ruminal acidosis will be emphasized.

Acute vs. Subacute Ruminal Acidosis in Dairy Cattle

Acute and subacute ruminal acidosis share a similar etiology but are very different clinical diseases. The general definitions used in beef feedlot cattle[32] for these two disorders have been applied to dairy cattle.[17,26] In acute ruminal acidosis, an excessive intake of rapidly fermentable carbohydrates results in a sudden and uncompensated drop in ruminal pH. As ruminal pH drops, ruminal lactic acid concentrations rise.[32] This cascade of often fatal consequences begins when ruminal pH drops below about 5.0.

Cows which have not been adapted to high grain diets are particularly susceptible to acute ruminal acidosis,[34] probably because they have not developed a viable population of lactic acid utilizing bacteria and because their ruminal papillae may be short and unable to absorb large quantities of volatile fatty acids (VFA).[13] Re-introducing a high grain diet to adapted

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, weights	8 Holstein steers, ~1000 lbs.	8 Holstein cows, ~1400 lbs.
Stage of feeding or lactation	Compensatory gain	Early lactation
Study design	2 x 2 crossover design	4 x 4 Latin square design
Average daily ruminal pH[c]	5.99	5.90
Forage in diet, %	26.3	52.9
Daily dry matter intake, lbs	29.1	47.1
Neutral detergent fiber, %	20.0	28.9
Non-fiber carbohydrates,[d] %	58.3	36.9
Non-fiber carbohydrates, lbs/day	17.0	17.4

[a]Data adapted from Prentice, Schaefer, and Oetzel.[33]
[b]Data adapted from Oetzel and Nordlund.[30]
[c]Ruminal pH was measured once per minute by indwelling ruminal electrode and averaged daily for each animal.
[d]Non-fiber carbohydrates (NFC), calculated as 100 – % crude protein - % neutral detergent fiber - % ether extract - % ash.

cattle after a period of feed deprivation may also trigger acute ruminal acidosis.[18,34] Researchers are able to induce acute ruminal acidosis by withholding feed for 12 to 24 hours and then allowing access to the same diet that the animal was previously receiving.[32]

The pathophysiological progression during acute ruminal acidosis includes high concentrations of ruminal lactic acid, peracute rumenitis, ruminal hyperosmolality, dehydration and systemic acidemia.[32,34] Clinical signs include complete anorexia, abdominal pain, tachycardia, tachypnea, diarrhea, lethargy, staggering, recumbency and death. Specific treatment protocols for acute ruminal acidosis are described in detail elsewhere.[18,34,35] Cows which survive the initial systemic effects of acute ruminal acidosis may later succumb to complications from severe mycotic or bacterial rumenitis.[34]

SARA is defined as periods of moderately depressed ruminal pH (about 5.5 to 5.0) that are between acute and chronic in duration.[17,26] Lactic acid does not consistently accumulate in the ruminal fluid of dairy cattle affected with SARA.[31] The depression of ruminal pH in dairy cattle with SARA is apparently due to the total accumulation of VFA alone and is not due to lactic acid accumulation.[31] Beef feedlot data support this conclusion.[7]

Defining the clinical syndrome that results from low but compensated ruminal pH as subacute follows the classification scheme originally proposed by Radostits *et al.*[34] Other authors[18,32,35] define this condition as either "chronic" or "subclinical" ruminal acidosis. In dairy cattle it appears inappropriate to define this condition as chronic, because the bouts of low ruminal pH are probably limited to short episodes–somewhere between calving and peak intake at about three to four months post-calving. The risk for SARA is very low outside of these periods in a dairy cow's lactation cycle. In contrast, beef feedlot cattle might be chronically exposed to ruminal pH in the range of 5.0 to 5.5 from the start of the feeding period until the time they are slaughtered. Moreover, inappropriate to define SARA as subclinical, because affected cattle do exhibit specific clinical signs. Unfortunately, the onset of many of these clinical signs is delayed for weeks to months after the time of the low ruminal pH insult.

The most consistent and immediate clinical sign of SARA is depressed feed intake.[32] This is a normal, physiological effort by the cow to restore her ruminal pH to 5.5 or greater by reducing the supply of carbohydrates available for fermentation in the rumen. Several mechanisms are apparently responsible for the feed intake reduction observed with low ruminal pH: 1) increased ruminal VFA concentrations cause ruminal stasis and impair intake.[18] 2) low ruminal pH may also be associated with increased osmolality of the ruminal contents, which in turn inhibits feed intake.[12] 3) inflammation of the ruminal epithelium (rumenitis) could also play a role in depressing feed intake following ruminal acidosis.

While feed intake variation in beef feedlots has been associated with ruminal acidosis[11] sporadic depression in dry matter intake in individual dairy cows is rarely observed because dry matter intake is not intensively monitored on commercial dairy farms. Additional research is needed to determine the nature of feed intake variation that may be caused by SARA in dairy herds.

Drops in milk production (in herds with daily milk weigh monitoring systems) or concentrate intake (in component-fed herds) may be observed in dairy herds with SARA. Other clinical signs associated with SARA include moderate ruminal distention, a doughy texture of the ruminal contents, and weak ruminal contractions.[34]

The low ruminal pH of SARA reduces the number of species of bacteria in the rumen, although the metabolic activity of the remaining bacteria is very high.[18] Protozoal populations are also limited as ruminal pH approaches 5.0. When fewer species of bacteria and protozoa are present, the ruminal microflora are less stable and less able to maintain normal ruminal pH during periods of sudden dietary changes.[18] Thus, preexisting SARA could increase the risk for acute ruminal acidosis in the event of accidental ingestion of excessive amounts of grain.

Although the low-ruminal-pH insult of SARA sets in motion a pathophysiological cascade of events, clinical signs are delayed in onset. Beginning with rumenitis, bacteria from the inflamed ruminal epithelium may colonize the papillae and leak into portal circulation. These bacteria may cause liver abscesses, with accompanying peritonitis around the abscess site. 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.

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.

Caudal vena cava syndrome can cause hemoptysis and peracute deaths due to massive pulmonary hemorrhage in cows affected with SARA.[26] In these cases, septic emboli from liver abscesses lead to lung infections which ultimately invade pulmonary vessels and cause their rupture.[34,35]

Clinical Presentation of Subacute Ruminal Acidosis in a Dairy Herd

SARA is diagnosed and prevented on a herd basis rather than on an individual cow basis. Clinical signs in dairy herds affected with SARA may include low or fluctuating dry matter intakes, low body condition scores, diarrhea, nosebleeds, unexplained deaths due to chronic inflammatory diseases and unexplained high cull rates due to vague health problems. Milk-fat depression and poor milk production in the second-and-greater-lactation cows relative to the first-lactation cows also may be seen. None of these signs alone are diagnostic for SARA; however, considered together they form the basis for a presumptive diagnosis of SARA in a herd.

Dry matter intake depression or fluctuations may be caused by SARA, but are rarely recorded in sufficient detail to be helpful in making a diagnosis. Low body condition scores in the face of adequate, or even high, total dietary energy intake may be caused by rumenitis and other chronic inflammatory conditions secondary to SARA.

Some cows with SARA exhibit a transient diarrhea that is light-colored and has a characteristic sweet – sour smell.[34] This clinical sign probably indicates extensive post-ruminal fermentation of carbohydrates. Whether this occurs with SARA depends on the amount of undigested carbohydrates passed out of the rumen. A qualitative evaluation of the manure in groups of cows is a useful part of a herd work-up for SARA.

A small portion of cows affected with SARA may exhibit sporadic, bilateral nosebleeds.[26] These occur secondarily to bacterial pneumonia or caudal vena cava syndrome, both of which can be traced back to SARA-induced rumenitis. It is helpful to include questions about the incidence of these problems as part of a herd work-up for SARA.

A clinical complaint of poor immune function (based on poor response to therapy for apparently routine bacterial infections) is often made in dairy herds ultimately diagnosed with SARA.[26] In theory, this observation could be explained by consumption of the immune system with the chronic inflammatory conditions caused by SARA, leaving the cow more vulnerable to other infectious agents. Research data are needed to support this observation.

A high unexpained death loss could be caused by a high prevalence of SARA in the herd.[26] Average death loss of adult cows in dairy herds is about 5% per year; herds that I definitively diagnose with SARA typically have death losses of 10% to 15% per year. SARA causes numerous chronic inflammatory conditions that are difficult to diagnose ante-mortem and can ultimately lead to death.

Milk-fat depression may occur in herds with a high prevalence of SARA. However, lack of milk-fat depression does not imply that the herd is free of SARA. It is true that milk-fat percentage is typically depressed in individual cows during bouts of low ruminal pH, but milk-fat content is not measured on a daily basis in individual cows in commercial dairy herds. Monthly evaluation of milk-fat tests in individual cows is of limited value in diagnosing SARA, because the episodes of depressed ruminal pH are sporadic. And because only a few cows may be affected at any given time, the effect of individual cows' milk-fat depression on the whole herd's milk-fat test is minimal.[26] Severe milk-fat depression that affects the herd's average milk-fat test can indicate SARA, although it is not specific for this condition. Other causes of herd milk-fat depression include excessive feeding of unsaturated fats, over-dosing ionophores, and low days in milk.

Milk production in older cows in herds with a high prevalence of SARA typically lags behind that of younger cows because of the chronic, cumulative health effects of SARA. This production effect can be evaluated by comparing the average mature equivalent (ME) milk production of the first-lactation cows to the average ME milk production of the older cows. In herds with good nutritional management and stable genetic improvement programs, first-lactation ME milk production is about the same to about 500 lbs higher than the older cows. Herds with a high prevalence of SARA typically have ME milk production in the first-lactation cows that is 1500 to 4000 lbs greater than for the second and greater lactation cows.

Peak milk ratios (average first-lactation peaks divided by the average peak milk of the older cows) give information similar to the ME milk production relationship. Peak milk ratios above about 78% indicate relatively poor milk production by the older cows. Other important causes of relatively poor milk production in the older cows include inadequate nutrient density to support older cows' peaks, failure to restore body condition after the first lactation, errors in dry cow management that affect only the older cows, and other nutritional management problems that affect only the older cows.

Methods of Measuring Ruminal pH in Dairy Herds

When considered individually, none of the herd-based clinical signs mentioned above are specific enough to make a diagnosis of SARA. And while combination of these herd problems makes more convincing evidence for SARA, they still do not constitute a definitive diagnosis. Definitive diagnosis of SARA requires documentation of low ruminal pH in a group of cows from the herd.

Ruminal pH can be measured on ruminal fluid collected by oral tube, through a ruminal cannula, or by

ruminal puncture with a needle (rumenocentesis). Orally collected samples of ruminal fluid are easily contaminated with varying amounts of saliva,[26,29] and ruminal cannulation of large numbers of cows in commercial dairy herds is impractical. This leaves rumenocentesis as the only practical method available for assessing ruminal pH in a group of cows. The pH of ruminal fluid collected by rumenocentesis is highly correlated but slightly lower (about .25 to .30 pH units) than ruminal pH collected through a ruminal cannula.[17] The procedure for collecting ruminal fluid by rumenocentesis has been described in detail elsewhere.[17,26]

Site of collection of ruminal fluid from within the rumen has some effect on ruminal pH,[8,21] as does method of collection.[17] Rumenocentesis works well for collecting ruminal fluid in the field because the fluid is collected from the same site in the rumen, and because the sample is not contaminated by feed particles or exposed to variable amounts of air.

A research-based diagnostic scheme for using ruminal pH, based on samples collected by rumenocentesis, has been developed[17] which requires testing 12 or more cows per diet group on the farm. Larger group sizes do not substantially affect the suggested minimum sample size. Only cows from calving to about four months postcalving are at enough risk for SARA to be considered for ruminal pH testing. Results of ruminal pH testing are interpreted according to the proportion of cows below the biological threshold of ruminal pH 5.5. Average ruminal pH of the group tested is not very important clinically, and is not evaluated.

If more than four of the 12 cows tested have ruminal pH $\leq$ 5.5, then the group is definitively classified as having SARA. If two to four of the 12 cows tested have ruminal pH $\leq$ 5.5 the group is classified as borderline, and if none or one of the 12 cows tested have ruminal pH $\leq$ 5.5 the group is definitively classified as negative for SARA. These classifications are based on a 75% confidence interval that the measured proportion does not overlap with the alarm level of 25% of the cows tested with low ruminal pH.

The sample size of 12 cows is adequate for groups with high (>30%) or low (<15%) prevalences of low ruminal pH.[17] In herds with borderline results, additional cows must be tested to make a definitive positive or negative diagnosis.[17] A presumptive diagnosis can be strengthened by combining ruminal pH with other herd-based clinical signs, even if the results of the ruminal pH testing are borderline. In many herds, however, the prevalence of SARA is best described as clinically borderline, and the herd should not be forced into a negative or positive classification.

The main disadvantage of rumenocentesis for herd-based diagnosis of SARA is that it requires considerable time and labor. Adequate restraint for rumenocentesis requires one person at the head of the cow with a nose twitch,[27] one at the rear applying a tail jack, and a third person to collect the fluid. Negative production or health effects have not been observed with a single ruminal puncture in adequately restrained cows.[17] However, multiple punctures of the same cow could lead to complications.[38]

Diagnostic Approach to SARA in Dairy Herds

Diagnosis of SARA in a dairy herd starts with a herd history of problems related to SARA, such as those described above. Each as a problem should be characterized in detail before considering SARA as a likely cause. For example, milk production problems need to be characterized over time (I typically evaluate the last two years of milk production data) and ME milk production averaged by lactation group. The herd should be evaluated for current prevalence of lameness, and the predominant cause(s) of lameness confirmed, either by an evaluation of the hoof trimmer's records or by examining currently affected cows. High cull rates should be investigated by reason for removal and days in milk at culling. High death losses should be investigated for cause of death, post-mortem or other diagnostic information, and days in milk at death. Milk-fat depression should be evaluated over time (again, I evaluate the past two years of herd performance), lactation, days in milk and, if applicable, by production or dietary group. If the complaint or herd observation reveals thin cows despite high-energy diets, then a representative portion of the herd should be body condition scored. Body condition scores should then be plotted by days in milk and evaluated against expected body condition curves. If the information gathered to this point indicates the herd could have a high prevalence of SARA, the diagnosis should be confirmed by evaluation of ruminal pH.

Once a diagnosis of SARA has been established, 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 intake, dairy cattle cannot tolerate diets as 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 effects of feeding excessive 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.

Dairy nutritionists have carefully defined fiber requirements for dairy cattle in terms of acid detergent fiber (ADF) and neutral detergent fiber (NDF).[24] Nutritionists often go beyond the measures of carbohydrate nutrition defined by the National Research Council to include nutrients such as non-fiber carbohydrates (NFC), starch, effective NDF (eNDF), physically effective NDF (peNDF),[21] and long fiber particles.[28] Each looks at a slightly different aspect of carbohydrate nutrition. Evaluating the dietary content for each nutrient is an important first step in determining the cause of SARA in a dairy herd, and 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 requires a meticulous investigation of how feed is delivered to the cows, accurate weights of feed delivered, and updated nutrient analyses of feeds delivered, particularly dry matter content of the fermented feed ingredients. Careful bunk sampling of total mixed rations (TMR) may uncover errors in feed composition or feed delivery.

Total intake of rapidly fermentable carbohydrates is probably more important than percentage of carbohydrates in the diet.[30] Herds or groups within herds with higher dry matter intakes are at inherently higher risk for SARA and may need a more conservative amount of carbohydrate nutrition than other herds or groups.

As genetic progress drives individual cows to eat more dry matter and to produce more milk, their risk for SARA will be increased, making it even more difficult to prevent SARA in high-producing cows.

Dairy herd diets 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.[26] 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 of thumb, cows should receive no more than 8 to 12 lbs of dry matter from grain in the first week after calving. Grain feeding should then increase by about .25 to .50 lbs/cow/day until peak grain feeding is reached at six to eight weeks post-calving. Maximal protein feeding can be reached by about three weeks post-calving.

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 than 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.

Dairy cattle groups are commonly fed for *ad libitum* intake (typically a 5% daily feed refusal) 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. Dairy cattle feeders face more of a challenge, as they must 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 pre- and post-fresh cow groups because they have rapid cow turnover, and because individual cows have rapidly changing dry matter intakes during these periods.

Inadequate ruminal buffering

Ruminant animals have a highly developed system for buffering 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.[15] Ruminal buffering has two aspects – dietary and endogenous buffering.

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.[5,36] Optimal DCAD for early lactation diets is about +400 mEq/kg of (Na +K) – (Cl + S).[5] 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. Saliva is secreted during chewing activity (eating and rumination) in response to the amount of physical fiber present, so time spent chewing is a rough estimate of saliva production.[1] Saliva buffers ruminal pH because it is rich in sodium, potassium, bicarbonates and phosphates.[40]

Coarse, fibrous feeds contain more effective fiber and stimulate more saliva production during eating than do finely ground feeds or fresh pasture.[3] Coarse, fibrous feeds also make up the mat layer of the rumen, which is the stimulus for rumination. Fiber particles longer than about 1.5 inches are most likely to contribute to mat layer formation. Rumination promotes much chewing activity–and therefore secretion of large amounts of saliva into the rumen– noticeably increasing ruminal pH.[1]

The ability of a diet and feeding system to promote maximal amounts of ruminal buffering should be considered when evaluating a herd diagnosed with SARA. Wet chemistry analysis of a carefully-collected TMR bunk sample can be particularly effective in determining the actual DCAD of the diet delivered to the lactation cows. Diets with measured DCAD values below about +275 to +400 mEq/kg of (Na + K) – (Cl + S) should be supplemented with additional buffers to provide more Na or K relative to Cl and S.

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.[20,28] 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.[19,39] Increasing chemical fiber content of the diet may compensate for short particle length.[4]

Diets with excessive long forage particles (more than about 15%) can paradoxically increase the risk for SARA 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, after feedings, is low in physically effective fiber. This makes diet consumed later in the feeding period 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 30 inches 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 lower ruminal pH).[13] Beef feedlots recognize the importance of gradually introducing steers to higher grain diets.[34]

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 dry period feeding had no effect on early lactation ruminal pH.[16] Ruminal pH was unexpectedly lower in cows at 106 average days in milk compared to cows at 15 average days in milk.[16] These results suggest that high dry matter intake is a greater risk factor for SARA than ruminal adaptation problems in dairy herds. Moreover, a controlled study in component-fed cows found no positive effect from increased grain feeding during the dry period on either early lactation ruminal pH or dry matter intake.[2] These results suggest that the practical impacts of ruminal adaptation may be small in dairy herds, particularly when cows are fed a TMR after calving.

Prevention of Subacute Ruminal Acidosis in Dairy Herds

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. Nutritional interventions that might prevent SARA without limiting grain feeding are highly desirable, and several of these approaches are summarized as follows.

Enhancing Ruminal Lactate Utilizers

An important aspect of a stable rumen environment is maintaining a balance between lactate production and lactate utilization by bacteria that convert lactate to less-dangerous VFA. Enhancing ruminal lactate utilizers reduces the risk for ruminal acidosis, particularly the acute form of ruminal acidosis. Supplementation with specific yeast strains may enhance lactate utilization within the rumen under certain dietary conditions.[12]

Selenomonas ruminantium is one of the bacteria that convert ruminal lactate to VFA. *S. ruminantium* is apparently stimulated by malate to utilize lactate .[22] Supplementing diets with malate as a feed additive may be cost-prohibitive; however, incorporation of forage varieties high in malate may allow for economical inclusion of malate in dairy diets.[9] Stage of maturity and variety affect malate concentrations in alfalfa.[9]

Preconditioning Microbes to Handle Lactate

Adding lactate to the diet or using feed ingredients high in lactate may improve the ability of the rumen to adapt to sudden increases in lactate production.[32] Direct-fed microbials might also be used to provide a steady source of rumen lactate. A mixture of direct-fed microbials added to the rumen of dairy cows at the 1 x 10^5 dose increased corn digestibility and increased ruminal pH, compared to higher doses of microbials.[25]

Supplementation with Ionophores

Feeding ionophores reduces ruminal lactate production, an effect apparently caused by inhibition of lactate-producing bacteria, competitive enhancement of lactate utilizers, and possibly by reducing meal size.[32] Ionophore products are approved in the USA for dairy replacement heifers, but not for lactating dairy cows.

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