

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

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Accepted 18 December 2007

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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.

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

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## 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 million<sup>1</sup> to US\$1 billion annually, with the cost per affected cow esti-

mated 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.

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<sup>1</sup> US\$1 = approximately UK£0.50; €0.67, as at 22 February 2008.

## 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 acidotic 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, *Acanobacterium 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 expectorate 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 (Counotte 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 pre-partum 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 (Svendensen, 1969; Markusfeld, 1987; Olson, 1991). Although a causal relationship has not been proven, increased backward and forward flow of ruminal derived gasses (SCFA, CO<sub>2</sub> and CH<sub>4</sub>) between the abomasum and the forestomachs is believed to result in abomasal atony and dilatation and subsequent displacement (Svendensen, 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).

#### *Laminitis*

North American researchers have asserted that laminitis is the most significant sequel to SARA (Oetzel, 2000; 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 hypoxaemia resulting in pododermatitis (Andersen and Jarløv, 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 (Nørgaard, 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).

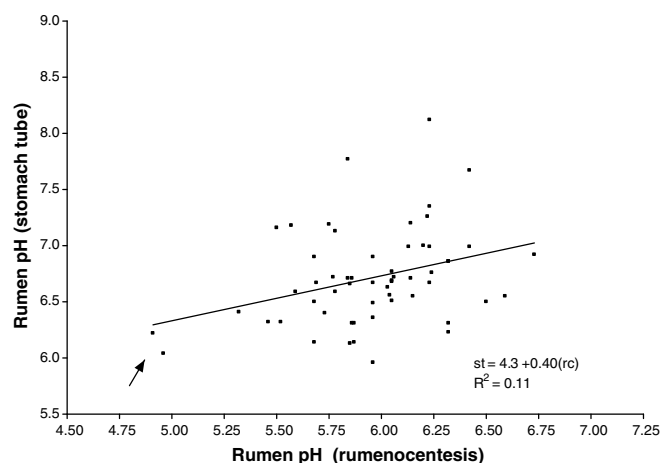


Fig. 1. X–Y plot, showing the relationship between rumen pH in samples obtained by either rumenocentesis (rc) or stomach tube (st) from apparently clinically healthy cows in six different dairy herds. Note the two rumenocentesis-derived samples (arrow) with pH values of 4.91 and 4.95, respectively (Enemark et al., 2004).

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

Table 1  
Mean difference between rumen pH values in rumen samples obtained by either stomach tube (st) or rumenocentesis (rc) in six different dairy herds (Enemark et al., 2004)

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

Table 2  
Most important changes in rumen fluid from cows and their significance (modified after Dirksen, 1979)

Colour	Odour	Viscosity	Flotation/sedimentation	pH	Methylene blue test	Glucose fermentation test	Number of protozoa <sup>a</sup>	Microbial composition <sup>b</sup>	Diagnosis
Grey-brown or green <sup>c</sup>	Aromatic	Slightly viscous	4–8 min	5.5–6.8	$< 3$ min	1–2 mL/h	+++	Gram negative > Gram positive	Active ruminal fermentation
Dark brown/green	Slight ammonia	Variable	Variable	6.8–8.5	?	↓	+ / +++	Gram negative > Gram positive	Rumen alkalosis
Milky/green	Sticky/sour	Watery	No/fast	5.2–3.8	$> 5$ min	↓	–	Gram positive > Gram negative	Acute rumen acidosis <sup>e</sup>
Slightly milky/brown	Sour	Slightly viscous	No/fast	6.2–5.3	$< 3$ min	$n/\uparrow$	+++	Gram negative > Gram positive <sup>d</sup>	Sub-acute rumen acidosis

<sup>a</sup> Number of protozoa: –, non; +, few; ++, some; +++, plenty;  $n$  = normal.

<sup>b</sup> Microbial compositions: Gram negative or Gram positive bacteria dominate. ↓ = decreased or prolonged.

<sup>c</sup> Depending on season (winter or pasture feeding). ↑ = increased or shortened.

<sup>d</sup> Absolute increases in Gram positive bacteria.

<sup>e</sup> 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 constituents 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 hypercalcaemia 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).

Table 3  
Biological parameters in milk of potential importance for monitoring the occurrence of SARA in dairy cattle (Enemark et al., 2002)

Marker	Normal	'Pathogenesis'	Changes due to SARA
Fat–protein–ratio (FPR)	1–1.5	Increased intra-ruminal propionate production, subsequent increase in blood glucose and increased lipogenesis in fat tissue resulting in lowered milk fat content	<1
Soxleth–Henkel–Fig. (SH) Titration of milk sample with sodium bicarbonate	6.4–6.8	Elimination of H <sup>+</sup> via the udder	>8.0
Lactose	4.4–5.2%	Increased intra-ruminal propionate formation results in increased blood glucose and hence increased milk lactose content	
Cl, Na, K	Cl: 25–31 mmol/L Na: 20–26 mmol/L K: 30–40 mmol/L	?	Cl: Na: K:
Milk–urea–nitrogen (MUN)	3.0–5.0 mmol/L	Energy (carbohydrate) content of ration in favour of protein results in reduced ruminal NH <sub>3</sub> -formation and hence reduced hepatic urea formation	<3.0 mmol/L

### 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; Fürll, 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 because acidotic conditions cause excretion of increased amounts of inorganic phosphate into the urine, acting as a buffer (Kutas, 1965; Lachmann and Seffner, 1979; Fürll, 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 from 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 peri-

parturient 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<sub>>1.18</sub> – 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. 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

Table 4  
Commonly occurring feeding and management deficiencies resulting in SARA and suggested corrections

Problem	Correction	Effect	Reference
Steaming up <4 weeks	Allow for 4–6 weeks stepwise adaptation	Optimal proliferation or rumen mucosa and ruminal microflora	Nordlund et al. (1995), Donovan et al. (2004) Oetzel (2003)
Steaming up is too intensive	Maximum increase of concentrate/day should be 0.25 kg	The rumen environment can absorb/neutralise VFA and lactate	
Only one TMR lactation and one TMR dry cow ration	Design rations for group of cows at certain lactation state	Energy content of ration targeted at ruminal mucosa capacity	Nordlund et al. (1995)
Errors in nutrient delivery (variation in DM, NE <sub>L</sub> and NDF)	Assess and control sources of error (sampling bunker silos, moisture content, accurate weights, bunk sampling)	Ruminal stability (balance between lactogenic and lactolytic bacteria)	Stone (2004), Shaver (2005), Oetzel (2003)
Grains too finely ground, steam flaked, extruded or/and wet	Particle size analysis of grains	Less rapid fermentation of grains in the rumen	Oetzel (2003)
Diets with high DCAD promoting low rumen pH	Adding buffer to the diet or stimulate chewing and rumination activity (7% fibre particle >3.5 cm), 27–30% NDF (70–80% from forage to ensure adequate eNDF), 35–45% of DM as NFC	Increased ruminal buffer capacity either directly or via increased saliva production	Oetzel (2003)
More than 15% long forage particles (promotes sorting)	Analysis of bunk samples along with adequate bunk space	Adequate content of long fibres prevent sorting, and adequate bunk space prevents slug feeding	Oetzel (2003)
Over-mixing or over-processing of the TMR (reduced particle size and eNDF content)	Control mixing time as well as condition of TMR scales	Homogeneous ration providing a stable ruminal environment	Mutsvangwa (2003)

VFA – volatile fatty acids; TMR – total mixed ration; DM – dry matter; NE<sub>L</sub> – net energy for lactation; NDF – neutral detergent fibre; DCAD – diet cation–anion difference; eNDF – effective NDF; NFC – non-fibre carbohydrates.

Table 5  
Recommended doses of various buffers added to the feed rations of lactating cows (Hutjens, 1991)

Product	Amount (g/day)
Sodium bicarbonate	110–225
Sodium sesquicarbonate	110–225
Magnesium oxide	50–90
Sodium bentonite	110–454
Calcium carbonate	115–180
Potassium carbonate	270–410

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<sup>5</sup> 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 microbials (DFM) in the form of two strains of *E. faecium* and yeast, both supplemented at 5 × 10<sup>9</sup> 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<sup>9</sup> cfu/day) was either given alone or together with *S. cerevisiae* (Both: 6 × 10<sup>9</sup> 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 cellulolytic 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.*

*bovis* and *Lactobacillus* spp., has been proposed. Ionophores 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 questionable (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 sub-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*.

## Acknowledgement

Thanks to Alltech and the School of Agriculture, Food Science and Veterinary Medicine, University College Dublin (UCD) for inviting me as a speaker to their Dairy Solution Symposium – Production diseases of the transition cow, Dublin, 28th and 29th September 2006.

## References

Allen, M., 1997. Relationship between fermentation acid production in the rumen and the requirement for physically effective fiber. *Journal of Dairy Science* 80, 1447–1462.

Andersen, P.H., 1994. Portal infusion of low dosage endotoxin: a model simulating translocation of ruminal endotoxin in cattle. *Acta Veterinaria Scandinavica* 35, 111–114.

Andersen, P.H., Jarlov, N., 1990. Investigation of the possible role of endotoxin, TXA<sub>2</sub>, PGI<sub>2</sub> and PGE<sub>2</sub> in experimentally induced rumen acidosis in cattle. *Acta Veterinaria Scandinavica* 31, 27–38.

Aslan, V., Thamsborg, S.M., Jørgensen, R.J., Basse, A., 1995. Induced acute ruminal acidosis in goats treated with yeast (*Saccharomyces cerevisiae*) and bicarbonate. *Acta Veterinaria Scandinavica* 36, 65–77.

Baily, J.L., 1998. Metabolic acidosis and protein catabolism: mechanisms and clinical implications. *Mineral and Electrolyte Metabolism* 24, 13–19.

Beauchemin, K.A., Yang, W.Z., Morgavi, D.P., Ghorbani, G.R., Kautz, W., 2003. Effects of bacterial direct-fed microbials and yeast on site and extent of digestion, blood chemistry, and subclinical rumen acidosis in feedlot cattle. *Journal of Animal Science* 81, 1628–1640.

Bigner, D.R., Goff, J.P., Faust, M.A., Burton, J.L., Tyler, H.D., Horst, R.L., 1996. Acidosis effects on insulin response during glucose tolerance tests in jersey cows. *Journal of Dairy Science* 79, 2182–2188.

Blom, J.Y., 1993. Disease and feeding in Danish dairy herds. *Acta Veterinaria Scandinavica Suppl.* 89, 17–22.

Boosman, R., 1990. Bovine laminitis, histopathologic and arteriographic aspects, and its relation to endotoxaemia. Ph.D. Thesis, Utrecht, p. 169.

Bramley, E., Lean, I.J., Costa, N.D., Fulkerson, W.J., 2005. Acidosis in dairy cows. *Journal of Animal Science* 83 (Suppl. 1), 251 (abstract).

Britt, J.H., 1995. Relationship between postpartum nutrition, weight loss and fertility. *Cattle Practice (BVCA)* 3, 79–83.

Britton, R.A., Stock, R.A., 1986. Acidosis, rate of starch digestion and intake. *Oklahoma Agricultural Experiment Station MP-121*, pp. 125–137.

Brown, M.S., Krehbiel, C.R., Galyean, M.L., Remmenga, Peters, J.P., Hibbard, B., Robinson, J., Moseley, W.M., 2000. Evaluation of models of acute and subacute acidosis on dry matter intake, ruminal fermentation, blood chemistry, and endocrine profiles of beef steers. *Journal of Animal Science* 78, 3155–3168.

Carter, R.R., Grovum, W.L., 1990. A review of the physiological significance of hypertonic body fluids on feed intake and ruminal function: salivation, motility and microbes. *Journal of Animal Science* 68, 2811–2832.

Chamberlain, A.T., Wilkinson, J.M., 2002. In: Chamberlain, T., Wilkinson, J.M. (Eds.), *Feeding the Dairy Cow*. Chalcombe Publications, Lincoln, UK, p. 241.

Cheng, K.-J., McAllister, T.A., Popp, J.D., Hristov, A.N., Mir, Z., Shin, H.T., 1998. A review of bloat in feedlot cattle. *Journal of Animal Science* 76, 299–308.

Cook, N.B., Nordlund, K.V., Oetzel, G.R., 2004. Environmental influences on claw horn lesions associated with laminitis and subacute ruminal acidosis in dairy cows. *Journal of Dairy Science* 87 (Suppl.), E36–E46.

Counotte, G.H.M., Prins, R.A., 1981. Regulation of lactate metabolism in the rumen. *Veterinary Research Communication* 5, 101–115.

Counotte, G.H.M., Lankhorst, A., Prins, R.A., 1983. Role of D, L-lactic acid as an intermediate in rumen metabolism of dairy cows. *Journal of Animal Science* 56, 1222–1235.

De Brabander, D.L., De Boever, J.L., Vanacker, J.M., Geerts, N.E., 2002. Evaluation and effects of physical structure in dairy cattle nutrition. In: *Proceedings of the 22nd World Buiatrics Congress*, Hannover, Germany, pp. 182–197.

Dewhurst, R.J., Evans, R.T., Mottram, T.T., Spanel, P., Smith, D., 2001. Assessment of rumen processes by selected-ion-flow-tube mass spectrometric analysis of rumen gases. *Journal of Dairy Science* 84, 1438–1444.

Dirksen, G., 1979. Digestive system. In: Rosenberger, G. (Ed.), *Clinical Examination of Cattle*, second ed. Verlag Paul Parey, Berlin and Hamburg, Germany, p. 186.

Dirksen, G., 1985. Der Pansenazidose-Komplex – neuere Erkenntnisse und Erfahrungen (1). *Tierärztliche Praxis* 13, 501–512.

Donovan, G.A., Risco, C.A., DeChant Temple, G.M., Tran, T.Q., van Horn, H.H., 2004. Influence of transition diets on occurrence of

- subclinical laminitis in Holstein dairy cows. *Journal of Dairy Science* 87, 73–84.
- Downer, J.V., Cummings, K.R., 1985. A ten year review of lactation study. *Journal of Dairy Science* 68 (Suppl. 1), 191–201.
- Duffield, T., Plaizier, J.C., 2004. Comparison of techniques for measurement of rumen pH in lactating cows. *Journal of Dairy Science* 87, 59–66.
- Dunlop, R.H., Hammond, P.B., 1965. D-lactic acidosis of ruminants. *Annals of the New York Academy of Science* 119, 1109–1132.
- Eastridge, M.L., 2000. Guidelines for low forage diets. In: *Proceedings of the Tri-state Nutrition Conference*, Fort Wayne, Indiana, US, pp. 97–110.
- Enemark, J.M.D., Jørgensen, R.J., 2000. Preliminary studies of on-farm assessment of Net acid-base excretion in cattle urine. *Danish Veterinary Journal* 83, 6–11.
- Enemark, J.M.D., Jørgensen, R.J., 2001. Subclinical rumen acidosis as a cause of reduced appetite in newly calved dairy cows in Denmark: results of a poll among Danish dairy practitioners. *Veterinary Quarterly* 23, 206–210.
- Enemark, J.M.D., Jørgensen, R.J., 2002a. Mammary lactate and renal inorganic phosphorous excretion in cows during steaming up and subsequent voluntary grain engorgement. In: *Proceedings of the XXII World Buiatrics Congress*, August, Hannover, Germany, p. 35.
- Enemark, J.M.D., Jørgensen, R.J., 2002b. On-farm determination of blood acid-base and related parameters in diagnosing subclinical rumen acidosis. *Danish Veterinary Journal* 85, 6–13.
- Enemark, J.M.D., Jørgensen, R.J., Enemark, P.S., 2002. Rumen acidosis with special emphasis on diagnostic aspects of subclinical rumen acidosis: a review. *Veterinarija ir Zootechnika* 20, 16–29.
- Enemark, J.M.D., Peters, G., Jørgensen, R.J., 2003. Continuous monitoring of rumen pH – a case study with cattle. *Journal of Veterinary Medicine A* 50, 62–66.
- Enemark, J.M.D., Jørgensen, R.J., Kristensen, N.B., 2004. An evaluation of parameters for the detection of subclinical rumen acidosis in dairy herds. *Veterinary Research Communication* 28, 687–709.
- Erdman, R.A., 1988. Dietary buffering requirements of the lactating cow: a review. *Journal of Dairy Science* 71, 3246–3266.
- Franzolin, R., Dehority, B.A., 1996. Effect of prolonged high-concentrate feeding on ruminal protozoa concentrations. *Journal of Animal Science* 74, 2803–2809.
- Fulton, W.R., Klopfenstein, T.J., Britton, R.A., 1979. Adaption to high concentrate diets by beef cattle. I. Adaption to corn and wheat diets. *Journal of Animal Science* 49, 785–791.
- Fürll, M., 1994. Diagnostik und Therapie chronischer Störungen des Säure-Basen-Haushaltes (SBH) bei Rindern. *Der Praktische Tierarzt* 75, 49–54.
- Garret, E., 1996. Subacute rumen acidosis – clinical signs and diagnosis in dairy herds. *Large Animal Veterinarian* 11, 6–10.
- Garret, E.F., Nordlund, K.V., Goodger, W.J., Oetzel, G.R., 1997. A cross-sectional field study investigating the effect of periparturient dietary management on ruminal pH in early lactation dairy cows. *Journal of Dairy Science* 80 (Suppl. 1), 169 (Abstract).
- Garret, E.F., Pereira, M.N., Nordlund, K.V., Armentano, L.E., Goodger, W.J., Oetzel, G.R., 1999. Diagnostic methods for the detection of subacute ruminal acidosis in dairy cows. *Journal of Dairy Science* 82, 1170–1178.
- Garry, F.B., 2002. Indigestion in ruminants. In: Smith, B.P. (Ed.), *Large Animal Internal Medicine*, third ed. Mosby, St. Louis and Baltimore, pp. 722–747.
- Goad, D.W., Goad, C.L., Nagaraja, T.G., 1998. Ruminal microbial and fermentative changes associated with experimentally induced subacute acidosis in steers. *Journal of Animal Science* 76, 234–241.
- Gozho, G.N., Plaizier, J.C., Krause, D.O., Kennedy, A.D., Wittenberg, K.M., 2005. Subacute ruminal acidosis induces ruminal lipopolysaccharide endotoxin release and triggers an inflammatory response. *Journal of Dairy Science* 88, 1399–1403.
- Gozho, G.N., Krause, D.O., Plaizier, J.C., 2006. Rumen lipopolysaccharide and inflammation during grain adaption and subacute ruminal acidosis in steers. *Journal of Dairy Science* 89, 4404–4413.
- Grummer, R.R., 1991. Effect of feed on the composition of milk. *Journal of Dairy Science* 74, 3244–3257.
- Hall, M.B., 2002. Characteristics of manure. In: *Proceedings of the Tri-state Dairy Nutrition Conference*, Fort Wayne, Indiana, US, pp. 141–147.
- Hibbard, B., Peters, J.P., Chester, S.T., Robinson, J.A., Kotarski, S.F., Warren, J., Croom, J., Hagler Jr., W.M., 1995. The effect of slaframine on salivary output and subacute and acute acidosis in growing beef steers. *Journal of Animal Science* 73, 516–525.
- Hofirek, B., Slosarkova, S., Ondrova, J., 1995. Effect of chronic metabolic acidosis on migration activity of polymorphonuclear leukocytes in sheep. *Veterinari Medicina* 40, 171–175.
- Höhling, A., Höltershinken, M., Holsten, N.B., Scholz, H., 2004. Influence of starvation on fermentation in bovine rumen fluid (in vivo). In: *Proceedings of the XXIII World Buiatrics Congress*, 11–16 July, Quebec, Canada, p. 183.
- Hollberg, W., 1984. Vergleichende Untersuchungen von mittels Schambye-Sørensen-Sonde oder durch Punktion des kaudodorsalen Pansensacks gewonnenen Pansensaftproben. *Deutsche Tierärztliche Wochenschrift* 91, 317–320.
- Höltershinken, M., Vlizzo, V., Mertens, M., Scholz, H., 1992. Untersuchungen zur Zusammensetzung von über Sonde bzw. Fistel genommenen Pansensaft des Rindes. *Deutsche Tierärztliche Wochenschrift* 99, 228–230.
- Höltershinken, M., Kress, V., Rathjens, U., Rehage, J., Scholz, H., 1997. Auswirkungen oral zu verabreichender Therapeutika auf Fermentationsvorgänge im Pansensaft ruminierender Rinder (in vitro). 7. Mitteilung: Wirkung von Trockenhefe bei chronischer Pansenacidose. *Deutsche Tierärztliche Wochenschrift* 104, 317–320.
- Horn, G.W., Gordon, J.L., Prigge, E.C., Owens, F.N., 1979. Dietary buffers and ruminal and blood parameters of subclinical lactic acidosis in steers. *Journal of Animal Science* 48, 683–691.
- Hutjens, M.F., 1991. Feed additives. *Veterinary clinics of North America. Food Animal Practice* 7, 525–540.
- Ivany, J.M., Rings, D.M., Anderson, D.E., 2002. Reticuloruminal disturbances in the bovine. *The Bovine Practitioner* 36, 56–64.
- Jørgensen, R.J., Erdman, R., Murphy, M., Olsson, A.C., Foldager, J., Nørgaard, P., Møller, P.D., Andersen, P.H., Nielsen, I., Østerås, O., Harmoinen, P., Kadari, K., Emanuelson, M., Reintam, E., 1993a. Rumen acidosis: identification of potential areas of research. Summary of group discussion. *Acta Veterinaria Scandinavica*, Suppl. 89, 153–154.
- Jørgensen, R.J., Thamsborg, S.M., Aslan, V., 1993b. A pilot study on health and appetite in beef calves fattened on pelleted lucerne versus concentrate ad libitum. *Acta Veterinaria Scandinavica*, Suppl. 89, 113–116.
- Keunen, J.E., Plaizier, J.C., Kyrzakakis, L., Duffield, T.F., Widowski, T.M., Lindinger, M.I., McBride, B.W., 2002. Effects of a subacute ruminal acidosis model on the diet selection of dairy cows. *Journal of Dairy Science* 85, 3304–3313.
- Keunen, J.E., Plaizier, J.C., Kyrzakakis, L., Duffield, T.F., Widowski, T.M., Lindinger, M.I., McBride, B.W., 2003. Short communication: effects of a subacute ruminal acidosis on free-choice intake of sodium bicarbonate in lactating dairy cows. *Journal of Dairy Science* 86, 954–957.
- Kjeldsen, A.M., Bossen, D., Fisker, I., 2002. Liver abscesses in fattening bulls. Report no. 96, Danish Cattle, p. 75.
- Kleen, J.L., Hooijer, G.A., Rehage, J., Noordhuizen, J.P.T.M., 2003. Subacute ruminal acidosis (SARA): a review. *Journal of Veterinary Medicine A* 50, 406–414.
- Kleen, J.L., Hooijer, G.A., Rehage, J., Noordhuizen, J.P.T.M., 2004. Rumenocentesis (rumen puncture): a viable instrument in herd health diagnosis. *Deutsche Tierärztliche Wochenschrift* 111, 458–462.

- Krajcarski-Hunt, H., Plaizir, J.C., Walton, J.P., Spratt, R., McBride, B.W., 2002. Effect of subacute ruminal acidosis on in situ fiber digestion in lactating dairy cows. *Journal of Dairy Science* 85, 570–573.
- Krause, K.M., Oetzel, G.R., 2005. Inducing subacute ruminal acidosis in lactating dairy cows. *Journal of Dairy Science* 88, 3633–3639.
- Krehbiel, C.R., Britton, R.A., Harmon, D.L., Wester, T.J., Stock, R.A., 1995. The effect of ruminal acidosis on volatile fatty acid absorption and plasma activities of pancreatic enzymes in lambs. *Journal of Animal Science* 73, 3111–3121.
- Kutas, F., 1965. Determination of net acid-base excretion in urine of cattle. *Acta Veterinaria Academiae Scientiarum Hungaricae* 15, 147–153.
- Lachmann, G., Seffner, W., 1979. Zur Problematik der metabolischen Azidose des Wiederkäuers. *Monatshefte der Veterinär-Medizin* 34, 44–46.
- Lachmann, G., Siebert, H., 1980. Die Bestimmung des Säure-Basen-Status in den Erythrocyten und im Lebergewebe beim Rind. *Monatshefte der Veterinär-Medizin* 35, 384–388.
- Lachmann, G., Siebert, H., Schäfer, M., 1985. Untersuchungen zum intraerythrozytären Säure-Base-Status bei dekompensierter metabolischer Azidose und parenteraler Pufferapplikation. *Archiv der Experimentellen Veterinärmedizin* 4, 598–605.
- LeBlanc, S.J., Lissimore, K.D., Kelton, D.F., Duffield, T.F., Leslie, K.E., 2006. Major advances in disease prevention in dairy cattle. *Journal of Dairy Science* 89, 1267–1279.
- Lechowski, R., 1997. The influence of metabolic acidosis in new-born calves on biochemical profile of the liver. *Comparative Haematology International* 7, 172–176.
- Leedl, J.A.Z., Coe, M.L., Russel, A.F., 1995. Evaluation of health and ruminal variables during adaptation to grain-based diets in beef cattle. *American Journal of Veterinary Research* 57, 885–892.
- Longland, A.C., Cairns, A.J., 2000. Fructans and their implications in the aetiology of laminitis. In: *Proceedings of the third International Research Conference on Feeding Horses*. Dodson and Horrell, Ringstead, UK, pp. 52–55.
- Maekawa, M., Beauchemin, K.A., Christensen, D.A., 2002. Effect of concentrate level and feeding management on chewing activities, saliva secretion, and ruminal pH of lactating cows. *Journal of Dairy Science* 85, 1165–1175.
- Manson, F.J., Leaver, J.D., 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. *Animal Production* 47, 185–190.
- Markusfeld, O., 1987. Aciduria in the postparturient dairy Cow. *British Veterinary Journal* 143, 119–127.
- Martin, S.A., Dean, G.F., 1989. Use of genetically engineered bacteria may aid in prevention of acidosis in cattle. *Feedstuffs* 2, 17–18.
- Mertens, D.R., 1997. Creating a system for meeting the fiber requirements of dairy cows. *Journal of Dairy Science* 80, 1463–1482.
- Morgante, M., Stelletta, C., Berzaghi, P., Giansella, M., Andrighetto, I., 2007. Subacute rumen acidosis in lactating cows: an investigation in intensive Italian dairy herds. *Journal of Animal Physiology and Animal Nutrition* 91, 226–234.
- Mortensen, K., 1993. Laminitis in cattle. PhD. Thesis, Royal Veterinary and Agricultural University, Copenhagen, Denmark, p. 253.
- Mottram, T.T., Ditcham, W.J.F., Bolam, H., Short, L., Camnell, S., Beever, D.E., Hobbs, P.J., 2000. Dimethyl sulphide and methane in the breath of cows: methods of capture and quantification. Project report CR/1031/00/0225. Silsoe Research Institute, Bedfordshire, UK, 20pp.
- Mutsvanga, T., 2003. Sub-acute ruminal acidosis (SARA) in dairy cows. April 2003, <<http://www.omafra.gov.on.ca/english/Livestock/day-airy/facts/03-031.htm>> (accessed 10.10.06).
- Mwansa, P., Makarechian, M., Berg, R.T., 1992. The effect of level of concentrate in feedlot diets on the health status of beef calves. *Canadian Veterinary Journal* 33, 665–668.
- Nelson, A., 1996. On-farm nutrition diagnostics. *The Bovine Proceedings* 29, 76–85.
- Nocek, J.E., 1997. Bovine acidosis: implications on laminitis. *Journal of Dairy Science* 80, 1005–1028.
- Nocek, J.E., Kautz, W.P., 2006. Direct-fed microbial supplementation on ruminal digestion, health and performance of pre-and postpartum dairy cattle. *Journal of Dairy Science* 89, 260–266.
- Nocek, J.E., Allman, J.G., Kautz, W.P., 2002a. Evaluation of an indwelling ruminal probe methodology and effect of grain level on diurnal pH variation in dairy cattle. *Journal of Dairy Science* 85, 422–428.
- Nocek, J.E., Kautz, W.P., Leedle, J.A.Z., Allman, J.G., 2002b. Ruminal supplementation of direct fed microbials on diurnal pH variation and in situ digestion in dairy cattle. *Journal of Dairy Science* 85, 429–433.
- Nordlund, K.V., Garret, E.F., 1994. Rumenocentesis: A technique for collecting rumen fluid for the diagnosis of subacute rumen acidosis in dairy herds. *The Bovine Practitioner* 28, 109–112.
- Nordlund, K.V., Garrett, E.F., Oetzel, G.R., 1995. Herd-based rumenocentesis: a clinical approach to the diagnosis of subacute rumen acidosis. *Compendium on Continuing Education for the Practicing Veterinarian – Food Animal* 17, S48–S56.
- Nørgaard, P., 2004. Use of image analysis for measuring particle size in feed, digesta and faeces. In: *Proceedings from the 10<sup>th</sup> International Symposium on Ruminant Physiology*, Copenhagen, Denmark, pp. 579–585.
- Oba, M., Allen, M.S., 2003. Effects of corn grain conservation method on feeding behaviour and productivity of lactating cows at two dietary starch concentration. *Journal of Dairy Science* 86, 174–183.
- Oetzel, G.R., 2000. Clinical aspects of ruminal acidosis in dairy cattle. In: *Proceedings of the 33rd Annual Convention of the American Association of Bovine Practitioners*, Rapid City, USA, pp. 46–53.
- Oetzel, G.R., 2003. Subacute ruminal acidosis in dairy cattle. *Advances in Dairy Technology* 15, 307–317.
- Olson, J.D., 1991. Relationship of nutrition to abomasal displacement and parturient paresis. *The Bovine Practitioner* 26, 88–91.
- Olsson, G.C., Bergsten, C., Wiktorsson, H., 1998. The influence of diet before and after calving on the food intake, production and health of primiparous cows, with special reference to sole haemorrhages. *Journal of Animal Science* 66, 75–86.
- Owens, F.N., Secrist, D.S., Hill, W.J., Gill, D.R., 1998. Acidosis in cattle: a review. *Journal of Animal Science* 76, 275–286.
- Penner, G.B., Beauchemin, K.A., Mutsvanga, T., 2006. An evaluation of the accuracy and precision of a stand-alone submersible continuous ruminal measurement system. *Journal of Dairy Science* 89, 2132–2140.
- Plaizir, J.C., Martin, A., Duffield, T., Bagg, R., Dick, P., McBride, B.W., 1999. Monitoring acidosis in the transition cow. *Journal of Dairy Science* 82 (Suppl. 1), 110.
- Ras, A., Janowski, T., Zdunczyk, S., 1996. Einfluss subklinischer und akuter Azidose ante partum bei Kühen auf den Graviditätsverlauf unter Berücksichtigung der Steroidhormonprofile. *Tierärztliche Praxis* 24, 347–352.
- Rebhun, W.C., 1995. Abdominal diseases. In: *Rebhun, W.C. (Ed.), Diseases of Dairy Cattle*. Williams and Wilkins, Baltimore, p. 530.
- Roby, K.A., Chalupa, W., Orsini, J.A., Elser, A.H., Kronfeld, D.S., 1987. Acid-base and electrolyte balance in dairy heifers fed forage and concentrate rations: effects of sodium bicarbonate. *American Journal of Veterinary Research* 48, 1012–1016.
- Rossow, N., Horvath, Z., 1988. In: *Rossow, N., Horvath, Z. (Eds.), Innere Krankheiten der Haustiere, Band II, Funktionelle Störungen*. Jena: Fischer, p. 246.
- Ruegg, P.L., 1996. Investigating herd problems and production on dairy farms. *The Bovine Proceedings* 29, 96–101.
- Sandholm, M., 1974. A preliminary report of a rapid method for the demonstration of abnormal gammaglobulin levels in bovine whole blood. *Research in Veterinary Science* 17, 32–35.
- Sarashina, T., Ichijo, S., Takahashi, J., Osame, S., 1990. Origin of abomasum gas in the cows with displaced abomasum. *Japanese Journal of Veterinary Science* 52, 371–378.
- Schwartzkopf-Genswein, K.S., Beauchemin, K.A., McAllister, T.A., Gibb, D.J., Streeter, M., Kennedy, A.D., 2004. Effect of feed delivery

- fluctuations and feeding time on ruminal acidosis, growth performance, and feeding behaviour of feedlot cattle. *Journal of Animal Science* 82, 3357–3365.
- Shaver, R.D., 1997. Nutritional risk factors in the etiology of left displaced abomasum in dairy cows: a review. *Journal of Dairy Science* 80, 2449–2453.
- Shaver, R., 2005. Feeding to minimize acidosis and laminitis in dairy cows. In: Proceedings of the seventh Western Dairy Management Conference, March, Reno, NV, US, pp. 157–166.
- Shu, Q., Gill, H.S., Hennessy, D.W., Leng, R.A., Bird, S.H., Rowe, J.B., 1999. Immunization against lactic acidosis in cattle. *Research in Veterinary Science* 67, 65–71.
- Shu, Q., Gill, H.S., Leng, R.A., Rowe, J.B., 2000. Immunization with a *Streptococcus bovis* vaccine administered by different route against lactic acidosis in sheep. *The Veterinary Journal* 159, 262–269.
- Steen, A., 2001. Field study of dairy cows with reduced appetite in early lactation: clinical examination, blood and rumen fluid analysis. *Acta Veterinaria Scandinavica* 42, 219–228.
- Stock, R., 2000. Acidosis in cattle: an overview. In: Proceedings of the 33rd Annual Convention of the American Association of Bovine Practitioners, Rapid City, USA, pp. 30–37.
- Stock, R., Britton, R.L., 1996. Acidosis. *NebGuide*, University of Nebraska – Lincoln. October 1995 <<http://www.ianr.unl.edu/pubs/animaldisease/g1047.htm>> (assessed 27.11.99).
- Stone, W.C., 1999. The effect of subclinical rumen acidosis on milk components. In: Proceedings of the Cornell Nutrition Conference of Feed Manufacturers, Syracuse, N.Y. Cornell University, Ithaca, NY, USA, pp. 40–46.
- Stone, W.C., 2004. Nutritional approaches to minimize subacute ruminal acidosis and laminitis in dairy cattle. *Journal of Dairy Science (Suppl.)*, E13–E26.
- Svendsen, P., 1969. Etiology and pathogenesis of abomasal displacement in cattle. *Nordisk Veterinaer Medicin* 21 (Suppl.1), 1–60.
- Svensson, C., Bergsten, C., 1997. Laminitis in young dairy calves fed a high starch diet and with a history of bovine viral diarrhoea virus infection. *Veterinary Record* 140, 574–577.
- Tarleton, J.F., Holah, D.E., Evans, K.M., Jones, S., Pearson, G.R., Webster, A.J.F., 2002. Biochemical and histopathological changes in the support structures of bovine hooves around the first time of calving. *The Veterinary Journal* 163, 196–294.
- Thoenfer, M.B., Pollitt, C.C., van Eps, A.W., Milinovich, G.J., Trott, D.J., Wattle, O., Andersen, P.H., 2004. Acute bovine laminitis: a new induction model using alimentary oligofructose overload. *Journal of Dairy Science* 87, 2932–2940.
- Van Eps, A.W., Pollitt, C.C., 2006. Equine laminitis induced with oligofructose. *Equine Veterinary Journal* 38, 203–208.
- Wallace, R.J., Newbold, C.J., 1993. Rumen fermentation and its manipulation: the development of yeast cultures as feed additives. In: Lyous, T.P. (Ed.), *Biotechnology in the Feed Industry*. Alltech Technical Publications, Nicholasville, Kentucky, pp. 173–192.
- Wallace, R.J., Newbold, C.J., 1995. Microbial feed additives for ruminants. In: Fuller, R., Heidt, P., Rusch, V., van der Waaij, D. (Eds.), *Probiotics: Prospects of Use in Opportunistic Infections*. Institute for Microbiology and Biochemistry, Herborn-Dill, Germany, pp. 101–125 <[http://www.old-herborn-university.de/Literature/books/OHUni\\_book\\_8\\_article\\_9.pdf](http://www.old-herborn-university.de/Literature/books/OHUni_book_8_article_9.pdf)> (accessed 30.07.07).
- Ward, W.R., Murray, R.D., White, A.R., Rees, E.M., 1996. Blood biochemistry and nutrition of dairy cows. In: Proceedings of the 19th World Buiatrics Congress, Edinburgh, Scotland, pp. 348–352.
- Wells, S.J., Trent, A.M., Marsh, W.E., Williamson, N.B., Robinson, R.A., 1995. Some risk factors associated with clinical lameness in dairy herds in Minnesota and Wisconsin. *Veterinary Record* 136, 537–540.
- Williams, P.E.V., Tait, C.A.G., Innes, G.M., Newbold, C.J., 1991. Effects of the inclusion of yeast culture (*Saccharomyces Cerevisiae* plus growth medium) in the diet of dairy cows on milk yield and forage degradation and fermentation patterns in the rumen of steers. *Journal of Animal Science* 69, 3016–3026.
- Yang, W.Z., Beauchemin, K.A., 2006. Effects of physically effective fiber on chewing activity and ruminal pH of dairy cows fed diets based on barley silage. *Journal of Dairy Science* 89, 217–228.
- Zebelli, Q., Tafaj, M., Steingass, H., Metzler, B., Drochner, W., 2006. Effects of physically effective fiber on digestive processes and milk fat content in early lactating cows fed total mixed rations. *Journal of Dairy Science* 89, 651–668.