Physiological, biochemical and histopathological effects of fermentative acidosis in ruminant production: a minimal review

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Abstract

Rumen acidosis is increasingly recognized as a significant disorder in ruminants that increases the morbidity and mortality of animals, especially for dairy cattle and sheep. Acidosis is not just D-lactate which disturbs the acid-base status and the severity of acidosis is related to many factors and not only due to the level of lactic acid production, resulting in difficulties in diagnosing acidosis. Therefore, an understanding of the physiological, biochemical, and histopathological effects of rumen acidosis is fundamental for developing effective methods of prevention and treatment of fermentative acidosis. The present review evaluates the physiology, biochemistry, and pathophysiology of fermentative acidosis as well as gives a conclusion and look-forward. The information will benefit the health and welfare of ruminants and contribute to modern systems of ruminant production.

Additional key words: acid accumulation; diagnosis; influence; livestock.

Resumen

Efectos fisiológicos, bioquímicos e histopatológicos de la acidosis fermentativa en la producción de rumiantes: una mini-revisión

Cada vez está más reconocido que la acidosis ruminal es un trastorno importante de los rumiantes que aumenta la morbilidad y la mortalidad de los animales, especialmente de vacas lecheras y ovejas. La acidosis no es sólo D-lac-tato, que perturba el estado ácido-base; la gravedad de la acidosis se relaciona con muchos factores y no sólo con el nivel de producción de ácido láctico, lo que presenta dificultades para el diagnóstico de la acidosis. Por lo tanto, para el desarrollo de métodos eficaces de prevención y tratamiento de la acidosis fermentativa, es fundamental una comprensión de los efectos fisiológicos, bioquímicos e histopatológicos de la acidosis ruminal. La presente revisión evalúa y presenta conclusiones de la fisiología, bioquímica y fisiopatología de la acidosis fermentativa. Esta información será beneficiosa para la salud y el bienestar de los rumiantes y contribuirá a los sistemas actuales de producción de rumiantes.

Palabras clave adicionales: acumulación de ácidos; diagnóstico; ganadería; influencia.

Introduction

When the ruminant ingests abruptly large amounts of readily fermentable carbohydrates (RFCs) or when the period of adaptation to RFCs is insufficient, RFCs are suddenly fermentated and volatile fatty acids (VFAs) are accumulated, resulting in the rumen pH to drop below 5.5 for a prolonged period and creating the condition of fermentative acidosis (Ding et al., 1996, 1997, 1998; Ding and Xu, 2003). In this condition, lactic acid-producing bacteria, such as Streptococcus bovis and Lactobacillus spp., are proliferated, leading to the accumulation of lactic acid, which is known as lactic acidosis (Gozho et al., 2005, 2006, 2007).

Although acidosis has drawn researchers’ attention for a long time, it remains a major health threat in ruminants. Krause and Oetzel (2006) pointed out that acidosis is still a major problem for the North American dairy industry and subacute ruminal acidosis (SARA) is increasingly recognized as a significant disorder in

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Abbreviations used: AGS (acidic gut syndrome), DMI (dry matter intake), FCs (readily fermentable carbohydrates), GGTP (gamma glutamine trans-peptidase), LPS (lipopolysaccaride endotoxin), SARA (subacute ruminal acidosis), VFAs (volatile fatty acids).
Acidosis and its primary causes

Usually, rumen pH tends to be relatively constant (normally at pH 7.5 or a bit lower), depending on what the animal eats and the time of the day. A relatively constant level of rumen pH is very important to maintain normal functioning of the rumen. The maintenance of the rumen pH at a relatively constant level depends on the balance between rates of acid production and absorption and the buffering capacity of the animal. Buffering capacity provided by buffers in the rumen and salivary glands plays a key role (Ding et al., 1996, 1997), though the respiratory and excretory systems also function in pH regulation via blood; for example, carbon dioxide ($CO_2$) is exhaled from the lung and excess $H^+$, $NH_3$, and $K^+$ are excreted in the urine (Rawn, 1989).

Normally, the production and excretion of acids are balanced to maintain systemic acid-base homeostasis. Ruminants are generally able to maintain ruminal pH levels within physiological limits through their own regulation of intake, endogenous buffer production, microbial adaptation, and VFA absorption (Krause and Oetzel, 2006; Bramley et al., 2008).

However, systemic acid-base homeostasis may be occasionally broken. For example, if the ruminant abruptly ingests large amounts of grain, starch or other feed that is rich in RFCs, much lactic acid will be formed and this is associated with a fall in pH in the rumen leading to fermentative acidosis, therefore, it is also known as ruminal acidosis (Nocek, 1997; Krause and Oetzel, 2006). Fermentative acidosis is considered to be due to faster and complete fermentation of RFCs in the rumen. Lactic acid accumulates only if its rate of production exceeds the rate of utilization or conversion to VFAs. This can result from either a proliferation of lactate producers (Gram-positive bacteria) or a failure of lactate utilizers (Gram-negative bacteria) to proliferate rapidly enough to utilize the increased quantity of the acid, which occurs when there is an accumulation of VFAs and a reduced pH (Rowe et al., 1989). A low level of ruminal pH reduces the number of species of bacteria in the rumen during SARA, although the metabolic activity of the bacteria remains very high (Garry, 2002).

An extensive range of carbohydrates has been reported to produce lactic acid either $in vivo$ or $in vitro$. Any feedstuff rich in soluble carbohydrates is capable of acting as a lactic acid precursor. In times of drought, there can be significant risk of acidosis for ruminants, where the animals are often given access to grain stubble after harvest. These ruminants usually do not go through any adaptation stage. A similar situation can occur when ruminants are introduced into crops, particularly brassicas, such as rapes ($Brassica chinensis$ L.), kales ($Brassica oleracea$ var. $acephala$ f. $tricolor$) or turnips ($Brassica rapa$ var. $rapa$), that are highly digestible; and also when ruminants selectively graze the most digestible material (RAGFAR, 2007; Walsha et al., 2009).

A rank order of grains and by-product feeds in terms of their lactic acid production potentials, based on results of fermentation and in the order of the greatest to the least potential to produce lactic acid, showed that steam-flaked barley $= $ barley $= $ wheat $> $ citrus pulp $> $ beet pulp $= $ corn $> $ high moisture corn $= $ sorghum grain (Cullen et al., 1986). Furthermore, steam flaking, rolling, popping or other processing methods increase the surface area or gelatinized starch and make carbohydrates more rapidly fermentable, thereby increasing the potential for fermentative acidosis (Huntington, 1988).
Acidosis is a condition with severe consequences for the animal. The clinical signs of acidosis vary depending on the severity of the disease (Braun et al., 1992) and can be either acute, posing a life-threatening situation, or subacute (chronic), resulting in reduced feed consumption and weight gain. Repeated bouts of SARA can damage the surface of the rumen wall (Krause and Oetzel, 2006). Once the rumen wall is damaged, bacteria and toxins produced by bacteria can enter the portal circulation, causing liver abscesses and an inflammatory response (Gozho et al., 2005, 2006, 2007). Clinical signs of acidosis in sheep are similar to those in cattle. Sheep differ from cattle in rumen function and feed tends to pass quicker through the rumen than in cattle. Sheep tend to have a higher incidence of intestinal disturbances compared to cattle, e.g. enterotoxaemia (Pulina, 2004).

The adverse effects of acidosis are attributed to the local effects of pH and excess lactic acid in the rumen (Juhász and Szegedi, 1968) because both D- and L-lactic acid are absorbed across the rumen wall and depress blood pH. This effect was also thought to be exacerbated by the slower clearance by the body of D-lactic acid originated from digestive microbes and is thus referred to as “D-lactic acidosis” (Blood and Henderson, 1963). However, the concentrations of D-lactate in blood during episodes of rumen lactic acidosis are normally very much lower than that of L-lactate (Godfrey et al., 1992). It is possible that L-lactic acid predominates in the fermentative production (Ryan, 1964) or both D- and L-lactic acid are produced in approximately equal amounts by digestive microbes (Cori and Cori, 1929). On the other hand, only L-lactate is produced by metabolism of animal tissues and the conversion from propionate by the epithelium of digestive tract (Giesecke and Stangassinger, 1980). Therefore, the concentration of L-lactic acid is usually higher than that of D-lactic acid (Ganter et al., 1993). Cori and Cori (1929) and Dunlop and Hammond (1965) found that there was no detectable difference in the absorption rate between the two isomers and suggested that the peak of L- and D-lactic acid into the blood should occur at the same time. However, liver tissue was found to be able to synthesize carbohydrate from D-lactic acid but scarcely any from L-lactic acid (Meyerhof and Lohmann, 1926). L-lactic acid is utilized 4 times more slowly in the rat than D-lactic acid because D-lactic acid can be deposited as liver glycogen (Cori and Cori, 1929).

The severity of acidosis is related to many factors and not only lactic acid production (Lindinger and Heigenhauser, 2008). It is important to emphasize the contribution of all acids to the disruption of the acid-base status during acidosis (Harmon, 1983). Rowe (1997) suggested that a new condition, “acidic gut syndrome” (AGS), should be recognized. This is characterized by the accumulation of acids in the digestive tract at concentrations that have not previously been considered harmful to animals or humans. The detrimental effects initiated by lactic acid and low pH may be mediated through direct action on the gut wall, through the production of bacterial endotoxin, through the combination of acids and endotoxins, or through other factors.

Adverse effects of acidosis include physiological, biochemical, and histopathological aspects which are discussed below.

Physiological effects of acidosis on the animal

A series of physiological effects may happen in acidotic animals and the effects may relate to many factors. Decreased dry matter intake (DMI) is an obvious cause for acidosis and has been used as a clinical sign to diagnose SARA (Kleen et al., 2003; Oetzel, 2003). A lowered feed intake was observed during periods of SARA (Olsson et al., 1998; Brown et al., 2000; Krajcarski-Hunt et al., 2002; Gozho et al., 2005, 2006; Fairfield et al., 2007). Reasons for the feed intake depression can include reduced fiber digestibility (Plaizier et al., 2001; Krajcarski-Hunt et al., 2002) and increases in VFAs, especially propionate, and in the osmolarity in the rumen (Allen, 2000; Gozho et al., 2006; Khafipoor et al., 2006, 2007; Enemark, 2009). Inflammation of the ruminal epithelium (rumenitis) could also play a role in depressing feed intake following ruminal acidosis (Krause and Oetzel, 2006). Rumenitis was caused by grain-induced SARA (Gozho et al., 2005, 2006, 2007) and reduced feed intake in the cow (Weingarten, 1996; Andersen, 2000).

The severity of rumenitis caused by SARA most likely depends on conditions in the rumen, such as pH, composition of microbial populations, existing challenges to the barrier function, and inflammation of the rumen mucosa. The severity of clinical signs appears to reach a maximum between 12 and 36 hours in sheep (Patra et al., 1995). If clinical signs are more severe, it may transform into acute acidosis.

The reduction in fiber digestion that occurs during SARA is most likely the result of the acid sensitivity
of the fibrolytic rumen bacteria. These bacteria generally cannot tolerate a rumen pH below 6.0, which will reduce their numbers in the rumen and, subsequently, reduce fiber digestion (Shi and Weimer, 2002).

Diarrhea has been associated with SARA in dairy herds by many scientists (Nocek, 1997; Kleen et al., 2003; Oetzel, 2003). Feces from cows with SARA may appear brighter and more yellowish than the feces of cows without SARA (Kleen et al., 2003). This is because fecal consistency is determined by the movement of water into the digestive tract and as a result of SARA, digesta is hypertonic compared to plasma (Huber, 1976). In SARA cases, the feces appear foamy with gas bubbles, and contain more than normal amounts of undigested fiber or grain (Hall, 2002). Foamy feces and diarrhea suggest extensive hindgut fermentation which has also been associated with SARA (Nordlund et al., 2004). Rumen stasis, as a result of low rumen pH, may also allow for the accumulation of free gas (Rebhun, 1995). However, the blood gas parameters are not notably affected in cases of chronic, metabolic acidosis (Lachmann and Siebert, 1980). Because there is an insufficient ruminal fiber mat, fiber is not effectively retained in the rumen (Hall, 2002). Nordlund et al. (1995) reported on herds with soft faeces that contained substantial amounts of undigested feed particles. Intermittent diarrhea and the presence of undigested particles indicate inadequate digestion and fast passage of feed.

Cows affected with SARA may develop caudal vena cava syndrome which is presented clinically as hemoptysis and peracute deaths due to massive pulmonary hemorrhage (Nordlund et al., 1995). In these cases, septic emboli from liver abscesses cause foci of pulmonary infection that ultimately invade pulmonary vessels and cause their rupture (Radostits et al., 1994; Rebhun, 1995). Therefore, SARA is associated with liver abscesses (Dirksen et al., 1985; Nocek, 1997; Kleen et al., 2003; Oetzel, 2003). These liver abscesses are caused by translocation of rumen bacteria, such as Fusobacterium necrophorum and Arcanobacterium pyogenes, to the portal blood as a result of decreased barrier function of the rumen mucosa (Dirksen et al., 1985; Nocek, 1997; Kleen et al., 2003). These bacteria can spread from the liver to other organs, such as the heart, lungs, and kidneys (Nordlund et al., 1995; Nocek, 1997; Kleen et al., 2003; Oetzel, 2003). The reduced barrier function of the rumen mucosa is associated with rumenitis and lesions in the rumen mucosa due to the low rumen pH that occurs during SARA (Nocek, 1997; Andersen, 2000; Kleen et al., 2003). Rumenitis also leads to a high incidence of liver abscesses (Church, 1993) in feedlot cattle.

If the treatment is not appropriate, acute acidosis can kill ruminants in the short term, and SARA can also kill ruminants through dehydration due to excessive water loss in the faeces (scouring). If animals survive this shock, rumenitis permits microbial contamination of the blood and animals may die after some weeks. Culling rate and number of inexplicable deaths within herds with SARA may be unacceptably high (Nordlund and Garret, 1994).

**Biochemical effects of acidosis on the animal**

Acidosis can result in a lot of biochemical effects in the ruminant. Rumen pH will fall when organic acids, such as VFAs and lactic acid, accumulate in the rumen, and if rumen buffering cannot keep pace with the accumulation of these acids. Because of rapid fermentation of RFCs in the rumen, increased concentration of lactic acid and VFAs and lowered pH have been reported in the rumen liquor of acidotic sheep (Phillipson and McAnally, 1942), cattle (Balch and Rowland, 1957) and goats (Sen et al., 1982). Many of the mechanisms by which depression of rumen pH compromises cow health are not well understood. Recent research has suggested that the production of immunogens in the rumen, such as lipopolysaccharide endotoxin (LPS) and histamine, reduction of the barrier function of the rumen, and translocation of immunogens from the rumen are part of these mechanisms (Plaizier et al., 2008). Rumen pH depression triggers the release of vasoactive substances, such as histamine and LPS, which damage the capillaries of the lamellae in the foot, thus causing hemorrhage, inflammation, and lameness (Nocek, 1997). On the other hand, grain-induced SARA increased lysis of Gram-negative bacteria, resulting in an increase of acute phase proteins, haptoglobin and serum amyloid-A, in cow plasma and milk, and stimulated translocation of LPS into the blood (Gozho et al., 2005, 2006, 2007). Moreover, Plaizier et al. (2008) reported that the reduction of milk fat and milk protein applied to an entire lactation.

Histamine is an important regulator of feed and water intake in cattle (Lecklin and Tuomisto, 1990; Rossi et al., 1998). Recent experiments have demonstrated that the permeability of rumen epithelia to
histamine is low when ruminal pH is normal, but absorption increases significantly when pH declines (Aschenbach et al., 2000). Thus, declining ruminal pH predisposes animals to increased absorption of histamine. Increased sugar levels in blood may be an indication of internal metabolism initiated by acidosis in the rumen and mediated via toxic substances, such as lactate and histamine (Nikolov, 1996). High levels of histamine are involved in the pathogenesis of bronchial constriction (Vignola et al., 1997) and cardiovascular shock (Nakamura et al., 1997).

Prentice (2000) reported a rise in serum haptoglobins (acute-phase inflammatory proteins) and an increase in the prevalence of ruminal biopsy samples with histological evidence of rumenitis when Holstein steers were fed to a low target ruminal pH.

However, Gozho et al. (2007) demonstrated that grain-induced SARA increased free LPS in the rumen, but not in peripheral blood, which disagrees with the hypothesis of Nocek (1997) that LPS damages the capillaries of the hoof. Intravenous administration of LPS causes immune activation, including the production of cytokines and increase in acute phase proteins in the peripheral blood (Werling et al., 1996; Waldron et al., 2003). Hence, the inflammation that accompanies grain-induced SARA could be the result of LPS translocation from the rumen to the liver. It is unclear if LPS is responsible for rumenitis, as low rumen pH can also lead to rumenitis (Nocek, 1997; Kleen et al., 2003; Oetzel, 2003). At present, the pathogenesis of rumenitis seems to be mediated by an increased production of VFAs, particularly butyrate and propionate, as well as a temporary rise in the ruminal lactate concentration and fluctuations in the osmolarity of the rumen fluid, which may be involved in the development of the rumenitis (Dirksen, 1985; Krehbiel et al., 1995).

The activities of some enzymes change in acidosis because the animal metabolism, environment and pH of the enzymes have changed. Elevated activities of amylase, creatinine phosphokinase and gamma glutamime trans-peptidase (GGTP) were revealed in serum enzyme profiles of acidotic sheep (Patra et al., 1996). The change in serum amylase may be attributed to change in carbohydrate metabolism (Kaneko, 1989) and lowering of rumen pH (Slyter, 1976). Creatinine phosphokinase activity is one of the most specific indicators of muscle damage (Kaneko, 1989) and hence increased activity of this enzyme may be a signal to muscle damage in acidosis. Increased activity of GGTP, a liver-specific enzyme, reflects hepatobiliary damage (Patra et al., 1996).

Grain-induced SARA causes an increase in acute phase proteins in blood, which is an indicator of inflammation. This has been shown by several studies (Gozho et al., 2005, 2006, 2007). Increased concentrations of urea and total protein in the blood have also been observed in experimentally acidotic sheep (Juhász and Szegedi, 1968). Chikagwa-Malunga et al. (2009) reported that increasing the supplementary level of Mucuna sp. (a leguminous used in animal feeding) with relatively high crude protein increased (p < 0.05) level and efficiency of microbial protein synthesis, ruminal fluid acidity, total VFA concentration, decreased (p < 0.05) coccidian oocyst scores, and tended (p < 0.10) to increase N retention. Rumenitis might initiate the production of certain acute phase proteins which are produced in the liver and then released into blood (Gozho et al., 2005, 2006).

The change of rumen osmolarity can cause serious problems in the acidic animal. Many of the symptoms, such as diarrhea, haemoconcentration, and pronounced dehydration associated with the grain engorgement syndrome, have been shown to be attributable to an increase in osmotic pressure of the rumen and not due to the absorption of lactic acid or any other toxic factor (Patra et al., 1995). Osmotic pressures in excess of 500 mOsmol L⁻¹ were reported in ruminal fluid of a heifer experimentally engorged with wheat (Ahrens, 1967).

Excess CO₂ formed in the rumen during fermentation in the pathological process also exacerbates the symptoms through paralyzing the respiratory centre, a relative oxygen deficiency (hypoxia) and absolute accumulation of CO₂ in the tissue so that the affected animals die of asphyxia (Juhász and Szegedi, 1968).

Many other blood components also change in the animal with acidosis. Pyruvic acid, inorganic phosphate, and haematocrit value increased in sheep overloaded with RFCs, whereas blood Na⁺ and Cl⁻ concentration declined (Juhász and Szegedi, 1968). Excessive amounts of excreted phosphate were found in the urine of cows fed high grain diets (Enemark et al., 2004). Serum urea changing is another characteristic in acidotic animals. A significant increase in serum urea is an index of a decreased glomerular filtration rate in acidotic sheep, possibly due to renal damage or reduction in effective renal blood flow, and a fall in the arterial blood pressure, which results in subnormal renal function (Dunlop, 1972).
Histopathological effects of acidosis on the animal

There is a series of histopathological changes in different organs of acidic ruminants. The rumen shows denudation and broken continuity (Vestweber and Leipold, 1974). There is micro-vesiculation in the stratum lucidum with infiltration of polymorphs in and around vesicles. A few vesicles coalesce to form larger ones. Mucosal papillae reveal infiltration of polymorphs and mononuclear cells. Ruminal papillae shows hydropic degeneration of epithelial cells that start in the deeper layer. With time, micro-vesicles increase in size and move towards the superficial layers with loss of integrity of epithelium in many areas. At the same time, the liver is congested with evidence of hemorrhage in the parenchyma (Patra et al., 1995; Puntenney et al., 2003). Sinusoids are distended with red blood cells. There are peri-portal and peri-vascular mononuclear infiltration, degenerative and focal necrotic changes in hepatocytes (Patra et al., 1995).

Ruminal epithelial cells are not protected by mucus and are vulnerable to the chemical damage by acids. Thus, low ruminal pH can lead to rumenitis (Nocek, 1997; Kleen et al., 2003; Oetzel, 2003) and eventually to ruminal parakeratosis, erosion and ulceration of the ruminal epithelium (Garry, 2002). Rumenitis is the fundamental lesion of SARA and initiates chronic health problems. Once the ruminal epithelium is inflamed, bacteria may colonize the papillae and leak into portal circulation. These bacteria may cause liver abscesses, which sometimes cause peritonitis around the site of the abscess. If the ruminal bacteria clear the liver (or if bacteria from liver infections are released into circulate on), they may colonize the lungs, heart valves, kidneys or joints (Nordlund et al., 1995; Nocek, 1997). The resulting pneumonia, endocarditis, pyelonephritis and arthritis are difficult to diagnose ante-mortem (Krause and Oetzel, 2006). Parakeratosis may affect VFA absorption in the long term when it occurs as a consequence of acute increased lactate production caused by induced clinical acute rumen acidosis (Krehbiel et al., 1995). Mucosal lesions in rumenitis may serve as an entrance for Fusobacterium necrophorum, and, more rarely, Acanobacterium pyogenes, with subsequent colonization in the submucosa in severe cases of SARA. 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; characterized by bloody, foaming expectorate around the muzzle and nostrils. Subacute ruminal acidosis may also be associated with sole ulcers (Nocek, 1997). Feed-induced acidosis has been shown to result in abomasal ulceration in goats, however, the pathogenesis is not yet fully understood (Aslan et al., 1995).

SARA may be associated with laminitis and subsequent hoof overgrowth and sole abscesses. Laminitis (pododermatitis aseptica diffusa) is defined as the inflammation of the dermal layers inside the hoof and has been associated with systemic metabolic insults (Nocek, 1997). Laminitis generally do not appear until weeks or months after the bout of ruminal acidosis that caused them. The true mechanistic causes of laminitis in dairy cows are assumed to be multi-factorial, the precise mechanism by which SARA increases the risk for laminitis has not been characterized (Nocek, 1997; Ruegg, 2000). Laminitis in horses can be caused by metalloproteinase enzymes that destroy the lamellar detachment (Kyaw-Tanner and Pollitt, 2004). The information from equine studies has shown that an exotoxin released from S. bovis may activate metalloproteinase enzymes and lead to the separation of equine lamellar explants (Mungall et al., 2001). These results could indicate a link between damage to the integrity of the hoof and S. bovis, which might proliferate during bouts of SARA. This disorder is prevalent in dairy cows during early lactation and in beef cattle in feedlots (Underwood, 1992).

The severity and the signs of acidosis depend on the animal. Rumen acidosis (rumen pH < 5.4) was recorded in some goats exposed to ad libitum concentrates in the post-partum period, however, no clinical signs of acidosis or laminitis were observed (Mgasa and Arnbjerg, 1993) because goats can suffer from rumen acidosis without suffering from metabolic acidosis (Giger-Reverdin et al., 2006). This appears to be because adult goats have a stable rumen structure and are resistant to laminitis even when exposed to high amounts of concentrates (Magasa and Arnbjerg, 1993).

Conclusion and look-forward

Summarily, the adverse effects of acidosis in ruminants are complex and a multidisciplinary approach is needed for an understanding, prevention and treatment
of acidosis. Currently, careful management and gradual adaptation to diets high in RFCs are the most important to prevent fermentative acidosis.

Improved diagnostic measures need to be developed and further research is required to define roughage/fiber requirements for feedlot animals. A computer model may have a potential role to predict the profile of nutrients available, lactic acid production and the efficacy of treatment of acidosis. Many genes may participate in various processes leading to the adaptation and restoration of normal systemic acid-base and electrolyte homeostasis. Ruminal acidosis will become an even more common problem as genetic progress and better feeding management allow cows to consume more feed.

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