The monitoring, prevention and treatment of sub-acute ruminal acidosis (SARA): A review

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Abstract

Sub-acute ruminal acidosis (SARA) has become an increasing problem in well-managed, high yielding dairy herds and the monitoring of groups of cows for signs of the condition is now crucial. Rumenocentesis may be ethically questionable but the technique remains the most reliable means of diagnosing SARA. Continuous measurement of ruminal pH may however be possible in the future. Parameters reflecting the metabolic acidosis caused by SARA are also promising tools, and measurement of milk fat content may be useful in individual mid-lactation cows although it is less valuable for bulk tank milk samples.

The prevention of SARA includes the establishment of feeding and management guidelines seeking to minimize rumen acidotic load. Regular monitoring may facilitate early recognition of the condition and limit economic losses. Some degree of SARA may however be inevitable and presents a challenge to the dairy industry as consumers become increasingly concerned about the welfare of production animals.

Keywords: SARA; Dairy cows; Management; Disease monitoring; Disease prevention

Introduction

Sub-acute ruminal acidosis (SARA), also known as chronic acidosis or sub-clinical rumen acidosis (SRA), is a well-recognised digestive disorder found particularly in well-managed dairy herds. Field studies in the United States have indicated that up to 19% of early lactation dairy cows as well as 26% of mid-lactation cows have SARA (Garret et al., 1997). Moreover, in one-third of the herds observed, 40% of all cows were found to have the condition. It has been estimated that the economic costs associated with SARA are US $500 million1 to US$1 billion annually, with the cost per affected cow estimated to be US$1.12 per day. These losses are mainly the result of reduced milk production, decreased efficiency of milk production, premature culling and increased death loss.

In a similar German/Dutch study, incidences of SARA in early and mid-lactation cows were found to be 11% and 18%, respectively (Kleen, 2004). It may therefore be appropriate to suggest that SARA is the most important nutritional disease of dairy cattle. This view is further substantiated by the fact that the addition of buffers to total mixed rations (TMR) is almost standard in North American dairy herds (Erdman, 1993). The challenge for dairy farmers, dairy nutritionists and bovine practitioners is to implement feeding management and husbandry practices to prevent SARA. To do so, however, demands that any ongoing SARA-problem can be recognized, which is not an easy task. This review deals with possible monitoring tools, and the treatment and prevention of SARA in dairy herds.
Occurrence

At the herd level, two distinct risk groups are usually defined: (1) cows in early lactation that are exposed to energy-rich rations too rapidly resulting in low rumen pH and (2) cows in mid-lactation which, due to their high feed intake, are particularly sensitive to sudden changes of feed or faults in feed composition and delivery (Nordlund et al., 1995). One Danish report showed a 0.2% occurrence of rumen acidosis (Blom, 1993) but this probably was not a reliable indication of the actual rate because few veterinarians include analysis of rumen fluids in their examination of dairy cattle (Enemark and Jørgensen, 2001). It is more likely that the figure indicates the occurrence of acute clinical rumen acidosis, which is easier to diagnose due to its specific history and its clear symptomatic picture.

A SARA related condition can also be seen during early lactation when dairy cows experiencing fluctuations in feed intake become inappetant (off-feed) due to periods of increased D- and L-lactate concentrations in the rumen fluid (Enemark and Jørgensen, 2002a; Höhling et al., 2004; Schwartzkopf-Genswein et al., 2004).

Monitoring SARA

Diagnosing SARA is difficult because clinical signs are subtle and delayed after the time of the acidic insult. As a result, routine monitoring and recording of related disease incidences, clinical signs and the dynamics of affected, para-clinical parameters may be the only ways to recognise SARA at an early enough stage to allow for corrective measures to management or feeding procedures.

SARA may be an aetiological factor for a number of diseases (Dirksen, 1985; Nordlund et al., 1995; Nocek, 1997) but, unfortunately, documentation is inadequate in many cases. However, increased incidence of one or more of the following diseases should raise suspicion about an ongoing SARA herd problem.

Rumenitis

Rumenitis is a frequent sequel to rumen acidosis. At present, the pathogenesis is not fully understood but an increased production of volatile fatty acids (VFA), particularly butyrate and propionate, as well as a temporary rise in the ruminal lactate concentration and fluctuations in the osmolality of the rumen fluid, may be involved in the development of the condition (Dirksen, 1985; Krehbiel et al., 1995).

The stage between parakeratosis (thickening of the stratum corneum of the rumen mucosa) and rumenitis appears undefined (Dirksen, 1985). Parakeratosis, when it occurs as a consequence of acute increased lactate production caused by induced clinical acute rumen acidosis, may affect VFA absorption in the long term (Krehbiel et al., 1995). Mucosal lesions in rumenitis may serve as an entrance for Fusobacterium necrophorum, and, more rarely, Acanthobacterium pyogenes, with subsequent colonisation in the submucosa. Embolic spread to the liver results in hepatic abscess formation (the rumenitis liver abscess complex), occasionally with metastasis to the pulmonary circulation via the posterior vena cava, causing rupture of minor pulmonary arteries into the bronchi (the caudal vena cava syndrome). Clinically, these episodes may lead to epistaxis and/or haemoptysis, characterised by bloody, foaming expectoration around the muzzle and nostrils. Generally, the outcome is fatal (Nordlund et al., 1995).

Metabolic acidosis

Lactate, particularly D-lactate, is responsible for the profound, uncompensated metabolic acidosis seen in cases of acute clinical rumen acidosis (Dunlop, 1972), whereas in cases of SARA the role of lactic acid is less clear. However, low rumen pH during episodes of SARA seems to be reflected in systemic metabolic acidosis (Brown et al., 2002). Probable decisive factors are the depth of the pH fall as well as the duration of episodes where pH is below a physiologically acceptable value (e.g. 5.5, Nocek, 1997).

It has not yet been determined whether lactate has any influence in metabolic acidosis (Coumoute et al., 1983; Höltershinken et al., 1997), as it apparently does not accumulate in the rumen fluid (Hibbard et al., 1995). However, lactate may play a role in inducing inappetance in early lactation, as a result of high dry matter intake (DMI) and over-eating following periods of feed deprivation, and rumen lactate may be a valid monitoring parameter (Enemark and Jørgensen, 2002a; Höhling et al., 2004).

Among the short-chained VFAs, only acetic acid reaches the peripheral circulation. Butyric acid is transformed largely in the rumen wall into hydroxy-butyric acid whereas all of the propionic acid is converted into glucose in the liver (Owens et al., 1998). German research has shown that serious cases of intracellular acidosis may occur even under low-grade, chronic acidosis conditions (Lachmann and Siebert, 1980; Lachmann et al., 1985). It is possible that this might compromise cellular function in the rumen wall and the liver, resulting in high VFA concentrations in the peripheral circulation causing metabolic acidosis (Owens et al., 1998).

Recent research has indicated that long-lasting metabolic acidosis may also cause damage to the organism in the form of reduced glucose dependent insulin secretion (Bigner et al., 1996), increased cortisol secretion (Ras et al., 1996), reduced phagocytic activity (Rossow and Horvath, 1988) and reduced migration speed of neutrophils (Hofirek et al., 1995). It has been shown in humans that metabolic acidosis results in increased protein catabolism and consequent impairment of growth (Baily, 1998) thus explaining the poor body condition despite adequate energy intake, often encountered in SARA herds (Nocek, 1997). Furthermore, bovine chronic metabolic acidosis prepartum affects steroid hormone concentrations around the time of calving, weakens the contractility of uterine smooth muscles (Ras et al., 1996), impairs the liver function...
(Lechowski, 1997) and causes dystocia (Ras et al., 1996). Also, weak newborn acidotic calves and increased disease incidence during the neonatal period have been documented (Ras et al., 1996).

Immunosuppression and suboptimal metabolism may be primary complications in long-lasting cases of metabolic acidosis and may explain reduced resistance to respiratory and other diseases (Mwansa et al., 1992) as well as low production results in herds suffering from SARA (Nordlund et al., 1995).

Feed intake

Decreased DMI is said to be a consistent clinical sign (Stock, 2000; Garry, 2002) and several studies have shown a lowered feed intake during periods of SARA (Olsson et al., 1998; Brown et al., 2000; Krajcarski-Hunt et al., 2002). The described changes in the feeding pattern in SARA cases may well be linked to changes in the osmolality of the rumen fluid because values that are considerably greater than 300 mOsm/L restrict feed intake and reduce the bacterial fermentation of fibre and starch (Carter and Grovum, 1990). Recent research has suggested that when given a choice of feeds, dairy cows alter their diet selection in an attempt to attenuate SARA (e.g. by consuming more hay) (Keunen et al., 2002), whereas sodium bicarbonate was not selected when the animals were given the choice (Keunen et al., 2003).

Abomasal displacement and abomasal ulcers

SARA has often been considered to be a risk factor for abomasal displacement (Svendsen, 1969; Markusfeld, 1987; Olson, 1991). Although a causal relationship has not been proven, increased backward and forward flow of ruminal derived gasses (SCFA, CO₂ and CH₄) between the abomasum and the forestomachs is believed to result in abomasal atony and dilatation and subsequent displacement (Svendsen, 1969; Sarashina et al., 1990). The theory is supported by the finding that a low fibre content in the feed ration is the most important single factor in the occurrence of abomasal displacement (Hultgren and Pehrson, 1996; Shaver, 1997; Cameron et al., 1998), and that the establishment of a functional fibre mat in the floating layer is believed to be of importance in the more gradual production and absorption of VFA in the forestomachs (Olson, 1991).

The occurrence of abomasal ulcers has been linked to intensive management and feeding of highly acidic diets consisting of concentrates and silage (Rebhun, 1995). The pathogenesis is not yet fully understood, but feed-induced acidosis has been shown to result in abomasal ulceration in goats (Aslan et al., 1995; Ivany et al., 2002; Cook et al., 2004) and a prevalence of >10% has been suggested as indicative of a SARA-problem in a herd (Nordlund and Garret, 1994; Garret, 1996). Numerous investigations have shown that there is a connection between starch content in feed rations and the occurrence of laminitis (Manson and Leaver, 1988; Mortensen, 1993; Wells et al., 1995; Nocek, 1997; Svensson and Bergsten, 1997). The pathogenesis is still uncertain, but it is presumed that vasoactive endotoxin released intraruminally is absorbed into the blood circulation and locally induces a vascular reaction causing vasoconstriction and hypoxemia resulting in pododermatitis (Andersen and Jarlov, 1990; Boosman, 1990; Andersen, 1994). A recent study pointed to the influence of metabolic changes at parturition (Tarlton et al., 2002).

Alimentary overload with oligofructose, one of the most abundant non-structural carbohydrates in several plant species including many grasses (Longland and Cairns, 2000), has been shown to induce signs of acute laminitis in heifers (Thoefner et al., 2004) as well as in horses (Van Eps and Pollitt, 2006) although the pathophysiological mechanism remains unknown.

Rumen tympany (bloat)

Bloat is of particular significance in fattening beef calves, but may also be a problem in dairy herds on high concentrate rations. The causal relations have not yet been established but the combination of reduced rumen motility caused by a low fibre ration and hence a low rumen pH, excessive production of mucopolysaccharides, and release of unknown macromolecules from rumen bacteria due to bacterial disintegration, are thought to result in the formation of a stable foam hindering eructation of gas (Cheng et al., 1998). Also rumen stasis, as a result of low rumen pH, may allow for the accumulation of free gas (Rebhun, 1995).

Reproduction

SARA may indirectly affect fertility in addition to calving and possibly the health of the newborn calf. Thus, a cycling feeding pattern or decrease in DMI during early lactation may, via the subsequent energy shortage, result in insufficient maturation of the first wave of post partum ova (Britt, 1995).

Economic consequences of SARA

It is obvious that SARA is of great economic importance to the dairy industries. Financial losses caused by SARA result from decreased milk production, decreased efficiency of milk production, premature culling, and increased death loss (Krause and Oetzel, 2005) and have been estimated to be US$1.12/day per cow in herds diagnosed with SARA (Stone, 1999). One American report showed that reduced feed intake alone, caused by SARA, led to reduced growth in beef calves, estimated to result
in a loss of US$10–13 per animal, plus additional losses from liver abscess formation, which occurred in 15% of cases (Stock and Britton, 1996). Under Danish conditions the incidence of liver abscesses among fattening bulls may reach 50% in certain herds (Kjeldsen et al., 2002).

Monitoring clinical signs

The clinical signs of SARA are subtle and often temporally separated from the inciting event, thus making diagnosis difficult. Sub-acute ruminal acidosis is considered to be a herd problem because the clinical signs are manifest in the herd rather than the individual, favouring the monitoring of groups of cows instead of diagnosing disease in individuals. This also allows for variation in ‘normal’ values among cows. At an individual level, many (if not all) of the signs described below, may have several causes besides SARA (Britton and Stock, 1986; Jørgensen et al., 1993a).

Feeding pattern

Cycling feeding pattern has been described as the most consistent symptom of SARA (Britton and Stock, 1986). Typically, the picture is one of cyclic feed intake as the cow eats its ration and subsequently refuses further feed due to a drastic fall in rumen pH and increased osmolality of the rumen fluid. Upon reestablishment of normal rumen conditions, appetite is often regained (Fulton et al., 1979). Such information is useful in herds with measured feed intake (automated feed dispensers), but in loose stalls changes in feeding behaviour will hardly be noticed, thereby making it useless as an indicator of SARA. As a result, rumination time may be reduced in cows with SARA. Some authors recommend that 40% of all cows should be ruminating at any one time (Maekawa et al., 2002), whereas others have suggested 80% (Chamberlain and Wilkinson, 2002). Monitoring of this parameter is likely to give an early indication of SARA.

Faeces

Faecal pH is normally not related to ruminal pH (Enemark et al., 2004) unless large amounts of starch by-pass the rumen and result in hindgut fermentation (Eastridge, 2000). In SARA cases, the faeces are bright, yellowish, have a sweet–sour smell (Kleen et al., 2003), appear foamy with gas bubbles, and contain more than normal amounts of undigested fibre or grain (Hall, 2002). Because there is an insufficient ruminal fibre mat, the fibre is not effectively retained in the rumen so the faeces contain 1–2 cm sized fibre particles compared to the more normal size of 0.5 cm (Hall, 2002). Faecal fibre particle size can be monitored routinely using an image analysis technique (Norgaard, 2004). Nordlund et al. (1995) reported on herds with loose faeces that contained substantial amounts of undigested feed particles. Intermittent diarrhoea and the presence of undigested particles indicate inadequate digestion and fast passage of feed.

Epistaxis, culling, death

Culling rate and number of inexplicable deaths within herds with SARA may be unacceptably high (Nordlund and Garret, 1994), and in the US epistaxis in cows from SARA herds is well known to the bovine practitioner and is considered almost pathognomonic (Nordlund et al., 1995; Garret, 1996).

Monitoring para-clinical parameters

Below is a brief description of some monitoring parameters in rumen fluid, blood, urine and milk, considered to be or likely to become relevant under field conditions.

Rumen fluid parameters

Monitoring of rumen pH is often used in the diagnosis of rumen acidosis. In Denmark, various types of stomach tubes are used to sample rumen fluid. Sampling and evaluation of rumen fluid has, however, never become part of the routine examination conducted by veterinary practitioners because it is too time consuming. Further, several investigations have shown that the diagnostic value of pH determination on rumen fluid sampled by stomach tube may be questionable because sample pH varies according to intra-ruminal localisation of the stomach tube, saliva contamination and time of sampling in relation to feeding (Hollberg, 1984; Höltershinken et al., 1992; Duffield and Plaizier, 2004; Enemark et al., 2004). An example elucidating the relationship between sampling procedure and pH is shown in Fig. 1 (Enemark et al., 2004).

![Fig. 1. X-Y plot, showing the relationship between rumen pH in samples obtained by either rumenocentesis (rc) or stomach tube (st) from apparently clinically healthy cows in six different dairy herds. Note the two rumenocentesis-derived samples (arrow) with pH values of 4.91 and 4.95, respectively (Enemark et al., 2004).](image-url)
The mean difference in rumen pH using the two methods (stomach tube and rumenocentesis) varies from 0.28 (Garret et al., 1999) to 0.76 (Enemark et al., 2004) or 1.1 (Nordlund et al., 1995). However, the relationship appears weak ($r^2 = 0.11$) (Enemark et al., 2004). These conditional variations add to the difficulties of comparing rumen pH in individual cows and herds. Furthermore, variation in herd level mean differences between sampling techniques (Table 1) does not allow for the use of regression analysis for comparing both techniques (Enemark et al., 2004).

Rumenocentesis is based on a method described by Hollberg (1984). The most useful cut-off point to differentiate animals as SARA positive has been identified as a pH value of 5.5. If the pH is $\leq 5.5$ the case should be considered as SARA positive and pH $\geq 5.8$ as negative (Garret et al., 1996). The interval between pH 5.5 and 5.8 is regarded as marginal and may suggest cows at risk of SARA. Garret et al. (1999) applied an evaluation model in which a herd, or a certain group of cows, is defined as having SARA when a rumen pH $< 5.5$ is found in more than four cows in a sample of 12. This model has its limitations, because it applies to herds with either a high ($>30\%$) or low ($<15\%$) prevalence of low ruminal pH.

Rumenocentesis is generally well accepted by cows. A German study revealed complications in 5.5% (9/164) of sampled cows (Kleen et al., 2004), typically haematomas and abscess formation. Promising results from studies using continuous measurements of rumen pH have shown that valid measurements for 24 h, 72 h and for as long as 21 days using indwelling probes (Plaizier et al., 1999; Nocek et al., 2002a,b; Penner et al., 2006), as well as 11 days with a ruminal wireless pH probe (Enemark et al., 2003), are possible. Therefore, it may be possible in future to monitor SARA in groups or herds using this method. In addition, Dewhurst et al. (2001) assessed the use of selected-ion-flow-tube mass spectrometric analysis of rumen gases from the rumen headspace and revealed that ammonia concentrations in rumen gas will be very low below rumen fluid pH 6, thus representing a further useful monitoring tool for SARA. Further research is needed to clarify whether this analysis can be used in conjunction with breath sampling (Mottram et al., 2000).

Besides pH, the most commonly applied analyses for rumen fluid, and their interpretation, are summarised in Table 2. Most of these tests can be applied under field conditions (Steen, 2001). The protozoal population is not believed to be affected at pH values between 6.2 and 5.3 but some research indicates that partial defaunation may be observed in cases of SARA (Jørgensen et al., 1993b) and that great individual differences exist (Franzolin and Dehority, 1996).

Recent studies by Enemark et al. (2004), Bramley et al. (2005) and Morgante et al. (2007) have indicated that the presence of high concentrations of ruminal valerate may be correlated with SARA as valerate is produced by lactolytic bacteria in the presence of lactate (Counotte and Prins, 1981; Leedl et al., 1995). Increased concentrations of ruminal valerate may therefore indicate a prior occurrence of SARA with accumulation of lactate.

### Milk parameters

#### Fat percentage

The fat percentage of milk is influenced by several factors, including lactation stage, breed and composition of feed rations (Grummer, 1991). Lowered milk fat content due to subacute ruminal acidosis is reflected in increased concentrations of valerate (Counotte and Prins, 1981; Leedl et al., 1995). Increased valerate concentrations may therefore indicate a prior occurrence of SARA with accumulation of valerate.

### Table 1

<table>
<thead>
<tr>
<th>Herd (number of cows)</th>
<th>Mean pH (st)</th>
<th>Mean pH (rc)</th>
<th>Mean difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (10)</td>
<td>7.09</td>
<td>6.02</td>
<td>1.07</td>
</tr>
<tr>
<td>2 (9)</td>
<td>6.64</td>
<td>5.92</td>
<td>0.68</td>
</tr>
<tr>
<td>3 (10)</td>
<td>6.56</td>
<td>6.25</td>
<td>0.33</td>
</tr>
<tr>
<td>4 (9)</td>
<td>6.36</td>
<td>5.64</td>
<td>0.72</td>
</tr>
<tr>
<td>5 (10)</td>
<td>7.05</td>
<td>6.06</td>
<td>1.04</td>
</tr>
<tr>
<td>6 (10)</td>
<td>6.57</td>
<td>5.88</td>
<td>0.69</td>
</tr>
</tbody>
</table>

### Table 2

<table>
<thead>
<tr>
<th>Colour/Green</th>
<th>Odour</th>
<th>Viscosity</th>
<th>Flotation/sedimentation</th>
<th>pH</th>
<th>Methylene blue test</th>
<th>Glucose fermentation test</th>
<th>Number of protozoa</th>
<th>Microbial composition</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grey-brown</td>
<td>Aromatic</td>
<td>Slightly</td>
<td>4–8 min</td>
<td>5.5–6.8</td>
<td>&lt;3 min</td>
<td>1–2 mL/h</td>
<td>+++</td>
<td>Gram negative &gt; Gram positive</td>
<td>Active ruminal fermentation</td>
</tr>
<tr>
<td>green</td>
<td>Ammonia</td>
<td>Viscous</td>
<td>Variable</td>
<td>6.8–8.5</td>
<td>?</td>
<td>↓</td>
<td>+/++</td>
<td>Gram negative &gt; Gram positive</td>
<td>Rumen alkalosis</td>
</tr>
<tr>
<td>Dark brown</td>
<td>Milky</td>
<td>Sticky</td>
<td>Watery</td>
<td>No/fast</td>
<td>5.2–3.8</td>
<td>&gt;5 min</td>
<td>↓</td>
<td>–</td>
<td>Gram positive &gt; Gram negative</td>
</tr>
<tr>
<td>green</td>
<td>Sour</td>
<td>Viscous</td>
<td>No/fast</td>
<td>6.2–5.3</td>
<td>&lt;3 min</td>
<td>n/↑</td>
<td>+++</td>
<td>Gram negative &gt; Gram positive</td>
<td>Sub-acute rumen acidosis</td>
</tr>
</tbody>
</table>

* Number of protozoa: –, none; +, few; ++, some; ++++, plenty; $n$ = normal.
* Microbial compositions: Gram negative or Gram positive bacteria dominate. ↓ = decreased or prolonged.
* Depending on season (winter or pasture feeding). ↑ = increased or shortened.
* Absolute increases in Gram positive bacteria.
* Intra-ruminal lactate concentration $>30$ mg/100 mL (3.3 mmol/L).
is frequently used in farms as an indicator of SARA and to predict the effectiveness of diet structure for chewing (Mertens, 1997; De Brabander et al., 2002). In two different studies, the correlation coefficient between ruminal pH and milk fat content in cows over 30 days in milk (DIM) was shown to be 0.305 and 0.390, respectively (Allen, 1997; Enemark et al., 2004). In the study of Enemark et al. (2004) the correlation coefficient was even negative for cows under 30 DIM ($r = -0.06$) underlining the fact that milk fat percentage in early lactation cows should not be used for the assessment of SARA.

The initial fat percentage generally registered in early lactation is influenced by several factors, including the general level of butterfat in the herd and the degree of fat mobilisation in the post partum cow. The depth of the initial drop in milk fat percentage between the first and second milk fat test post partum is thus hardly suitable for the evaluation of the fermentation pattern in the rumen.

To improve its diagnostic value, milk fat tests should be performed frequently (once a week) (Erdman, 1993). If carried out monthly, as in Denmark, brief periods of low fat percentages may remain unnoticed. However, advanced methods of in-line measurement of milk fat content will allow for daily assessment of milk fat content in the near future. At group or herd level, lactation curves may be useful as they can reveal a sudden drop of 1–2% in the average fat percentage of cows in mid-lactation, as may occur during sudden changes in feed such as an insufficient fibre supply. However, other factors in addition to rumen pH can affect milk fat content, such as changes in starch fermentability. Oba and Allen (2003) showed that increasing starch fermentability in a diet with 30% starch resulted in a 15% decrease in milk fat, without altering rumen pH. Also, addition of dietary fat, in particular unsaturated fatty acids, will enhance the effect of low rumen pH on milk fat content. Therefore, the interpretation of a low milk fat content has to take into account the use of dietary lipids, and their level of unsaturation. Although milk fat depression and SARA can arise in similar situations, milk fat depression cannot be simply considered as a sequel of SARA (Kleen et al., 2003).

Other biochemical markers in the milk have been linked to SARA and some potentially important markers are listed in Table 3. Inadequate experience in the use of these parameters under commercial conditions does however exclude them as monitoring tools for the time being, but future research may prove some of them to be valuable.

### Blood parameters

For many years, the integration of metabolic profiles as part of a monitoring programme in dairy herds has been routinely performed in the United States, Great Britain and Germany (Nelson, 1996; Ruegg, 1996; Ward et al., 1996), but up to now blood gas parameters have not been available for on-farm use. The recent launch of a transportable acid-base laboratory (IRMA7, Blood Analysis System, Diametrics Medical) makes it possible to include blood gas parameters as well as various electrolytes in the monitoring of dairy cows (Enemark and Jørgensen, 2002b).

Lachmann and Siebert (1980) found that the blood gas parameters were not notably affected in cases of chronic, metabolic acidosis, whereas Brown et al. (2000) demonstrated decreased blood pH and bicarbonate as well as base excess (metabolic acidosis) in steers with SARA. These findings were in accordance with results obtained by Horn et al. (1979) and Goad et al. (1998). Fürll (1994) emphasised the diagnostic value of acidosis-induced hypercalcemia and hyperphosphataemia and Aslan et al. (1995) demonstrated a positive blood glutaraldehyde coagulation test (Sandholm, 1974), presumably caused by rumenitis in clinical rumen acidosis-induced in goats. Whether such tests might be of any value in cases of SARA has not yet been examined, but it is obvious that rumenitis might initiate the production of certain acute phase proteins (haptoglobin and serum amyloid-A) that could be monitored (Gozho et al., 2005, 2006).

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**Table 3**

<table>
<thead>
<tr>
<th>Marker</th>
<th>Normal</th>
<th>‘Pathogenesis’</th>
<th>Changes due to SARA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat/protein-ratio (FPR)</td>
<td>1–1.5</td>
<td>Increased intra-ruminal propionate production, subsequent increase in blood glucose and increased lipogenesis in fat tissue resulting in lowered milk fat content</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Søxleth–Henkel-Fig. (SH)</td>
<td>6.4–6.8</td>
<td>Elimination of $\text{H}^+$ via the udder</td>
<td>&gt;8.0</td>
</tr>
<tr>
<td>Lactose</td>
<td>4.4–5.2%</td>
<td>Increased intra-ruminal propionate formation results in increased blood glucose and hence increased milk lactose content</td>
<td>?</td>
</tr>
<tr>
<td>Cl, Na, K</td>
<td>Cl: 25–31 mmol/L, Na: 20–26 mmol/L, K: 30–40 mmol/L</td>
<td>Cl: Na: K:</td>
<td>≤&lt;3.0 mmol/L</td>
</tr>
<tr>
<td>Milk-urea-nitrogen (MUN)</td>
<td>3.0–5.0 mmol/L</td>
<td>Energy (carbohydrate) content of ration in favour of protein results in reduced ruminal NH$_3$-formation and hence reduced hepatic urea formation</td>
<td>?</td>
</tr>
</tbody>
</table>
Urine parameters

The relatively small lung capacity of ruminants means that this organ only plays a minor part in the acid-base regulation. Acid elimination via the kidneys, on the other hand, is paramount. In SARA, where animals experience a compensated metabolic acidosis, renal excretion of $H^+$ is increased. The collection of urine samples from individual cows may present problems for inexperienced personnel. In our experience, efficient manual stimulation of the perineal area frequently, but not always, provokes spontaneous urination.

A positive connection has been established between rumen pH and urine pH (Roby et al., 1987; Fürl, 1994) but it should be borne in mind that aciduria can be caused by several conditions (Markusfeld, 1987) and is seen in cows on an anionic salt programme. Assessment of the renal net acid-base excretion (NABE), determined by urine titration, is claimed to be more accurate than pH determination on an anionic salt programme. Assessment of the renal net acid-base excretion (NABE), determined by urine titration, is claimed to be more accurate than pH determination because acidic conditions cause excretion of increased amounts of inorganic phosphate into the urine, acting as a buffer (Kutas, 1965; Lachmann and Seffner, 1979; Fürl, 1994). An on-farm test for simple assessment of NABE has been developed and was found to correlate well to the reference method ($r = 0.991$) (Enemark and Jørgensen, 2000). In a field study by Enemark et al. (2004) a correlation coefficient of $r = 0.57$ between urine pH and renal NABE was found, which could reflect excessive amounts of excreted phosphate in the urine of cows fed high grain diets. In an unpublished study, the correlation between rumen pH and NABE was found to be $r = 0.33$ ($N = 288, P < 0.01$), whereas the correlation between urine pH and rumen pH was $r = 0.28$ ($n = 323, P < 0.01$) (J.M.D. Enemark, unpublished data). NABE may therefore not be considered as a real time parameter of the rumen environment but rather a monitoring tool for metabolic acidosis in cattle.

Treatment and prevention

Some of the greatest advances in dairy health during the last 25 years have been associated with a shift to disease prevention, rather than treatment, and the increasing focus on groups or herds (LeBlanc et al., 2006). Furthermore, the clinical effects (economic losses) of SARA are delayed on the time of the acidotic insult, which makes prevention much more favourable to treatment. However, in more severe cases of SARA, therapeutic measures applicable to acute lactic rumen acidosis may be applied (Dunlop and Hammond, 1965).

Feeding and management

SARA is so closely linked to feeding conditions that correction of feed rations and/or feed management is essential to solve the problem. Simply put, prevention of SARA comes down to the need to allow for proper adaptation of the ruminal mucosa and the ruminal microflora in the periparturient period as well as keeping ruminal pH in physiological ranges despite high energy intake in the postpartum period (Kleen et al., 2003). The introduction of physically effective fibre (peNDF$_{>1.18}$ – the proportion of DM that is retained by a 1.18 mm screen multiplied by dietary neutral detergent fibre [NDF]) (Mertens, 1997) has provided us with a potential tool to estimate chewing, saliva production and rumen buffering and potential tool to regulate rumen pH (Yang and Beauchemin, 2006; Zebelli et al., 2006).

A revision of the feeding schedule will in itself only rarely reveal any major deficiencies. Table 4 lists frequent feeding and management problems as they occur in relationship to SARA and suggests how to these problems can be resolved.

Buffers and SARA

In North American feed lots, chemical buffers are regularly added to feed rations (Hutjens, 1991; Erdman, 1988) and have been shown to be beneficial in the prevention of acidosis in dairy cows (Garry, 2002). They may be added in cases where the fibre content in the feed rations is too low (Erdman, 1988). There is documentation showing that the addition of 150 g of sodium bicarbonate to the lactation feed per day had a positive effect on the milk yield (Downer and Cummings, 1985). Similarly, a positive effect has been demonstrated on feed intake and milk fat percentage (Erdman, 1988).

Table 5 lists recommended doses of various compounds used as buffers. The ideal buffer should be water-soluble and have a $pK_a$ value close to the optimal physiological pH of the rumen fluid. Sodium bicarbonate ($pK_a = 6.25$) meets these requirements and is the most frequently applied buffer, whereas the other compounds mentioned have only limited or no buffer effect although they do have an alkalinising or neutralising effect. Normally, a single buffer is used but combinations of several buffers are possible with a documented positive influence on milk yield, fat percentage and dry matter intake (Hutjens, 1991). Buffers, especially bicarbonate, may prevent an overgrowth of acid tolerant lactobacilli where feeding a high proportion of concentrate may cause a pH depression (Garry, 2002). A new rumen buffer known as Probimax Acid Buf (Engormix) is based on calcified seaweed and is thus a natural product. It claims to have more than twice the capacity of sodium bicarbonate and to increase milk yield and feed conversion. It claims to have more than twice the capacity of sodium bicarbonate and to increase milk yield and feed conversion. However, at present there are no studies to document these effects.

In the view of the author, the use of buffers may be justified during an acute problem with SARA but they should not be used on a routine basis to compensate for suboptimal feeding management.

Direct fed microbials (DFM) and SARA

It has been suggested that yeast cultures be added to feed rations. Most products contain a mix of live and dead yeast but documented research shows varying effects of the
addition of yeast (Williams et al., 1991; Aslan et al., 1995; Höltershinken et al., 1997). In a review by Nocek and Kautz (2006) it was shown that three different organisms (Enterococcus faecium, Lactobacillus plantarum, Saccharomyces cerevisiae) administered at 10^9 cfu/mL reduced diurnal rumen acidity and improved digestion of corn silage. Equally, there was an enhanced ruminal digestion of forage DM, increased milk production and DM consumption when feeding early lactation cows with direct fed microorganisms (DFM) in the form of two strains of E. faecium and yeast, both supplemented at 5 × 10^9 cfu/day), but these cows experienced also a lower milk fat percentage.

Beauchemin et al. (2003) could not show significant effects on the site and extent of digestion or blood chemistry and the occurrence of SARA, when using DFM (E. faecium, S. cerevisiae) in feedlot cattle. E. faecium (6 × 10^9 cfu/day) was either given alone or together with S. cerevisiae (Both: 6 × 10^9 cfu/day). She concluded that DFM were of limited value for feedlot cattle already adapted to high grain diets. The most consistently reported response to the use of S. cerevisiae is a trend to increased total culturable and cellulytic bacteria recovered from the rumen (Wallace and Newbold, 1993), although the increase in many studies did not reach statistical significance. The interested reader should consult the comprehensive review on DFM in ruminants by Wallace and Newbold (1995). At present there is not enough evidence to justify the use of DFM for controlling SARA.

**Stimulation of lactolytic flora**

Genetic manipulation of lactolytic bacteria is a relatively new idea with the aim of increasing the lactate conversion capacity and acid resistance of the bacteria (Martin and Dean, 1989) but no commercially available product has yet been developed. Supplementation of dicarboxylic acids, such as fumarate and maleate, may also act in this way, but documentation is not yet available (Owens et al., 1998).

**Immunization**

A study from Australia has shown that the risk of lactic acidosis in sheep could be reduced by immunization with a live S. bovis vaccine (Shu et al., 2000). Similar effects have been shown in cattle (Shu et al., 1999). It might therefore be speculated that immunization could provide some degree of protection against SARA but the research to prove this hypothesis is not yet available.

**Antibiotics**

The use of antibiotics in the prevention of SARA, thereby controlling the lactate production of mainly S.


 References

 bosis and Lactobacillus spp., has been proposed. Iono-
phores like monensin have proved to increase total tract nutrient digestion, but did not affect DM intake, milk yield and composition, or ruminal pH characteristics when given as a premix to cows with grain-induced SARA (Osborne et al., 2004). In another study, no effect of monensin on ruminal pH during SARA could be shown (Mutsvangwa et al., 2002). The use of these compounds therefore appears to be doubtful. In any case, in the European Union, their use is obsolete. Finally, the use of drugs with the goal of maintaining ruminants on a non-ruminant diet is question-
able (Dirksen, 1985).

Conclusions
The aetiology of SARA and its occurrence in early lactation places it in the borderland between traditional veterinary science and nutritional science. Accordingly, the success of a directed effort against SARA depends on cooperation between veterinary and nutritional researchers. The complex aetiology and pathogenesis, together with the clinical course of the disease, complicate its demarcation, diagnosis, monitoring and prevention. Among the monitoring parameters described in the present paper, none can stand alone and be applied unambiguously to confirm SARA at the herd level. At present, routine monitoring of rumen pH by rumenocentesis may be the most efficient way for SARA monitoring, but other tools are under development, such as checking the acidity of the urine or continuous measurement of rumen pH. These must be combined with a thorough knowledge of the feeding regime of the herd and systematic routine health recording.

Conflict of interest statement
The author (Jörg M. D. Enemark) has no financial or personal relationship with other people or organisations that could inappropriately influence or bias the paper entitled The monitoring, prevention and treatment of subacute ruminal acidosis (SARA): A Review.

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