



# Technical Bulletin

## Ruminal Acidosis in Dairy Cattle

Rumen pH is determined by the dynamic balance between the intake of fermentable carbohydrates, buffering capacity of the rumen, and the rate of acid absorption from the rumen. Ruminal acidosis is a metabolic disorder that affects feedlot and dairy cattle. Acidosis in cattle is usually due to ingestion of large quantities of carbohydrate rich feed, which leads to excessive production and accumulation of acids in the rumen.

Acidosis in cattle can be present in two forms:

- (i) **Acute ruminal acidosis:** Drop in pH is severe and clinical signs are prominent. It is mostly seen in feedlot cattle.
- (ii) **Sub-acute ruminal acidosis (SARA):** Drop in pH is gradual and clinical signs are not apparent. SARA is common in dairy herds.

**Table 1: Differentiating Acute and Sub acute Ruminal Acidosis**

Parameters	Acute acidosis	Sub-acute acidosis
Clinical signs	Present	Absent
Mortality	Yes	No
Rumen pH	<5	5-5.5
Lactic acid	50-120 mM	0-5 mm
Volatile fatty acids	<100 mm	150-225 mm
Lactic acid producing bacteria	Increase	Increase
Lactic acid utilizers	Decrease	Increase
Ciliate protozoa	Decrease	Decrease
Unnoticed incidences	14%	44%
Duration of acidosis	<90 minutes in a day	111-180 minutes in a day

(Plaizier et al., 2008; Calsamiglia et al., 2008)



In dairy cattle, rumen pH fluctuates considerably between 0.5 to 1 pH unit during a 24-hour period.

Sub-acute ruminal acidosis (SARA) is defined as frequent and prolonged depression of the ruminal pH to values between 5 and 5.5. The low ruminal pH is caused by excessive accumulation of volatile fatty acids (VFAs) without persistent lactic acid accumulation and is often restored by the animal’s own physiological response. There are no apparent symptoms of SARA and mostly such cases remain untreated.

Sub-acute ruminal acidosis (SARA) can have severe economic implications on farm productivity. It is more common in dairy cattle in their early and mid-lactation periods. SARA detection in a dairy farm is difficult as it does not present any pathognomonic symptoms and manifestation of clinical signs are delayed.

Grain-based diets, which have a higher proportion of non-structural carbohydrates, high-quality fermentable forages like legumes, and lack of dietary physically effective fiber (peNDF), are the main causes of SARA.

**Table 2: Rumen Environment and Microbiological Profile**

Index	Ruminal ecosystem
Normal ruminal pH	6.5 - 6.8
Optimum temperature	39°C
Gas production	CO <sub>2</sub> : CH <sub>4</sub> (65: 35)
Volatile Fatty Acid profile	Acetate : Propionate : Butyrate (65 : 20 : 10)
Ruminal microbial population (Count per ml of rumen liquor)	Bacteria (1010-11), Protozoa (106), Fungi (103), Bacteriophage (109)
Ruminal bacteria	Bacteria are mostly Gram negative & obligate anaerobes. However, lactic acid producing bacteria are facultative anaerobes attached to the ruminal wall because some Oxygen is present in the periphery.
Ruminal protozoa	The majority are ciliate protozoa. The ruminal microbial population is decided by protozoa because protozoa feeds on bacteria.
Ruminal fungi	Mostly flagellated fungi. They penetrate substrate by breaking ligno-cellulosic bond of fiber diet.
Ruminal bacteriophage	It causes lysis of bacterial cells so that bacterial protein is easily available to animals as a source of amino acids.



# Role of Saliva in Buffering Mechanism of Rumen

The ruminal pH is in the range of 6.5-6.8 and fluctuates by  $\pm 2.5$  points depending on the type and frequency of feeding. The major contributors to the buffering action in the rumen are phosphate-bicarbonate buffer, urea and mucous (secreted in the saliva). Cattle produce about 200-300 L of saliva daily having 100-140 mEq of bicarbonates which is responsible for 30-40% of the buffering capacity in the rumen.

## Buffering Capacity of Feed

Forage with high buffering capacities resist large reductions in the rumen pH. Legumes tend to have a higher buffering capacity than grass and barley silage. If the diet is predominantly rich in grain, the feed's buffering action is significantly reduced which affects the rumen pH. Diets rich in concentrate favour the synthesis of propionate by the acrylate pathway, leading to the formation and accumulation of lactic acid in the rumen, causing a drop in pH. In severe cases, the rumen's muscular activity is affected, and atony may occur.

The rumen papillae which absorb and transport VFA from the rumen to the bloodstream, may erode. Thus, Gram-negative bacteria may leak into systemic circulation, causing septicaemia and giving rise to various disorders like ruminitis, rumen parakeratosis, metabolic acidosis, lameness, hepatic abscessation and pneumonia which may be fatal.

The pH reaches a minimum value in 2 to 3 hours post feeding and increases continuously until the next feeding. However, the pattern of pH is much more stable ( $\approx 6.5$ ) when the animal is maintained on ad-libitum hay.

The optimal pH for cellulolysis, proteolysis and deamination is between 6 and 7. Ruminal cellulolysis is totally inhibited at a pH less than 6.0 and dry matter digestibility decreases with decreasing pH levels.

## Etiology

SARA occurs when ruminal buffering is inadequate in contrast to the volatile fatty acid (VFA) production. This can be caused by different reasons:



### **(i) Fiber content in diet**

Excessive carbohydrate consumption in place of dietary fiber consumption can cause SARA. Long fiber particles (greater than 4 mm) encourage chewing, which increases salivation. Saliva has a buffering effect in the rumen due to its high sodium bicarbonate content and a high pH value.

### **(ii) Failure to adapt to rapid diet changes**

Dietary changes such as a shift from dry diets rich in roughage to early lactation diets rich in concentrates are responsible for development of acidosis. Rumen bacterial population and rumen papillae need time to be ready to digest large amounts of carbohydrates and absorb large amount of VFAs. In general, major causes for sub-acute ruminal acidosis are lack of coarse fiber and/or excess concentrate in the diet, feed sorting, rapid diet changes and engorging (Plaizier et al., 2006).

## **Relationship Between Acidosis and Laminitis**

Laminitis is the most common clinical sign of sub-acute ruminal acidosis (SARA) in cattle. It causes economic loss, impaired animal health and susceptibility to other diseases (Nelson and Cattell, 2000). Cattle fed with a diet high in concentrates are more prone to laminitis (Kelly and Leaver, 1990). SARA can cause release of certain vasoactive substances like histamine and lipopolysaccharide endotoxins into circulation by the disintegration of Gram-negative bacteria. It may cause injury to microvasculature of corium followed by hypoxia to limb extremities leading to laminitis (Plaizier et al., 2009). Hoof discoloration, ulcers, abscesses, sole haemorrhages and misshapen hooves have been observed due to SARA in cattle (Oetzel, 2000).

## **Relationship Between Acidosis and Milk Fat Concentration**

Ruminal pH is positively associated with milk fat concentration (Kolver and de Veth 2002). A milk fat: milk protein ratio of less than 1.15:1 may indicate a risk of acidosis. Therefore, monitoring bulk vat milk fat percentage may be a useful indicator of acidosis in dairy herds. The relationship between ruminal pH and milk fat concentration is not absolute and is influenced by the stage of lactation, dietary fat content and body fat mobilisation (Westwood and Lean 2001). A reduction in the concentration of milk fat should be considered as an indicator of low ruminal pH and rumen function tests should be initiated.



# Consequences of SARA

- Decreased milk production
- Less milk fat
- Decreased fibre digestion
- Ruminal atony
- Diarrhoea
- Lameness

Symptoms of acidosis start with limited cud chewing and a subsequent drop in productivity. Some other significant changes which may develop because of acidosis are:

## (i) Rumenitis

Accumulation of VFA's like butyrate, propionate and lactate may be involved in the pathogenesis of rumenitis. Parakeratosis results from acute acidic conditions also affecting the long-term absorption capacity of the ruminal mucosa and making it susceptible to the entry of Gram-negative bacteria like *Fusobacterium necrophorum*. The bacteria can also migrate to liver as emboli, leading to the rumenitis liver abscess complex.

## (ii) Abomasal displacement

Abomasal displacement may be caused by increased ruminal gas and VFA flux, which is a result of low functional fiber levels in the diet.

## (iii) Bloat

Bloat is a condition where the release of macromolecules like mucopolysaccharides and unknown macromolecules from Gram-negative bacteria give rise to the formation of a static foam leading to a drop in pH and accumulation of gas.



**Table 3: Changes observed during Acidosis**

Indicators and Risk parameters	Common Observations
Animal Performance	Impaired rumen fermentation in lactating dairy cattle may result in lower milk production and lower milk fat.
Rumination	Reduced rumen motility during acidosis indicates absence of rumination activity.
Locomotion	Increased rumen histamine production and bacterial endotoxin release can cause lameness.
Manure Consistency	Liquid faeces indicate poor rumen efficiency because of a higher passage rate, an unbalanced diet and decreased gut fermentation.
Undigested Grain	Undigested/ partially digested grains in the faeces due to poor rumen efficiency

## Preventive Measures

### (i) Adequate fiber

Under circumstances where green fodder is limited, it is recommended that good-quality cereal and legume hays should be provided to prevent acidosis. It is important to ensure adequate and uniform distribution of forage amongst the herd. The amount of forage in the diet should be calculated to provide more than 32% NDF, with a majority (>80%) being sourced from long forage.

### (ii) Rumen buffers and neutralising agents

As SARA is a muted condition, it displays symptoms which are delayed and hence makes its prevention difficult. Nevertheless, adequate nutrition and adaptation of microflora to the feed is crucial in preventing SARA incidences in the herd. Physically effective fiber (peNDF) in diets stimulates saliva production, and hence ruminal buffering, assisting in maintaining rumen pH. Exogenous preventive measures like buffers and direct fed microbials also provide an effective tool for monitoring and preventing SARA.

The mode of action and effects of specific dietary buffers and direct fed microbials (DFMs) are described below:



- **Sodium bicarbonate**

Sodium bicarbonate acts as a buffer in the same way as endogenous sodium bicarbonate found in saliva. Sodium bicarbonate works in an optimal pH range of 6.2 to 6.5 with a pKa value of 6.25. Apart from its buffering action, sodium bicarbonate also acts as source of sodium and provides a positive dietary cation-anion balance for lactating cows.

- **Magnesium oxide**

Magnesium oxide is classified as a slow-releasing neutralising agent because of its undefined pKa and relative insolubility in water (Erdman 1988). However, Erdman (1988) reported the effectiveness of magnesium oxide in raising rumen pH and milk fat percentage.

The acid-consuming capacity of magnesium oxide is between 41.9 and 49 mEq per day which is significantly higher than other buffers and neutralising agents such as sodium bicarbonate with 11.9 mEq per day (Schaefer, Wheeler et al. 1982). Water solubility, however, is variable and dependent on the particle size of the magnesium oxide product (Erdman 1988).

- **Sodium sesquicarbonate**

Sodium sesquicarbonate is a mixed crystal of sodium bicarbonate and sodium carbonate with a pH of 9.9 which is higher than Sodium bicarbonate. Dietary supplementation of Sodium sesquicarbonate decreases the molar proportions of butyrate and valerate. It improves milk fat and milk yield.

- **Zeolite**

Zeolite functions as an alkalizing agent and has a strong capacity for H<sup>+</sup> exchange at different pH ranges (Yong et al., 1990). The high affinity of zeolites for water and osmotically active cations may facilitate ruminal fermentation, and osmotic activity may regulate pH in the rumen by buffering against hydrogen ions of organic acids.

- **Calcium carbonate**

Calcium carbonate has a long-term buffering activity in the rumen. It has low water solubility and a high capacity to consume acids. Haaland et al. (1982) found that Calcium carbonate had the greatest effect on buffering capacity between pH 4.5 and 5.0. Rumen ammonia and normal VFA concentrations were not affected with the addition of limestone (a source of Calcium carbonate) to the diet.



- **Direct Fed Microbials (DFMs) and Yeast**

DFMs and Yeast prevent lactate accumulation and allow better fiber digestion by improving the reducing conditions of rumen and stimulation of fibrinolytic bacteria. Supplementation of DFMs and Yeast enhance conversion of lactate to propionate which enables ruminal pH stabilization. Nocek, and Kautz (2006) showed in a study that three different organisms (*Enterococcus faecium*, *Lactobacillus plantarum*, *Saccharomyces cerevisiae*) administered at 10<sup>5</sup> cfu/ml stabilized rumen acidity and improved digestion.

## Conclusion

Acidosis is a much more complex condition than a simple drop in ruminal pH. In dairy cattle, sub-acute ruminal acidosis (SARA) is a major threat to the productivity of the herd. Apart from dietary management, prevention of SARA through addition of alkalizing or buffering agents is important for profitable dairy farming.