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Review

Understanding and preventing subacute ruminal acidosis in dairy herds: A review☆

K. Marie Krause, Garrett R. Oetzel *

Food Animal Production Medicine Section, Department of Medical Sciences, School of Veterinary Medicine, University of Wisconsin-Madison, 2015 Linden Drive, Madison, WI 53706-1102, USA

Abstract

Feeding diets high in grain and other highly fermentable carbohydrates to dairy cows increases milk production, but also increases the risk of subacute ruminal acidosis (SARA). SARA is defined as periods of moderately depressed ruminal pH, from about 5.5 to 5.0. SARA may be associated with laminitis and other health problems resulting in decreased production.

Although ruminal pH varies considerably within a day, cows possess a highly developed system to maintain ruminal pH within a physiological range. However, if the acid production from fermentation is more than the system can buffer, ruminal pH compensation fails and ruminal pH may drop drastically.

The risk of developing SARA can be reduced by adopting a feeding regime, which balances ruminal buffering with the production of volatile fatty acids from fermentation of carbohydrates. This can be achieved by providing adequate dietary fibre containing sufficient long particles. However, excessive amounts of long particles might lead to sorting and ultimately increase the risk of SARA. The level of fibre and long particles needed to maintain rumen health depends on the fibre source used. SARA is also dependent on the grain source fed and the degree of grain processing. Feeding highly fermentable grain increases the requirement for fibre.

Feeding diet components separately appears to increase the risk for SARA compared to feeding a total mixed ration. Also, management practices that cause cows to eat fewer, larger, or irregular meals may be associated with increased incidence of SARA. Important management factors include feed

Abbreviations: DCAD, dietary cation–anion difference; DM, dry matter; eNDF, effective NDF; NDF, neutral detergent fibre; NFC, non-fibre carbohydrates; peNDF, physically effective NDF; SARA, subacute ruminal acidosis; TMR, total mixed ration; VFA, volatile fatty acids

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* Corresponding author. Tel.: +1 608 265 5476; fax: +1 608 265 8020.
E-mail address: groetzel@wisc.edu (G.R. Oetzel).

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access time, consistency of feeding schedule, and available bunk space. When dairy cows are fed for high production, good management practices are important even when dietary factors are optimal.

Keywords: Subacute ruminal acidosis; Dairy cattle; Nutritional management

Contents

1. Introduction

Ruminal acidosis is the consequence of feeding high grain diets to ruminant animals, who are adapted to digest and metabolise predominantly forage diets. Feeding diets that are progressively higher in grain tends to increase milk production, even in diets containing up to 0.75 concentrates (Kennelly et al., 1999). However, short-term gains in milk production from feeding high grain diets are often substantially or completely negated by long-term compromises in cow health.

Compromises in dairy cow health due to ruminal acidosis 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 ruminal acidosis has been recognised as a major risk factor for laminitis (Nocek, 1997; NRC, 2001). Lameness (along with secondary reproductive failure and low milk production) is commonly the most important cause of premature and involuntary culling 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., U.S.) are probably at the highest risk for ruminal acidosis. Attempts to lower milk fat percentage to meet quota limitations can also be associated

with ruminal acidosis. The economics of dairy production under these circumstances favour heavier grain feeding. However, producers, nutritionists, and veterinarians might be ignorant of the long-term health costs of ruminal acidosis and therefore may be reluctant to decrease grain feeding.

Areas of the world with relatively more expensive grains and/or milk production quotas probably experience less ruminal acidosis than the U.S. However, bouts of ruminal acidosis are always possible whenever grains or very high quality forage are consumed by dairy cows.

2. Types of ruminal acidosis

Acute and subacute ruminal acidosis share a similar aetiology but are very different clinical diseases. The general definitions used in beef feedlot cattle (Owens et al., 1998) for these two disorders have been applied to dairy cattle (Nordlund et al., 1995; Garrett et al., 1999). 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 (Owens et al., 1998). 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 (Radostits et al., 1994), probably because they have not developed a viable population of lactic acid utilising bacteria and because their ruminal papillae may be short and unable to absorb large quantities of volatile fatty acids (Dirksen et al., 1985). Re-introducing a high grain diet to adapted cattle after a period of feed deprivation may also trigger acute ruminal acidosis (Garry, 2002; Radostits et al., 1994). Researchers are able to induce acute ruminal acidosis by withholding feed for 12–24 h and then allowing access to the same diet that the animal was previously receiving (Owens et al., 1998).

The pathophysiological progression during acute ruminal acidosis includes high concentrations of ruminal lactic acid, peracute rumenitis, ruminal hyperosmolality, dehydration and systemic acidemia (Owens et al., 1998; Radostits et al., 1994). Clinical signs include complete anorexia, abdominal pain, tachycardia, tachypnea, diarrhoea, lethargy, staggering, recumbency and death. Specific treatment protocols for acute ruminal acidosis are described in detail elsewhere (Garry, 2002; Radostits et al., 1994; Rebhun, 1995). Cows that survive the initial systemic effects of acute ruminal acidosis may later succumb to complications from severe mycotic or bacterial rumenitis (Radostits et al., 1994).

Subacute ruminal acidosis (SARA) is defined as periods of moderately depressed ruminal pH (about 5.5–5.0) that are between acute and chronic in duration (Garrett et al., 1999; Nordlund et al., 1995). Lactic acid does not consistently accumulate in the ruminal fluid of dairy cattle affected with SARA (Oetzel et al., 1999); however, transient spikes of ruminal lactate up to 20 mM can be discovered if ruminal lactate concentrations are measured frequently during the day (Kennelly et al., 1999). The depression of ruminal pH in dairy cattle with SARA is apparently due to the total accumulation of volatile fatty acids (VFA) alone and is not due to lactic acid accumulation (Oetzel et al., 1999). Beef feedlot data support this conclusion (Britton and Stock, 1987).

Defining the clinical syndrome that results from low but compensated ruminal pH as subacute follows the classification scheme originally proposed by Radostits et al. (1994).

Other authors (Garry, 2002; Owens et al., 1998; Rebhun, 1995) define this condition as either chronic or subclinical ruminal acidosis. It is not appropriate to describe this condition as chronic in dairy cattle, because the bouts of low ruminal pH are probably limited to short episodes somewhere between calving and about 5 months post-calving (Oetzel, 2000). The risk for SARA is very low outside of these time periods in a dairy cow's lactation cycle. In contrast, beef feedlot cattle could be chronically exposed to ruminal pH in the range of 5.0–5.5 from the start of the feeding period until the time they are slaughtered. It is also not appropriate to define SARA as subclinical, because affected cattle do exhibit specific clinical signs (Oetzel, 2000). Unfortunately, the onset of many of these clinical signs is delayed for weeks to months after the time of the low ruminal pH insult.

3. Physiology of ruminal pH

Ruminal pH drops below physiological levels when ruminants consume excessive amounts of rapidly fermentable (non-fibre) 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 fibre compared to beef feedlot cattle, this is offset by their much higher dry matter (DM) intakes. De Brabander et al. (1999) found that increasing DM intake was associated with a higher requirement for physical structure in the diet. Total consumption of non-fibre carbohydrates (NFC) is often similar between dairy cows and feedlot cattle. 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 DM intake and the density of NFC in the diet. High intakes in dairy cattle are associated with lower ruminal pH (Oetzel and Nordlund, 1998). This suggests that ruminal acidosis will become an even more common problem as genetic progress and better feeding management allow cows to consume more feed.

Clinical data from our herd investigations also supports the concept that total DM intake is a major determinant of ruminal pH. With increasing days in milk, we have observed increasing risk for low ruminal pH (Fig. 1). This pattern parallels the normal pattern of increasing DM intake until about the 3rd month of lactation.

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 0.5–1.0 pH unit within a 24 h period are common (Dado and Allen, 1993; Nocek et al., 2002). This represents a five- to 10-fold change in hydrogen ion concentration in the rumen. A typical pattern of ruminal pH variation during the day is presented in Fig. 2.

Increasing the frequency of feeding (e.g., six times *versus* twice daily) might decrease the variation in post-feeding ruminal pH, but can also lead to increased DM intake and ultimately result in lower mean ruminal pH (Oetzel and Nordlund, 1998). A 24 h tracing of ruminal pH in a cow fed twice *versus* six times daily is shown in Fig. 3.

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

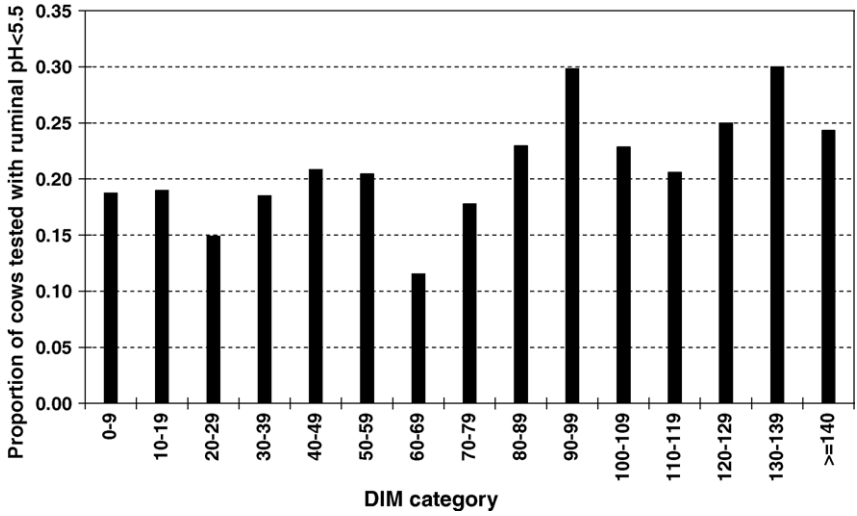

Fig. 1. Risk of low ruminal pH (<5.5) by days in milk (DIM) from 662 cows in 55 herds. Samples were collected by rumenocentesis 6–10 h post-feeding in commercial dairy herds in Wisconsin, USA, between 1996 and 2003 (Oetzel et al., previously unpublished data). Nadir usually occurs between 6 and 8 h post-feeding in TMR-fed herds *vs.* 2–4 h post-feeding in component-fed herds.

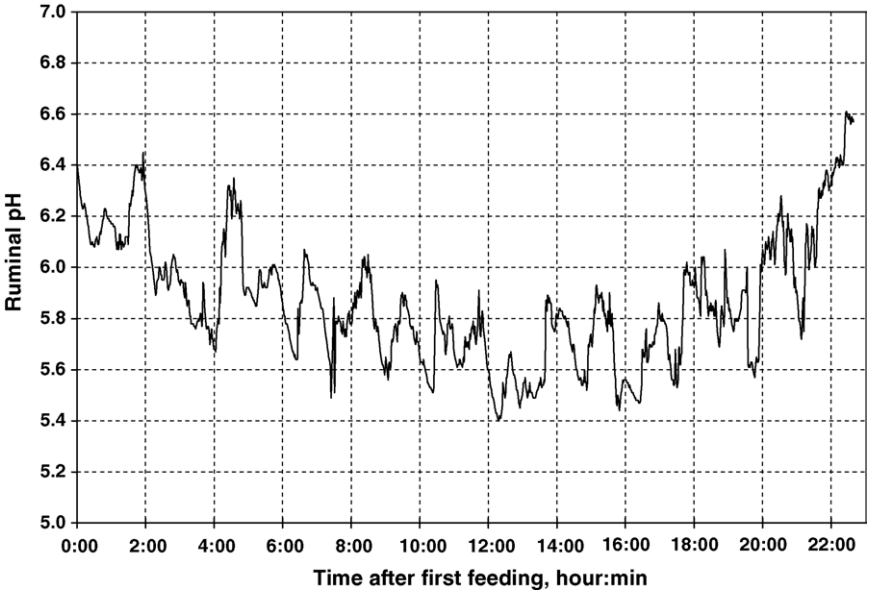

Fig. 2. Post-feeding variations in ruminal pH over a period of 24 h. The cow was fed dry, cracked corn grain and finely chopped alfalfa silage twice daily (12 h interval). Dry matter intake of the current day was 22.7 kg. Average ruminal pH for that day was 5.87 with a standard deviation of 0.25 and a range from 5.40 to 6.61 (previously unpublished data from Krause and Combs, 2003).

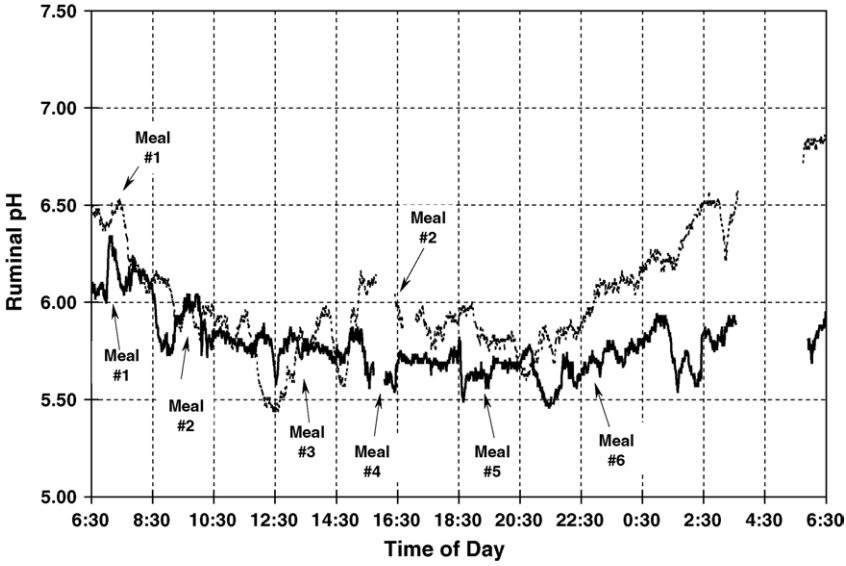

Fig. 3. Ruminal pH over a 24 h period of a cow fed twice daily (dotted line) and the same cow fed six times daily (solid line). Mean ruminal pH was 6.02 when fed twice daily and 5.78 when fed six times daily. DM intake increased from 17.4 to 21.3 kg DM/day when increasing number of times fed (previously unpublished data from Oetzel and Nordlund, 1998).

electrode (Figs. 2 and 3) provides the most information possible about post-feeding variations in ruminal pH. Woodford and Murphy (1988) reported that different diets might result in similar mean ruminal pH but in different area under a certain pH value. Also, Krause et al. (2002) found that dietary factors such as forage particle size and level of ruminally fermentable carbohydrates affected area below pH 5.8 to a greater degree than mean ruminal pH. This emphasises the importance of considering not only mean ruminal pH, but also post-feeding variations when assessing the effect of diets on rumen health. The use of continuous monitoring of ruminal pH can help us answer the question whether mean ruminal pH, the lowest pH value, or the time span of the period during which rumen pH is below a threshold value is decisive to the significance of SARA.

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 5.5–7.0. Low ruminal pH may be associated with increased osmolality of the ruminal contents, which in turn inhibits feed intake (Carter and Grovum, 1990). In beef cattle, depressed DM intake becomes especially evident if ruminal pH falls below about 5.6 (Fulton et al., 1979). Inflammation of the ruminal epithelium (rumenitis) could also play a role in depressing feed intake following ruminal acidosis. The precise pH thresholds for subtle reduction or variation of intake in dairy cattle are not known.

Besides carefully regulating their feed intake, ruminant animals stabilise ruminal pH by 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 (Firkins, 1997). Cows produce a large amount of buffers via their saliva. Saliva buffers ruminal pH because it is rich in sodium, potassium, bicarbonates and phosphates (Van Soest, 1994), contributing approximately half of the bicarbonate entering the rumen (Owens et al., 1998). Unfortunately, saliva production is not triggered by declining ruminal pH, but rather is determined by the amount of time the cow spends eating, ruminating and resting (Maekawa et al., 2002).

The ability of the rumen to rapidly absorb organic acids also contributes to the stability of ruminal pH. Absorption of VFA from the rumen occurs passively across the ruminal wall (Bergman, 1990). This passive absorption is enhanced by papillae that project away from the rumen wall. These papillae provide a very large surface area for VFA absorption. Ruminal papillae increase in length when cattle fed higher grain diets (Dirksen et al., 1985); this presumably increases the surface area and absorptive capacity, which in turn protects the animal from acid accumulation in the rumen. If the absorptive capacity of these cells is impaired (e.g., chronic rumenitis with fibrosis), it becomes much more difficult for the animal to maintain a stable ruminal pH.

Mean ruminal pH values might not be dramatically affected by large dietary changes, but the lowest (nadir) pH values are often greatly affected by diet. An example is the study by Kennelly et al. (1999), who reported mean ruminal pH values of 6.31 and 6.15 (not statistically different) for cows fed diets containing 0.50 and 0.75 concentrate, respectively, whereas nadir pH appeared to be very different (5.9 *versus* 5.5) for 0.50 and 0.75 concentrate diets, respectively. Another example is Krause and Combs (2003), who found that partially replacing alfalfa silage with corn silage did not affect mean ruminal pH, but did decrease nadir pH significantly. These responses are consistent with the nature of regulation of ruminal pH 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.

Cattle develop SARA as ruminal pH drops below the physiological threshold of about 5.5. Fortunately, ruminal VFA have a pK_a of about 4.9 and therefore rapidly shift toward the undissociated (protonated) form at ruminal pH below 5.5. This shift removes a free hydrogen ion from the ruminal fluid. It also facilitates VFA absorption across the ruminal epithelium, because VFA are passively absorbed only in the undissociated form.

Unfortunately, gains in VFA absorption at ruminal pH below 5.5 can be offset by lactate production. At high growth rates (high levels of starch and sugars) *Streptococcus bovis* begins to ferment glucose to lactate instead of VFA, which further decreases ruminal pH and creates a niche for lactobacilli that produce even more lactate (Russell and Hino, 1985). This is a dangerous situation, because lactate has a much lower pK_a than VFA (3.9 *versus* 4.8, Fig. 4). At a ruminal pH of 5.0, for example, lactate is 5.2 times less dissociated than VFA. As a result, lactate remains in the rumen and contributes to the downward spiral in ruminal pH.

Additional adaptive responses are invoked when ruminal pH drops and lactate production begins. Lactate-utilising bacteria, such as *Megasphaera elsdenii* and *Selenomonas*

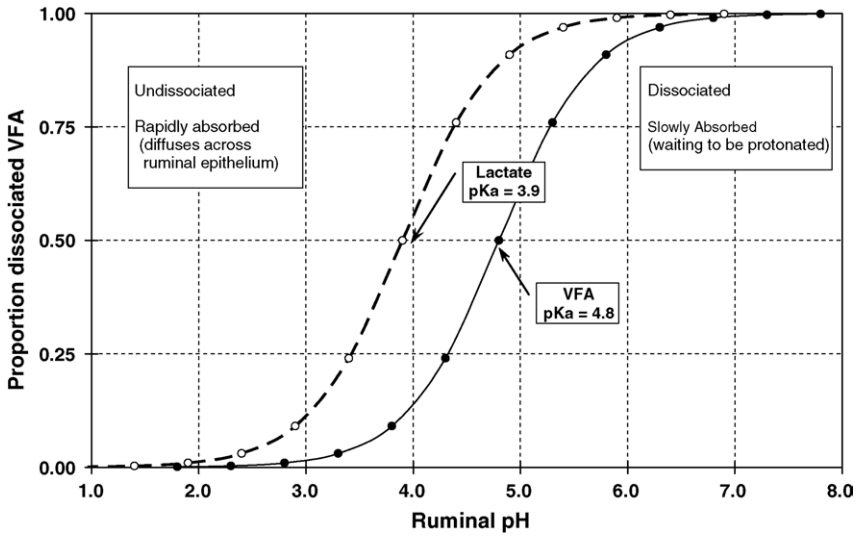

Fig. 4. Titration curve for the major ruminal VFA (acetate, propionate and butyrate, solid line) and lactate (dashed line).

ruminantium, start metabolising lactate and begin to proliferate (Goad et al., 1998). These beneficial bacteria convert lactate to other VFA, which are then easily protonated and absorbed. The majority of the lactate produced can be metabolised by these bacteria (Counotte and Prins, 1981); however, as pH decreases towards 5.0 the growth of these bacteria is inhibited, and lactate production exceeds utilisation (Russell and Allen, 1983). Also, the turnover time of lactate utilisers is much slower than for *S. bovis* (Mackie and Gilchrist, 1979). Thus, lactate conversion in the rumen may not be invoked quickly enough to fully stabilise ruminal pH.

Periods of very high ruminal pH, as during feed deprivation, may inhibit the growth rate of certain populations of lactate utilisers, which are sensitive to higher ruminal pH (Mackie and Gilchrist, 1979). This leaves the rumen ecosystem more susceptible to severe ruminal acidosis. Besides disrupting microbial balance, feed deprivation also 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 may be more important risk factors for SARA than is diet formulation itself. An example of severe ruminal pH depression following a period of feed deprivation and re-feeding is presented in Fig. 5.

Low ruminal pH during SARA reduces the number of species of bacteria in the rumen, although the metabolic activity of the bacteria that remain is very high (Garry, 2002). Protozoal populations also do not survive extended exposure to pH below 5.5 (Quinn et al., 1962). 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 (Garry, 2002). Thus, pre-existing SARA could increase the risk for acute ruminal acidosis in the event of accidental ingestion of excessive amounts of grain.

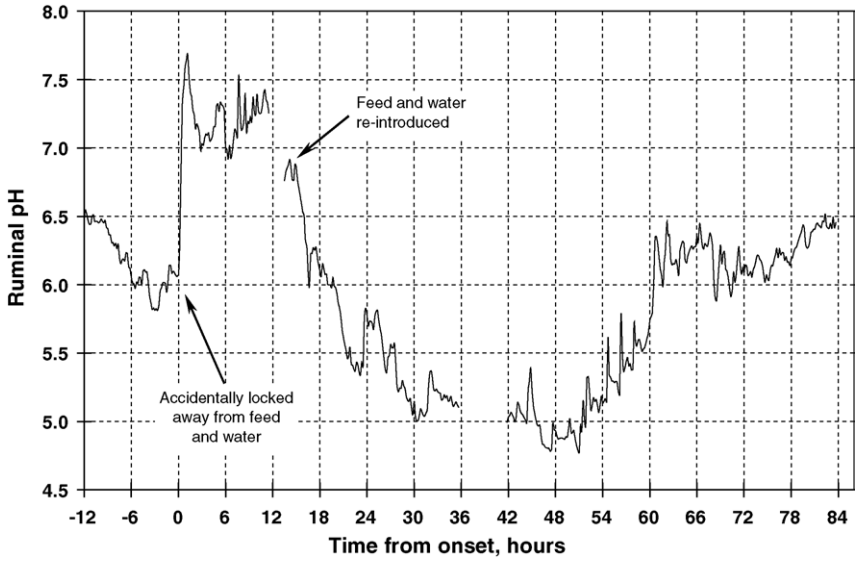

Fig. 5. Ruminal pH following a period of feed deprivation and re-feeding in a Holstein steer (previously unpublished data from Prentice et al., 2000).

4. Pathophysiology of ruminal acidosis in dairy cattle

Unlike abomasal cells, ruminal epithelial cells are not protected by mucus and are vulnerable to the chemical damage by acids. Thus, low ruminal pH can lead to rumenitis and eventually to ruminal parakeratosis, erosion, and ulceration of the ruminal epithelium (Garry, 2002). Rumenitis is the fundamental lesion of SARA and initiates chronic health problems. Once the ruminal epithelium is inflamed, bacteria may colonise 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 colonise the lungs, heart valves, kidneys, or joints (Nordlund et al., 1995; Nocek, 1997). The resulting pneumonia, endocarditis, pyelonephritis and arthritis 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.

There are few research reports that directly attribute specific health problems to low ruminal pH. In one study (Prentice, 2000), we were able to document a rise in serum haptoglobins (acute-phase inflammatory proteins) and an increase in the prevalence of ruminal biopsy samples with histological evidence of rumenitis when Holstein steers were fed to a low target ruminal pH.

Cows affected with SARA may develop caudal vena cava syndrome, which presents clinically as hemoptysis and peracute deaths due to massive pulmonary haemorrhage (Nordlund et al., 1995). In these cases, septic emboli from liver abscesses cause foci of pulmonary infection that ultimately invade pulmonary vessels and cause their rupture (Radostits et al., 1994; Rebhun, 1995).

Subacute ruminal acidosis may also be associated with laminitis and subsequent hoof overgrowth, sole abscesses, and sole ulcers (Nocek, 1997). These foot problems generally do not appear until weeks or months after the bout of ruminal acidosis that caused them. Laminitis has been associated with systemic metabolic insults (Nocek, 1997), but the precise mechanism by which SARA increases the risk for laminitis has not been characterised. New information from equine studies has shown that an exotoxin released from *S. bovis* may activate metalloproteinase enzymes and lead to the separation of equine lamellar explants (Mungall et al., 2001). These results indicate a link between damage to the integrity of the hoof and *S. bovis*, which might proliferate during bouts of SARA. This theory has not been evaluated in cattle.

5. Economic importance of subacute ruminal acidosis

Very few studies have examined the epidemiology of acute and subacute ruminal acidosis in dairy cows. Gröhn and Bruss (1990) evaluated the incidence of acute ruminal acidosis in 61,124 Finnish Ayrshire cows. The data were based on veterinary diagnoses collected from 2 days before calving until the subsequent calving. The incidence of acute ruminal acidosis was 0.3% throughout lactation, but was highest during the 1st month post-calving and relatively non-existent within 3 months. SARA is estimated to cost the U.S. dairy industry between US$ 500 million to US$ 1 billion a year (Donovan, 1997). Stone (1999) calculated US$ 400 to US$ 475 lost income per cow per year due to SARA in a case study of a 500-cow dairy in New York state. This estimate was based on an observed decrease in milk production of 3 kg/cow/day and decreased milk fat and true protein from 37 to 34 g/kg and 29 to 28 g/kg, respectively. The financial impact of associated disorders, such as lameness and its deleterious effect on reproduction was not estimated, but was probably higher than the cost of lost milk production. Although SARA is commonly expected to negatively affect cow health, there are very few studies investigating this relationship as the primary objective.

6. Nutritional management for prevention of subacute ruminal acidosis

Fermentation acid production in the rumen needs to be balanced with fermentation acid removal and neutralisation in order to achieve optimal ruminal conditions and optimal production. This relationship between fermentation acid production and the requirement for fibre has been reviewed in detail by Allen (1997). When this balance between acid production and acid removal/neutralisation is not achieved the cow will suffer from SARA. Consequently, causes of SARA in dairy herds may be grouped into three major categories: inadequate ruminal buffering caused by inadequate dietary fibre and/or inadequate physical fibre, excessive intake of rapidly fermentable carbohydrates, and inadequate ruminal adaptation to a highly fermentable diet. Although these categories will be discussed separately here, it is important to realise that each of them cannot be viewed alone when investigating the risk of SARA in a dairy herd.

6.1. Inadequate ruminal buffering

Ruminal buffering has two aspects: endogenous and dietary buffering. Endogenous buffers are produced by the cow and secreted into the rumen via the saliva. The formulations of diets based on neutral detergent fibre (NDF) as a percentage of the ration DM has been recommended because of the positive relationship between NDF and rumen fill and the negative relationship between NDF and energy density of the diet (Mertens, 1994). However, dietary fibre level is usually not used alone because ruminal fermentation of fibre is variable (Nocek and Tamminga, 1991), and because physical characteristics of fibre influence ruminal fermentation and utilisation, animal metabolism and milk fat production independently of the amount of chemically measured NDF (Mertens, 1997). In order to account for this, the NRC (2001) recommends that a minimum proportion of the recommended level of dietary NDF come from forage. Others (NRC, 1996; Pitt et al., 1996) have adopted the concept of effective NDF (eNDF) to describe the ability of a feed to replace forage such that milk percentage is maintained. Mertens (1997) introduced the concept of physically effective fibre (peNDF), which is primarily related to particle size of the fibre. Chewing activity is used to determine peNDF. Although guidelines with regard to level of dietary fibre and fibre from forage are available, it is important to realise that the cow's fibre requirement is affected by many interacting components within the diet as well as how the feed is allocated and processed. In Europe a system has been developed which seeks to incorporate both feed characteristics (e.g., fibre content, particle size and acidotic effect) together with feeding management (i.e., number of concentrate meals per day) in order to formulate dairy cow rations for good rumen function (De Brabander et al., 1999).

Corn silage is a popular forage for dairy cows in the U.S., and its use is also becoming more common in Europe and Austral-Asia. However, feeding a large proportion of a lactation diet as corn silage puts cows at higher risk for SARA compared to diets containing more dry hay or hay crop silages. Corn silages vary considerably in their fibre digestibility, due to genetic design such as brown midrib varieties (Oba and Allen, 1999, 2000) or due to growing and harvest conditions (Bal et al., 1997). Tests that estimate fibre digestibility can be very useful in identifying corn silages with unusually high rates and extents of ruminal fermentation. Unfortunately, most of these tests first require grinding the corn silage sample, which interfere with precise evaluation.

Corn silages also vary considerably in the extent of processing of the corn grain (e.g., kernel processing). A test for measuring the extent of grain processing within corn silage, based on the work of Ferreira and Mertens (2001), is now commercially available in the U.S. Combining these test results with digestibility data and particle length data furthers our ability to feed increasing amounts of corn silages without increased risk for SARA.

Corn silage is also difficult to feed because it typically does not contribute enough long particles to a total mixed ration (TMR). Very long chopping of corn silage is not recommended, because it impairs fermentation in the silo and increases the risk for sorting at the feed bunk (Kononoff et al., 2003). It is a common practice to add chopped long-stem dry hay or chopped dry straw to TMR containing a high proportion of the forage as corn silage. However, it can be difficult to process dry forage so that it distributes evenly throughout the TMR and is not easily sorted by the cows.

In many areas of the world forages are not the cheapest sources of nutrients, and non-forage fibre sources are used to provide fibre and other nutrients. Several short-term studies have demonstrated that fibrous by-products can replace forage NDF without negatively affecting milk production or cow health (Clark and Armentano, 1997; Harminson et al., 1997). A major consideration when replacing forages with fibrous by-product feeds is their relatively low potential for stimulating chewing activity. Because of their low lignin content and large proportion of potentially digestible fibre (Garleb et al., 1988), NDF from by-product feeds will ferment and pass from the rumen more quickly than fibre from forages. As less fibre is retained in the rumen to stimulate rumination, chewing, and saliva flow, diets containing by-products feeds appear to require more ration NDF.

Many dairy nutritionists use the number of cows in a herd ruminating at any given time as an indicator of healthy rumen function. A common goal is at least 0.4 (Eastridge, 2000). This guideline was confirmed by (Maekawa et al., 2002) when feeding a TMR for *ad libitum* intake. Importantly, the authors found that the timing of these observations was not critical for TMR diets, whereas when forage and concentrates were allocated separately, a lower proportion of the cows ruminated during the day (0.35) compared to the night (0.47).

Another way to assess the endogenous buffering capacity of a diet is by measuring the particle length distribution of the TMR actually consumed by the cows using the Penn State Forage Particle Separator (Lammers et al., 1996; Oetzel, 2001). Diets with less than 0.07 long particles (particles retained on the top screen of the separator) put cows at increased risk for SARA, particularly if these diets are also borderline or low in chemical fibre content (Grant et al., 1990; Woodford and Murphy, 1988). Increasing chemical fibre content of the diet may compensate for short particle length (Beauchemin et al., 1994).

Diets with excessive (over about 0.15) long forage particles can paradoxically increase the risk for SARA. This happens when the long particles are unpalatable and sortable. Leonardi and Armentano (2003) and Martin (1999, 2000) observed extensive TMR sorting in the feed bunk in university and on-farm trials, respectively. Data on particle size of TMR and orts and DM intake indicated that cows sorted against the coarse particles. This sorting against the coarse particles was more evident for the TMR containing 400 g/kg compared to 200 g/kg alfalfa hay (DM basis), and the variation in sorting among cows was large. Martin (1999, 2000) determined the particle size of TMR and bunk mix in a free-stall barn at 6 h intervals post-feeding on a commercial dairy. From a projection of the coarse particle intake at each time period, it appeared that intake of coarse particles was less than predicted during hours 0–12 post-feeding and more than predicted during hours 13–14 post-feeding. Socially dominant cows in free stall housing would be particularly susceptible to SARA in this scenario, because they are likely to consume more of the fine TMR particles soon after feed delivery. Cows lower in social rank then consume a lower energy diet later in the feeding period. Thus, cows on both ends of the social spectrum become thin and produce poorly. Limiting bunk space to less than 0.45 m per cow likely 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.

If sorting is determined to be a problem then options such as feeding smaller amounts of TMR more frequently; adding less, or higher quality hay to the TMR; or processing the

hay finer may be considered. If the TMR DM is over about 550 g/kg, water or a liquid feed supplement can be added to reduce diet DM to 500 g/kg or less.

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 DM intake and milk yield (Block and Sanchez, 2000; Sanchez et al., 1994). Optimal DCAD for early lactation diets is about +400 mequiv./kg of (Na + K) − (Cl + S) (Block and Sanchez, 2000). Mid-lactation cows have an optimal DCAD of about +275 to +400 mequiv./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.

The ability of a diet and feeding system to promote maximal amounts of ruminal buffering should be evaluated as part of the work-up of 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 mequiv./kg of (Na + K) − (Cl + S) should be supplemented with additional buffers to provide more Na or K relative to Cl and S.

6.2. *Excessive intake of rapidly fermentable carbohydrates*

Excessive intake of rapidly fermentable carbohydrates is the most obvious cause of ruminal acidosis. An important goal of effective dairy cow nutrition is to feed as much concentrate as possible, in order to maximise production, without causing ruminal acidosis. This is a difficult and challenging task because the indications of feeding excessive amounts of fermentable carbohydrates (decreased DM intake and milk production) are very similar to the results from feeding excessive fibre (again, decreased DM intake and milk production). An important distinction is that even slightly over-feeding fermentable carbohydrates causes long-term health problems, while slightly under-feeding fermentable carbohydrates reduces milk yield but does not compromise cow health.

Controlling the level and type of NFC in the ration is essential to preventing ruminal acidosis. Dietary NFC includes organic acids, sugars, starch and soluble fibre such as pectic substances. Hoover and Miller (1995) suggested restricting NFC to 350–400 g/kg of diet DM when the NFC is largely sugar or starch, or to 400–500 g/kg when other carbohydrates predominate. Hall (1999) tentatively suggested that target levels of the components within NFC should be 5 g/kg DM sugars, 100 g/kg DM soluble fibre, and 200 g/kg DM starch. These recommendations presume that effective fibre requirements are met.

Optimal NFC concentration in a diet is not the same for all diets, because it depends on the extent and rate of carbohydrate fermentation for each diet. Several reviews (Theurer, 1986; Mills et al., 1999; Owens et al., 1997) document that ruminal starch degradability ranges from 0.3 to almost 1 across grain types and degree of processing. Krause et al. (2002) reported that replacing dry, cracked corn with ground high moisture corn in a TMR fed to lactating dairy cattle decreased mean ruminal pH from 5.99 to 5.85. Similarly, Yang et

al. (2000) reported that mean ruminal pH decreased linearly with increasing processing of barley grain fed to lactating dairy cows. In feedlot cattle, a greater risk of ruminal acidosis has been reported when more rapidly fermented grain sources such as wheat (Elam, 1976) and steam-flaked sorghum (Reinhardt et al., 1997) are fed. Particle size analysis of grains is a useful adjunct test when assessing the risk for SARA in a dairy herd.

However, total intake of rapidly fermentable carbohydrates is probably more important than NFC percentage in the diet (Oetzel and Nordlund, 1998). Herds or groups within herds with higher DM intakes are at inherently higher risk for SARA and should be fed lower NFC diets than other herds or groups.

Dairy herds that use component feeding often increase grain feeding in early lactation faster than the expected rise in DM intake. This puts cows at great risk for SARA, because they cannot eat enough forage to compensate for the extra grain consumed (Nordlund et al., 1995). Declining forage intake in early post-partum cows has also been demonstrated in herds where concentrate feeding was increased too rapidly (Chase, 1993). Careful modelling of early lactation diets in such herds often reveals drastic fibre deficiencies around 1–3 weeks post-calving (Fig. 6).

Evaluating the dietary content for both fibre and non-fibre carbohydrates 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 DM content of the fermented feed ingredients). Careful bunk sampling and wet

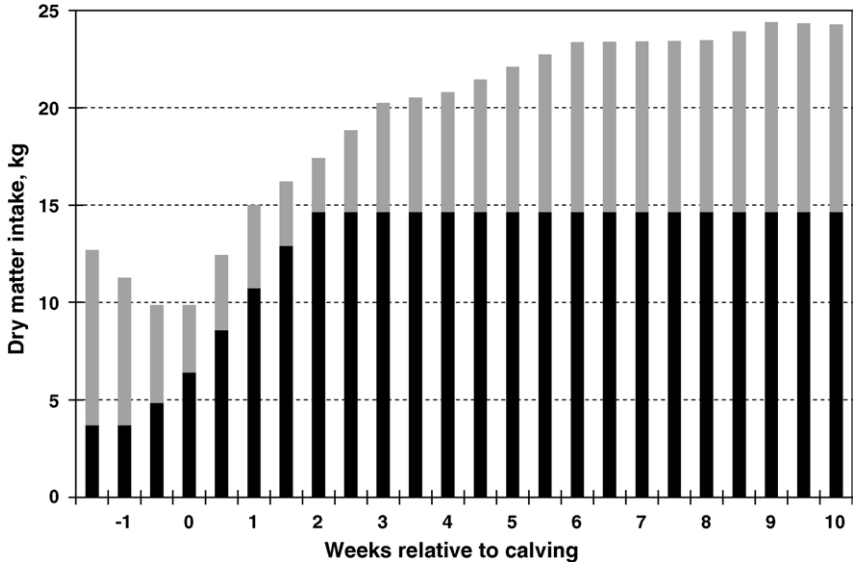

Fig. 6. Modeled concentrate (dark/bottom portion of bars) and forage (gray/top portion of bars) dry matter intake in early lactation cows where concentrate is rapidly introduced and fed separately.

chemistry analysis of total mixed rations may uncover unknown errors in feed composition or feed delivery.

6.3. Inadequate adaptation to highly fermentable, high carbohydrate diets

Ruminal adaptation to diets high in fermentable carbohydrates apparently has two key aspects, microbial adaptation and lengthening of the ruminal papillae (Dirksen et al., 1985). These principles suggest that increasing grain feeding toward the end of the dry period should decrease the risk for SARA in early lactation cows. However, a field study in TMR-fed herds found no effect of dry period feeding on early lactation ruminal pH (Garrett et al., 1997). And ruminal pH in this study was unexpectedly lower in cows at 106 average days in milk compared to cows at 15 average days in milk. These results suggest that high DM 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 DM intake (Andersen et al., 1999). These results suggest that the practical impacts of ruminal adaptation with regard to pH may be small or even inconsequential in dairy herds, and particularly when cows are fed a TMR after calving.

6.4. Feed additives for prevention of subacute ruminal acidosis

Dietary buffers cannot eliminate the causes of ruminal acidosis, but they can help manage the problem. The most common buffer in dairy cattle rations, sodium bicarbonate, has been shown to increase DM intake and milk production/milk fat percentage (Erdman, 1988). However, the response to feeding buffers depends upon the type of forage(s) fed and their physical structure. Buffer supplementation increased milk yield and milk fat yield when corn silage was the main forage, whereas results with grass/legume silages were inconsistent (Erdman, 1988; Staples and Lough, 1989). This difference in response could be explained by increased risk for SARA when corn silage is fed, as mentioned earlier. Also, added buffers are probably more likely to be beneficial in diets containing marginal amounts of effective fibre.

An important aspect of maintaining a stable rumen environment is maintaining a balance between lactate production and lactate utilisation by bacteria that convert lactate to less dangerous VFA. Enhancing ruminal lactate utilisers reduces the risk for ruminal acidosis (particularly the acute form of ruminal acidosis). Supplementation with specific yeast strains may enhance lactate utilisation within the rumen under certain dietary conditions (Dawson, 1995).

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 (Owens et al., 1998). Direct-fed microbials might also be used to provide a steady source of lactate in the rumen. A mixture of direct-fed microbials added to the rumen of dairy cows at the 1×10^5 dose increased corn digestibility and increased ruminal pH compared to higher doses of microbials (Nocek et al., 1999).

Selenomonas ruminantium is one of the bacteria that convert ruminal lactate to VFA. *S. ruminantium* is apparently stimulated to utilise lactate by malate (Martin and Streeter, 1995).

Although Martin et al. (1999) did not report any effect on ruminal lactate concentration, they did find that ruminal pH increased linearly with increasing malate supplementation in steers fed high grain diets. 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 (Callaway et al., 2000). Stage of maturity and variety affects malate concentrations in alfalfa (Callaway et al., 2000).

6.5. Management tools

Dairy cattle groups are commonly fed for *ad libitum* intake (typically a 5% daily feed refusal) in order to maximise potential DM intake and milk yield. However, slightly limiting intake in dairy cattle at high risk for SARA could in theory reduce their risk of periodic over-consumption and SARA. Restricting feed intake in feedlot cattle reduced daily DM intake variation, but did not affect ruminal pH (Choat et al., 2002). Feed restriction has also been found to improve feed efficiency in feedlot cattle (Hicks et al., 1990). 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 4 hours a day. This can be accomplished, 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. Providing daily feed refusals would especially apply to the pre- and post-parturient groups because they have rapid cow turnover and because individual cows have rapidly changing DM intake just before and after calving.

Meal size and frequency may be an extremely important aspect of nutritional management of SARA. Ruminal pH declines following meals, and the rate of pH decline increases as meal size increases and as dietary NDF concentration decreases (Allen, 1997). Cows are apparently able to self-regulate their ruminal pH very effectively if they have continuous and predictable access to the same TMR every day. However, even modest bouts of feed restriction can cause cows to subsequently consume meals that are too large (Fig. 5). Milton (1998) reported that deviations of 2–4 h from a normal feeding schedule greatly increased the risk of acidosis in feedlot cattle.

Feeding cows a TMR instead of feeding feeds separately avoids feeding large meals of grain, which reduces the risk of acidosis (Hernandez-Urdaneta et al., 1976). This observation is also supported by Østergaard and Gröhn (2000), who found that feeding concentrates separately from forage, and not the concentrate to forage ratio within a TMR, was associated with increased odds of metabolic disorders. Feeding a TMR permits greater control over the concentrate to forage ratio of diet actually consumed by the cow. Maekawa et al. (2002) found that cows fed a targeted concentrate to forage ratio of 50:50 (DM basis) actually consumed a ration closer to 0.6 concentrate and 0.4 forage when concentrate and forage was allocated separately. Also, these cows had lower minimum ruminal pH than cows fed a TMR with a concentrate to forage ratio of 50:50. Thus, an advantage of feeding TMR appears to be the ability to prevent low ruminal pH associated with increased intake of concentrates.

Good feedbunk management practices are critical for SARA prevention, even when chemical fibre, particle length, and grain processing are optimal. Bunk management

practices that cause cows to eat fewer and larger meals more quickly may be associated with an increased incidence of SARA. Factors that can cause slug feeding include limited feed access time, restricted feeding, and inconsistent feeding schedule. In free stall housing, limited bunk space, infrequent TMR push up and bunk competition are additional detriments to well-regulated feed intake. Milton (1998) reported that feedlot cattle fed for *ad libitum* intake had a higher frequency of meals (8.2 *versus* 4.5 per day) and smaller average meal size (1.6 *versus* 3.5 kg) than cattle fed to a clean bunk.

Design of the feeding alley might also affect the risk of SARA. Feeding TMR in a drive-by bunk at 10 cm above the cow alley rather than in an elevated bunk increases salivary flow and reduces sorting (Albright, 1993), which may help reduce the risk of SARA.

Because primiparous cows have lower DM intakes than older cows (because of lower body weight), it would seem that they should be a lower risk for SARA. However, clinical data from our herd investigations shows that primiparous cows may actually be at higher risk (Fig. 7). Primiparous cows sampled had a higher prevalence of SARA (29% *versus* 19% in second or greater lactation cows) and also appeared to be at risk for SARA earlier in lactation than the older cows. These are observational data only, and should be interpreted with caution. Primiparous cows may need time to learn to regulate their feed intake when consuming a high-energy diet, and they may have difficulty getting access to feedbunks for small, frequent meals when older cows are present in the same group. These concepts are supported by the results of Krohn and Konggaard (1979), who found that primiparous cows

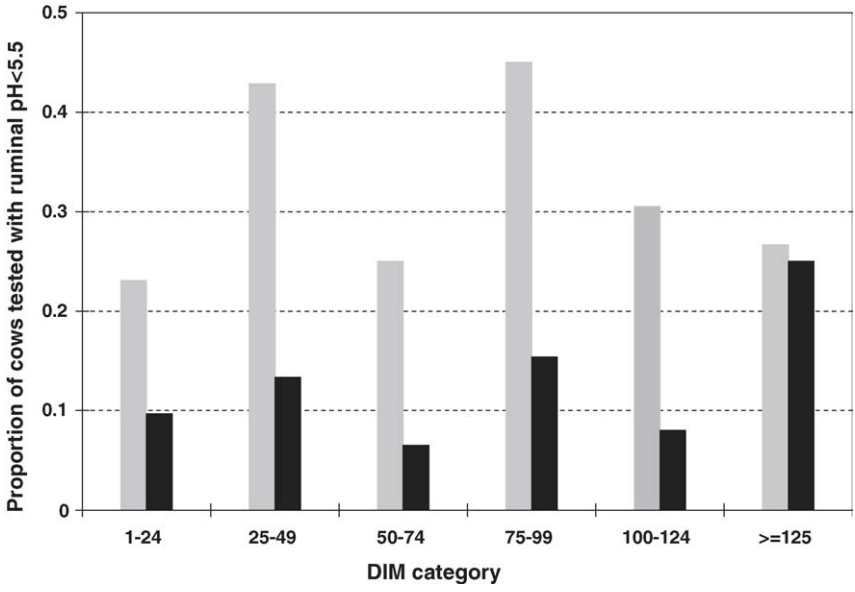

Fig. 7. Risk of low ruminal pH (<5.5) by days in milk (DIM) for lactation =1 (gray bars) and lactation >1 (black bars) from 397 cows in 36 herds. Samples were collected by rumenocentesis 6–10 h post-feeding in commercial dairy herds in Wisconsin, USA, between 1996 and 2003 (Oetzel et al., previously unpublished data).

fed in a separate group spent 10–15% more time eating and consumed 0.5–2.0 more meals per day than herd mates grouped with mature cows. Additionally, primiparous cows spend more time eating and perhaps have a slower eating rate than multiparous cows, as observed both by Campling and Morgan (1981) and Beauchemin and Rode (1994).

7. Conclusion

Subacute ruminal acidosis is an important determinant of dairy cow welfare and dairy herd profitability. The prevalence of SARA increases as cows consume more total DM and as cows consume diets containing higher proportions of grain. Milk production may transiently increase when grain feeding is excessive, but these gains are offset by the long-term problems in cow health that follow SARA. Dairy producers and nutritionists face the difficult challenge of providing diets that optimise energy intake and total milk yield without causing SARA.

Ruminant animals posses complex and multi-faceted systems for maintaining stable ruminal pH; thus, the nutritional approach to SARA prevention is complicated as well. One key aspect of SARA prevention is adequate ruminal buffering, which includes both dietary and endogenous buffers. This is accomplished by providing diets with proper amounts of cations relative to anions and adequate physical fibre that cannot be separated from other components of the diet. Another key aspect of SARA prevention is careful control of the intake of rapidly fermentable carbohydrates. This is accomplished by providing adequate chemical fibre, processing grains properly, including high fibre concentrates as needed and offering feed to cows in a manner that allows them to consume small, frequent meals on a regular basis. Feed additives and supplements can provide an additional margin of safety for preventing SARA.

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