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Ruminal Acidosis: A Systematic Review

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ABSTRACT

Ruminal acidosis is a metabolic disorder that affects ruminants, including cattle, sheep, and goats, due to a pH imbalance in the rumen. The rumen has a diverse population of microorganisms involved in carbohydrate metabolism, with anaerobic microorganisms in the rumen and cecum playing a crucial role. During healthy rumen metabolism, microbial fermentation produces volatile fatty acids, including acetic acid, propionic acid, and butyric acid. Excessive intake of feeds with high soluble carbohydrate content can cause ruminal acidosis by altering the ratio of volatile fatty acids produced through microbial fermentation, which in turn changes the rumen pH. Acidosis is defined as a decrease in the alkalinity of body fluids relative to their acid content. The pH of body fluids may or may not decrease during acidosis, depending on the degree of bicarbonate compensation. Impaired central nervous system function can occur even if blood pH remains stable due to low bicarbonate concentrations, which are buffered by bicarbonate. While a blood pH below 7.35 is required for a clinical diagnosis of acidosis, other clinical signs such as ruminal pH, anorexia, variable feed intake, diarrhea, and lethargy are commonly used to diagnose acidosis in beef cattle.

INTRODUCTION

Carbohydrates are fermented by anaerobic microorganisms in the rumen and cecum, resulting in the production of volatile fatty acids (VFA) and lactate. Steers fed feedlot diets have been measured to produce more than 55 mol of VFA per day (Azizi et al., 2020). These organic acids are absorbed by ruminants from the rumen and/or cecum for metabolism by tissues. An increase in carbohydrate supply leads to an increase in total acid supply and the prevalence of lactate in the mixture.

Lactate is typically found in low concentrations in the digestive tract. However, sudden increases in carbohydrate supply can cause lactate to accumulate, resulting in ruminal concentrations that can sometimes reach 100 mM. This metabolic disorder has been referred to as 'D lactic acidosis' by Dunlop, (1972) and has been associated with overeating, acute impaction, grain blockage, founder, and grain overload. The term 'acidosis' refers to digestive disorders of the rumen and intestines in ruminants. It is typically categorized into acute, chronic (or subclinical), and subconscious types. Acute acidosis is an overt disease that occurs when easily fermented carbohydrates are consumed in quantities sufficient to lower digestive pH. Chronic acidosis, on the other hand, results in reduced feed intake and performance, but animals may not exhibit any symptoms. The clinical diagnosis of acidosis relies on measuring the acidity of ruminal or blood samples. A ruminal pH of 5.6 or lower is considered chronic acidosis, while a pH of 5.2 or lower is considered acute acidosis, according to Jennings et al., (2018). The variation in feed intake from one day to the next was used as an indicator of subclinical or chronic acidosis (Sanchez et al., 2021). This is based on the idea that increased day-to-day variability in feed intake by individual animals is associated with feeding acidic diets (DeClerk et al., 2020).

The lack of a comprehensive study on acidosis and the lack of a study in which general literature information was given in a collective manner constituted the subject of this study and a systematic review study on acidosis in cattle was carried out by utilizing the basic literatures. The aim of the study was to create a general review framework of the mechanism of acidosis.

MATERIALS AND METHODS

The study was prepared following the PRISMA 2020 Systematic Review Guidelines (Page et al., 2021). The words acidosis, nutritional diseases in ruminants, asidosis in ruminants, ruminal asidosis, etiology of asidosis were searched in Google Scholar, Elsevier and Wiley in Turkish and English. The information obtained from 1 printed

book chapter, 1 congress paper, 3 board reports, and 35 research articles and reviews were used in the study from 1991 to 2024. Related information is summarized in the flow diagram in Figure 1.

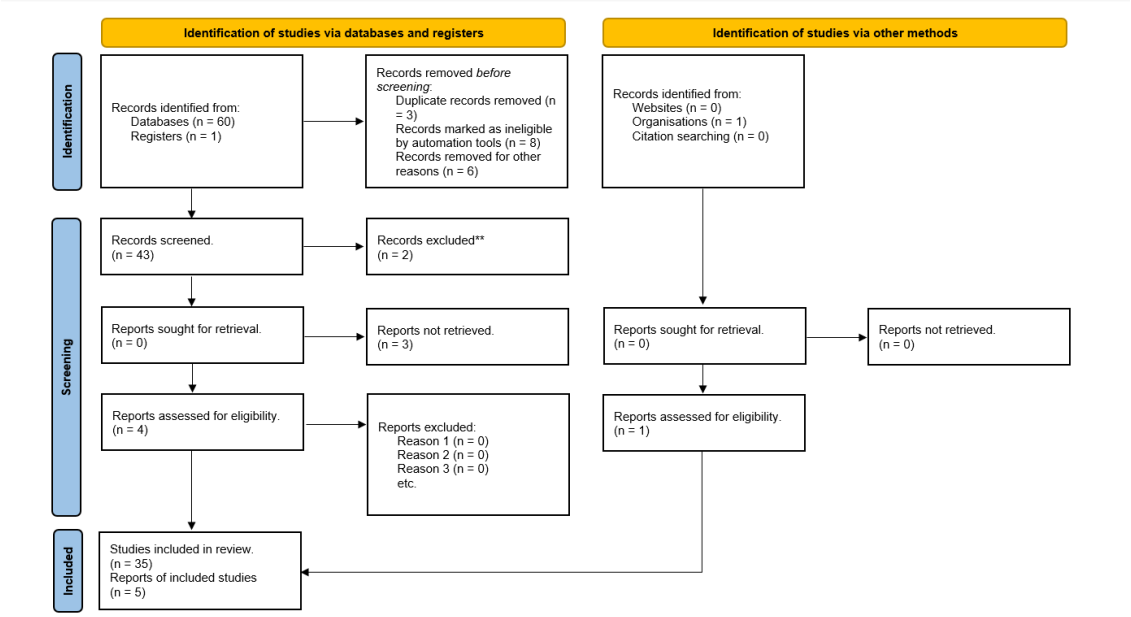

Figure 1. From: Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al., The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ 2021; 372: n71. doi: 10.1136/bmj.n71. For more information, visit: http://www.prisma-statement.org/

Etiology of Acidosis

Acidosis in ruminants is usually caused by overconsumption of large amounts of starch or rapidly fermentable carbohydrates. This usually occurs when animals are first adapted to a high-concentrate ration and/or when animals switch from bulk feeding to a chemostatic intake arrangement. Additionally, acidosis can occur when grazing animals are given a large amount of a starch-rich supplement (Owens et al., 1998; Millen et al., 2016; Monteiro and Faciola, 2020).

Starch Concentration and Glucose Conversion: Depending on the grain source, processing and type of starch, the rate at which starch is converted to glucose varies. Some grain sources and cereal varieties extract starch more readily, which is preferred by distilleries, and are likely to be hydrolyzed to glucose faster than others. In the endosperm of milo and maize, protein-embedded starch granules are exposed to fewer surfaces for microbial attack. Heat and pressure treatment cause the starch granules to expand into thin sheets, which ferment rapidly. According to Vieria-Neto et al., (2021), heat and pressure treatment of grains, reduction of particle size, and storage at high moisture levels increase starch availability and the likelihood of acidosis. Certain strains of microbes bind to grain particles and release glucose from starch granules. Flake quality can be measured using various methods, such as test weight, birefringence, and rate of gas production during incubation with yeast or rumen contents, as well as glucose or maltose release during incubation with amyloglucosidase or amylase. These methods should reflect the degree of starch exposure and/or fermentation rate. To achieve maximum energy efficiency, it is desirable to have a high degree of fermentation. However, to prevent acidosis, a slow fermentation rate is preferred. Unfortunately, the digestion rate and extent are usually positively correlated across different grain sources and processing methods.

Conventionally, glucose has not been considered an important metabolic intermediate in the rumen because rumen concentrations are normally extremely low. However, many studies found that glucose concentrations in the rumen can exceed 160 mg/dL, which is higher than the concentration found in blood (Sommai et al., 2020; Klotz et al., 2023; Deimeters et al., 2024). In one of acidosis studies, rumen glucose levels exceeded 1,400 mg/dL. Glucose is released from starch by amylase. However, it is unclear whether the higher concentration of glucose is due to faster hydrolysis or a reduction in the rate of glucose utilization by rumen microorganisms (Deimeters et al., 2024). Ruminal pH changes are shown in Figure 2.

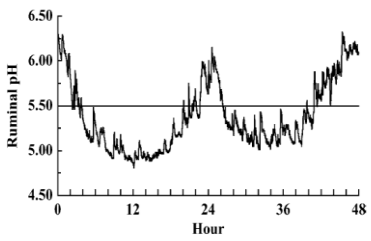

Figure 2. Ruminal pH over a 48-h period in a steer adapted to a 92.5% concentrate diet based on dry rolled maize fed once daily at 0 and 24 h. (Ruminal pH was monitored with a submersible pH electrode suspended from the plug of the ruminal cannula and recorded every minute (data from Cooper et al., (1998)

The presence of free glucose in the rumen can have at least three side effects. Firstly, non-competitive rumen bacteria can grow rapidly when high amounts of glucose are provided. Streptococcus bovis, which thrives only when free glucose is present, is the main cause of lactic acidosis (Chen et al., 2016). However, concentrations of this organism in the rumen of cattle fed high concentration feeds are very low (Semwogerere et al., 2020). Other bacteria that are directly involved in starch fermentation may be more significant sources of lactate. In fact, lactate accumulates faster from starch than from glucose in vitro. Additionally, other opportunistic microorganisms, such as coliforms and amino acid-decarboxylating bacteria, can thrive in the rumen of cattle fed concentrate feeds (Eger et al., 2018; Semwogerere et al., 2020) and produce or release endotoxins or amides (e.g., histamine) during lysis (Beauchemin, 2018). Thirdly, glucose that is released from starch increases the osmolality of the rumen contents. This increase in osmolality worsens acid accumulation in the rumen by inhibiting VFA absorption.

Limiting Starch and Glucose Intake: To prevent acidosis, two common management practices are diluting the ration with roughage or modulating starch intake. Ration-derived roughage reduces feed rate and meal size, while increasing the concentration of dry roughage increases chewing time and saliva production. Increasing the degree of chewing can reduce the size of grain particles entering the rumen, which in turn can increase the rate of fermentation. However, it is important to note that the increased buffer input from saliva due to longer chewing time or ruminating can neutralize and dilute rumen acids. To reduce the starch content of the ration, consider using starch-extracted concentrates such as distillation or fermentation by-products and intermediates instead of cereal grains (Figure 3). Total feed intake can also be restricted by using a limited maximum intake feeding scheme, as described by Lawrance et al., (1995).

For experimental purposes, researchers typically induce acute acidosis by withholding feed for 12 to 24 hours and then feeding (or ruminally dosing) 150% of the normal daily feed ration. This demonstrates that increasing meal size can accelerate acidosis and suggests that daily variation in feed intake between days in an animal will increase the potential for acidosis. Regularity of intake is also recognized as a sign of subclinical acidosis. Fulton et al., (1979) observed that animals' feed intake was typically low after a bout of acidosis, indicating a cyclic pattern of feed intake reflecting repeated bouts of acidosis. Detecting such fluctuations in intake is easy when animals are fed individually. However, when feeding 20 or more animals together, it may be difficult to detect daily fluctuations in intake unless all animals experience acidosis simultaneously, such as during feed changes or mishaps during processing or mixing.

The effects of feed intake regulation on acidosis were investigated in trials conducted in New Mexico, California, and Nebraska (Galyean and Goetsch, 1993; Zinn et al., 1995; Cooper et al., 1999). Although altering daily feed intake slightly decreased feed efficiency and reduced performance in the New Mexico trial, animal health was largely unaffected. Many studies reported that the inclusion of monensin and monensin-tylosin combinations in the diet of feedlot cattle reduced the daily variation in feed intake (Dunlop,1972; Cooper et al., 1999; Beauchemin 2018; Klotz et al., 2023; Kachhadia et al., 2023; Chu et al., 2023; Huot et al., 2023). The inclusion of monensin in the ration has reduced the incidence of digestive deaths in feedlot cattle, probably due to the inhibition of some lactate-producing bacteria and the reduction in daily feed intake variation (Cooper et al., 1997; Nagaraja and Titgemeyer, 2007; Millen et al., 2016; Neumann et al 2018; Huot et al., 2023).

Meal frequency can be as important as total feed intake as a cause of acidosis. For example, cannulated cattle typically have higher feed intake. Weather changes and processing cattle for cannulation or vaccination often disrupt feeding patterns and can lead to overconsumption and acidosis. Proper timing of processing can be beneficial so that cattle are not deprived of feed; limiting intake after work or weather changes can also be beneficial. Estrogen implants have been shown to increase meal frequency, which may reduce the potential for acidosis. The effects of meal frequency may also explain why more skittish animals and some breeds are more prone to acidosis. However, if meal frequency is important, one would expect the incidence of acidosis to be higher when cattle are restricted or programmed fed. To date, restricted feeding has not been reported to increase the incidence of acidosis, perhaps because the total amount of feed provided is not excessive. On the other hand, if excessive amounts of feed are given inadvertently or during the transition from restricted feeding to free choice, acidosis can be expected (Beauchemin 2018; Deimeters et al., 2024).

The role of ruminal protozoa in acidosis is not clear. By ingesting starch particles and storing glucose as polysaccharides, protozoa delay starch fermentation by bacteria, help delay acid production and stabilize ruminal fermentation (Cooper et al., 1997; Nagaraja and Titgemeyer, 2007; Millen et al., 2016; Perez et al., 2024). Given the large amounts of starch consumed by ruminants, the quantitative importance of starch consumption by protozoa seems questionable. However, the ruminal bacterial population is normally reduced when protozoa are present; this reduction may also delay fermentation. Protozoal numbers in the rumen are typically reduced when fed highly concentrated diets, possibly because the long ration fiber provides a fibrous cover in the rumen to which protozoa attach and remain long enough to multiply. Free fatty acids and detergents also reduce protozoal numbers, and a low pH can cause deafunition. However, in addition to stabilizing normal fermentation, protozoal presence in the rumen can be detrimental. Since bacteria have much higher amylase activity than protozoa, amylase activity is higher when degrading per unit protein (Castillo-González et al., 2014). Due to changes in acid or osmolality associated with acidosis, large amounts of amylase are secreted, which accelerates the production of glucose from starch and increases the likelihood of acidosis.

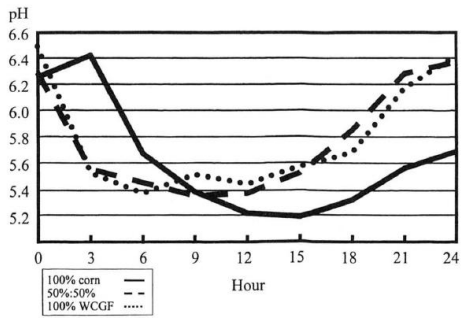

Figure 3. The role of moist corn starch in acidosis (Stock, 2000)

Protozoal stimulants or inhibitors may affect the tendency and severity of acidosis. High levels of dietary fat often led to ruminal instability as protozoal numbers are reduced by highly concentrated rations and removed by unsaturated fatty acids. Huffman et al., (1992), suggested that by coating the grain and reducing the rate of fermentation, added oil should reduce the incidence of acidosis. However, in vivo loading studies with corn and wheat found that the amount of oil had no effect on pH when it fell below 6.0, suggesting that oil was ineffective in preventing subacute acidosis (Krehbiel et al., 1995).

Inclusion of lactobacilli cultures in the diet may prolong ruminal retention of protozoa (Monteiro and Faciola, 2020), reduce fermentation and ruminal lactate production, and help maintain a higher ruminal pH (Cooper et al., 1999). Williams et al., (1991), observed that the mean and peak L-lactate concentration in the ruminal fluid of cattle fed a barley-straw ration indicated that ruminal pH was higher when a yeast culture was added to the ration.

Glycolysis: Anaerobic microorganisms typically thrive when free glucose is available. High concentrations Yet, high concentrations of free glucose in the rumen during acidosis suggest that glycolysis may be partially blocked. In rumen fluid incubation studies, less than half of the glucose incubated with rumen contents (1% wt/vol) was lost within 6 hours, supporting the concept that free glucose is not readily catabolized for unknown reasons.

Essential Fatty Acid and Lactate Production and Utilization: Bacteria in the rumen are typically categorized as either 'lactate producers' or 'lactate users'. The balance between these two groups determines whether lactate accumulates or not. The end products of bacterial strains can vary depending on substrate availability and culture conditions (Monteiro and Faciola, 2020). Most microorganisms that utilize lactate are sensitive to low pH, while most lactate producers are not. Under anaerobic conditions, pyruvate is converted to lactate to regenerate NAD used in glycolysis. Normally, lactate does not accumulate in the rumen at concentrations above 5 mM. Concentrations exceeding 40 mM are indicative of severe acidosis. Two forms of lactate are produced by ruminal and silage microorganisms: the D+ and L form. The L form, which is identical to that produced by exercising muscle from glucose, can be readily metabolized by liver and heart tissue. In contrast, D+ lactate, which typically accounts for 30 to 38% of the total lactate present in the rumen, is not produced by mammalian tissues. The accumulation of free lactate in silage serves to stop fermentation and stabilize the mass. Acidosis is often accompanied by the presence of other microbial products such as ethanol, methanol, histamine, tyramine, and endotoxins, which may have systemic effects (Monteiro and Faciola, 2020; Perez et al., 2024).

Control of Lactate Production and Utilization: Rumen acidosis can be caused by lactate-producing Streptococcus bovis and lactobacilli, coliforms that may lead to anaphylactic shock and sudden death, and amino acid-degrading microorganisms associated with tyramine and histamine production (Amin and Mao, 2021). These microorganisms can be controlled with antibiotics or bacteriophages. To prevent acid accumulation, inoculation with lactate-utilizing microorganisms that can tolerate low pH may be beneficial. Some examples of such microorganisms include Megasphaera elsdenii (Owens et al., 1998), Lactobacillus acidophilus (Monteiro and Faciola, 2020), and three species found in Active Rumen Microorganisms (ARM). Survival in the face of fierce competition from other microbial species makes long-term replacement of rumen microflora difficult. However, individual animals exhibit consistent differences in rumen metabolism. During the 6-month ad libitum feeding period, ruminal lactate production ranked 10th when feeding with corn starch. This suggests that the mix of microbial species within an animal remains constant, although some animals may be more prone to acidosis.

Ruminal pH Decline: Ruminal pH is determined by the relative concentrations of bases, acids, and buffers. Ammonia is the primary rumen base, while bicarbonate and phosphate are the two primary buffers under neutral pH conditions. When pH drops below 5, VFA and lactate also act as buffers (Beauchemin, 2018). During acidosis, when the pH drops to 5.0, the ionization of acids increases slightly. However, the primary cause of the increased hydrogen ion concentration is the added lactate (Figure 4). Lactate is responsible for lowering the pH more severely than similar amounts of other rumen acids due to its low pK (pH point of maximum buffering) of 3.8 compared to 4.8. The acidic pH leads to an increase in osmotic pressure due to greater ionization of acids and the presence of free glucose. Compared to normal concentrations, the change in osmolality during acidosis is much greater than the change in hydrogen ion concentration. Normally, absorption from the rumen prevents acid accumulation. However, the high osmolality of the rumen contents reduces the rate of acid absorption, exacerbating acidity and osmolality. This, in turn, increases the conversion of pyruvate to lactate by lactate dehydrogenase activity, making recovery from acidosis more difficult (Millen et al., 2016). The severe drop in rumen pH is difficult to reverse due to the increased pyruvate hydrogenase activity and promotion of pyruvate to lactate conversion caused by low pH (Perez et al., 2024).

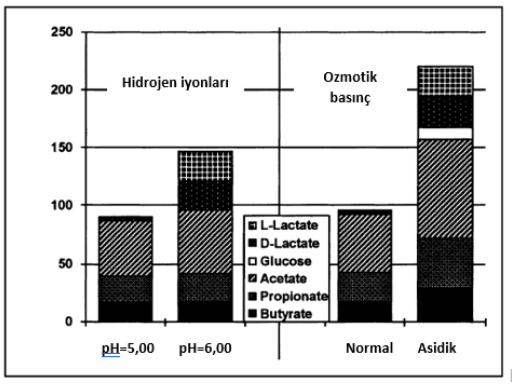

Figure 4. Relative contributions of various organic compounds to ruminal acidity and osmolality under normal or acidotic conditions. Bar heights indicate (from bottom to top) the relative contributions to hydrogen ion and osmolality from butyrate, propionate, acetate, glucose, D-lactate and L-lactate (Owens et al., 1998)

Ruminal Osmolality

The normal range for ruminal osmolality is 240-265 mOsm/L with roughages and 280-300 mOsm/L with concentrates (Owens et al., 1998). The main solutes in rumen fluid are minerals, urea, lactate, and glucose. Protein dissolved in blood significantly contributes to the osmotic pressure, which typically ranges from 285 to 310 mOsm. If rumen osmolality significantly exceeds blood

osmolality, water in the blood is rapidly drawn through the rumen wall. In a study has demonstrated in histological studies that rapid flow to neutralize osmotic pressure causes the ruminal papillae to swell, which can pull patches of rumen epithelium into the rumen, stripping the inner surface layers of the rumen wall from the underlying layers. This rapid water flow can result in damage to the rumen wall or small intestine, which can later be identified as abscess sites. When sepsis occurs, the microorganisms responsible for liver abscesses in the rumen can enter the bloodstream, causing hyperkeratosis or parakeratosis in the repaired tissues of the digestive tract (Eger et al., 2018). This can inhibit the absorption rate of VFA for months or even years after the damage has occurred (Huot et al., 2023). The absorption of VFA through the postruminal passage is possible, but its presence in the abomasum may hinder postruminal starch digestion by inhibiting acidification, protein, and mineral digestion. Therefore, a single episode of non-fatal acidosis can have long-lasting effects. An increase in osmotic pressure in the rumen is detected by the reticulorumen wall, which then inhibits feed intake (Nagaraja and Titgemeyer, 2007; Kachhadia et al., 2023). In addition, bacterial digestion of fiber and starch in the rumen is inhibited by osmotic pressures above 350 mOsm, leading to stagnation of rumen contents. High osmolality (>300 mOsm), coupled with obstruction of outflow causing swelling of the abomasal cavity, makes it challenging to remove fluid and acid from the rumen. While ruminal hypertonicity can reduce the frequency of rumen contractions, it is important to note that inhibited intestinal motility or hypertonicity in the abomasum can stop flow and worsen rumen acidification. Additionally, altered motility or tonicity can cause fluctuating feed intake, leading to chronic acidosis (Cooper et al., 1999; Trotta et al., 2021; Fu et al., 2022). It is crucial to maintain proper motility and tonicity in both the rumen and abomasum to prevent these issues.

Acid Absorption

Lactate and VFA are passively absorbed by the rumen and intestinal epithelium. The absorption rate is higher when concentrations are high, pH is low, and osmolality is normal (Snyder and Credille, 2017).

The percentage of each organic acid in the undissociated form increases with decreasing pH, resulting in a higher absorption rate. Butyrate is partially metabolized as an energy source for the rumen wall during absorption, and glucose is partially converted to D-lactate. Lactate is produced in the intestines and absorbed from them (Singh et al., 2022). Therefore, the total lactate load for the liver can greatly exceed lactate absorption from the rumen.

Blood Osmolality

Blood osmolality increases during acidosis due to two reasons. Firstly, the high osmotic pressure within the rumen draws fluid from the plasma within the rumen. Concentrates blood components and increases blood osmolarity, cell density, and water absorption. Additionally, if the rate of absorption of rumen acid or glucose exceeds the rate of metabolism or excretion, these compounds can accumulate in the blood and directly increase osmotic pressure. High histamine concentrations and vascular damage due to uncontrolled increases in blood pressure within the hoof are associated with acidosis-specific hoof disease and laminitis (Annatte et al., 2019). Osmoreceptors in the rumen sense food intake, rumination in the portal system or liver, and inhibition of saliva secretion (Jiang et al., 2017; Loncke et al., 2020). Fluctuations in blood osmolarity may be the cause of the short-term reduction in feed intake observed in subclinical acidosis. Therefore, it is recommended to consider acute acidosis and chronic acidosis as separate diseases, despite their similar rumen acid bases.

Acid Metabolism and Excretion

Metabolism of VFA results in the conversion of glucose for storage or carbon dioxide for energy. This process occurs rapidly, maintaining low blood concentrations. Previous studies, reviewed by Kachhadia et al., (2023), indicate that D-lactate is not metabolized as efficiently as L-lactate, suggesting that it is eliminated from the blood through renal excretion. Much research suggests that ruminant tissues can metabolize D-lactate. The conversion of L-lactate to glucose by calf hepatocytes is reduced in the presence of butyrate, indicating that interactions between absorbed acids may affect the metabolism rate (Annatte et al., 2019; Loncke et al., 2020; Trotta et al., 2021; Fu et al., 2022).

Classification Of Ruminal Acidosis

Ruminal acidosis is classified into two types according to the rate of occurrence. The first is acute ruminal acidosis, which occurs when large amounts of highly fermentable carbohydrates are consumed in a short period of time (Owens et al., 1998). Acute acidosis is characterized by a rapid decrease in ruminal pH, decreased fiber digestion and excessive lactic acid production. The accumulation of lactic acid in the rumen can be detrimental to the microbial population and cause a decline in rumen function (Monteiro and Faciola, 2020; Amin and Mao 2021; Perez et al., 2024). The other type of ruminal acidosis is subacute, a more chronic form of acidosis that occurs when animals consume moderately fermentable feedstuffs over an extended period of time. The ruminal pH drops, but not as drastically as in acute cases. Subacute rumen acidosis can be more difficult to diagnose, as symptoms may be less pronounced.

1. Subacute Ruminal Acidosis (SARA) is a milder form of ruminal acidosis characterized by recurrent or chronic episodes of low ruminal pH. It is often associated with high-grain diets, inadequate fiber intake, or improper feed management practices. Causes include reduced feed intake, inconsistent milk production, mild diarrhea, and occasional lameness (Fu et al., 2022).
2. Acute Ruminal Acidosis (ARA) is a severe and sudden form of ruminal acidosis, typically caused by a rapid overload of fermentable carbohydrates. This results in a drop in rumen pH to below 5.0, reaching highly acidic levels. ARA can occur due to nutritional errors, sudden changes in diet, or inadvertent consumption of large quantities of easily fermentable carbohydrates. Symptoms of the condition include deep depression, anorexia, diarrhea, dehydration, rapid and weak pulse, lying down, and laminitis. If left untreated, severe cases can lead to shock and death.
3. Chronic ruminal acidosis refers to a prolonged state of low ruminal pH that persists over a long period of time. The ruminal pH remains consistently below the normal range of 6.0 to 6.5. Chronic acidosis can occur as a result of long-term imbalanced diets, insufficient fiber, or constant exposure to stressors. Clinical symptoms include variable appetite, decreased milk production,

weight loss, poor body condition, and increased susceptibility to secondary health problems.

4. Subclinical ruminal acidosis (SARA) refers to a condition in which rumen pH is intermittently below optimal levels without obvious clinical signs. Rumen pH is between 5.6 and 5.2 for most of the day. Similar to SARA, subclinical acidosis is primarily caused by highly concentrated diets, low fiber intake, or improper nutritional management. Diagnosis of subclinical acidosis often requires pH monitoring devices such as rumenocentesis or permanent pH probes. Although there are no obvious clinical signs, subclinical acidosis can have a detrimental effect on rumen health, feed conversion and animal performance.

It is important to note that the classifications provided here are general categories. The severity and presentation of ruminal acidosis may vary depending on individual animal factors, management practices, and environmental conditions. Prompt diagnosis and appropriate management are critical to preventing and reducing the adverse effects of ruminal acidosis.

Symptoms of Ruminal Acidosis

Symptoms of ruminal acidosis can vary depending on the severity and duration of the condition. Some common symptoms included;

- Decreased feed intake,
- Decreased rumination,
- Diarrhea or abnormal stools
- Pain (abdominal kicking, etc.)
- Decreased milk production
- Lameness or reluctance to move
- Dehydration and
- Cachexia in severe cases

If ruminal acidosis is suspected, it is important to consult a veterinarian for accurate diagnosis and treatment. Treatment may include reducing the intake of fermentable carbohydrates, adjusting the diet to include more fiber, providing supportive care, and administering medications to restore rumen function and microbial balance (Stock, 2000; Millen et al., 2016; Kachhadia et al., 2023).

Prevention and Protection

Prevention of ruminal acidosis includes proper management practices such as gradual feed changes, providing a balanced and consistent ration, avoiding sudden feed changes, and ensuring adequate fiber intake to maintain a healthy rumen environment. Regular monitoring of rumen pH and observation of animal behavior can also help identify early signs of acidosis and allow for immediate corrective action (Dunlop, 1972; Stock, 2000; Millen et al., 2016; Kachhadia et al., 2023). Reduce lactate concentrations; control ruminal pH, glycolysis, osmolality, acid absorption, blood pH, and blood osmolality; increase acid metabolism and excretion are important to prevent ruminal acidosis.

Balanced Diet: Adequate fiber should be provided. Ensure that ruminants have access to high quality forages such as hay or pasture to maintain proper rumen function and encourage rumination and salivation. At least 40% of the ration should be roughage. When transitioning animals to a high-concentrate diet, a gradual transition should be made over a period of 7-10 days to allow rumen microorganisms to adapt to the new feed. The adaptation period helps prevent sudden changes in rumen pH. Consistent feeding times and amounts should be maintained to establish a routine for rumen fermentation and to minimize fluctuations in rumen pH.

Feed Management: Diets should be formulated to meet the nutritional requirements of the individual animal or herd. Care should be taken to provide adequate levels of carbohydrates, protein, minerals and vitamins in the diet. Avoid overfeeding grain and other highly fermentable carbohydrates. The amount of starch in the diet should be limited to prevent excessive acid production. Finally, grains should be processed appropriately to improve digestibility and reduce the risk of acidosis. Techniques such as grinding or crushing can enhance rumen fermentation (Galyean and Goetsch, 1993; Krehbiel et al., 1995; Zinn et al., 1995).

Dietary Practices: To promote more consistent rumen fermentation and minimize acid fluctuations, the number of daily feedings can be increased, and the daily feed ration can be divided into several small meals. Adequate feeding space must be provided to prevent aggressive competition between animals, which can lead to overconsumption and digestive disorders (Sommai et al., 2020; Trotta et al., 2021).

Water Management: Clean, fresh water should be always available. Adequate water intake is very important for rumen function and maintenance of rumen pH. Water quality should also be monitored to ensure that it does not contain any contaminants or substances that could affect the health of the rumens (Millen et al., 2016; Jiang et al., 2017; Huot et al., 2023; Kachhadia et al., 2023).

Environmental Factors: Stressful events such as sudden ration changes, transportation or extreme heat, which can disrupt rumen function and increase the risk of acidosis, should be kept to a minimum. Adequate ventilation should be provided in barns or housing facilities to provide a comfortable environment and prevent heat stress (Fu et al., 2022; Chu et al., 2023).

Regular Monitoring: Rumen pH monitoring techniques such as rumenocentesis or permanent pH probes can be used to assess rumen pH and detect early signs of acidosis. Similarly, animal health, body condition and production parameters should be monitored regularly to detect any changes or abnormalities that may indicate ruminal acidosis (Stock, 2000; Singh et al., 2022).

CONCLUSION

Ruminal acidosis is defined as an acid imbalance in the digestive system of ruminants. This condition usually occurs due to factors such as excessive consumption of high grain diets, inadequate fiber intake, or rapid changes in diet. High levels of fermentation, lactic acid accumulation and a decrease in pH are characteristic features of ruminal acidosis. In our review article, we thoroughly examined the available information in the literature and focused on understanding and addressing this important health problem by providing a comprehensive analysis of the etiology, clinical manifestations, diagnostic methods, and treatment strategies of ruminal acidosis. In doing so, we highlighted various feeding strategies to prevent and control ruminal acidosis, as well as methods to improve animal health and production efficiency. However, we also identified knowledge gaps in this area and potential opportunities for future research. Finally, we emphasize that ruminal acidosis requires a multidisciplinary approach and the importance of combining scientific and practical efforts to

improve animal welfare. The aim of this review is to provide a resource to guide future studies into the understanding and management of ruminal acidosis.

Conflict of Interest

The authors declare that they have no competing interests.

Authorship contributions

Concept: S.K, G.E. E.G, Design: S.K, G.E., E.G, Ş.D., Data Collection or Processing: S.K, G.E., E.G, Ş.D., B.G.G., Analysis or Interpretation: S.K., E.M.Ç., A.K., E.G., B.G.G., Literature Search: S.K., N.A., Writing: S.K., N.A.

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