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REVIEW ARTICLE

Ruminal acidosis: strategies for its control

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ABSTRACT. Ruminal acidosis in ruminants is a metabolic disorder of gastrointestinal origin that occurs in animals with a high feed intake of cereal grains diets, which affect the performance. According to clinical manifestations it can be classified as: a) acute lactic acidosis with prolonged exposure to ruminal pH ≤ 5.0, triggering a systemic acidosis, with clinical manifestations and changes in biochemical patterns, starting the first twelve hours of ruminal acidosis and it takes 48 to 120 hours to reestablish, b) subacute ruminal acidosis (SARA), it has no clinical manifestations, rumen pH decreases in a range of 5.5 to 5.0 and is held for 111 to 180 minutes in 24 hours, this decrease causes an imbalance in the ruminal metabolism, as well as the fluid balance. Ruminal acidosis can be prevented by adding sodium bicarbonate to the ration, or zootechnic additives such as *Saccharomyces cereviseae* and *Megasphaera elsdenii* as well as essential oils (cinnamaldehyde and eugenol). It is also important to consider the forage/concentrate ratio, the forage particle size, the NDF content of the ration, as well as the cereal's rate of degradation in the rumen. The objective of this review was to address the ruminal acidosis pathophysiology, its effects on animal welfare and control strategies to minimise the detrimental impact on animal production.

Key words: metabolic disorder, ruminal subacute acidosis.

RESUMEN. La acidosis ruminal es un trastorno metabólico de origen digestivo, que se presenta en los rumiantes al ingerir dietas altas en granos de cereales, y que afecta el rendimiento productivo. Según sus manifestaciones clínicas se clasifica en: a) acidosis láctica aguda en la que hay una exposición prolongada a un pH ruminal ≤ 5,0, lo que desencadena una acidosis sistémica con manifestaciones clínicas, así como cambios en los patrones bioquímicos que se inician en las primeras doce horas de la acidosis ruminal y tardan de 48 a 120 horas en restablecerse, y b) acidosis subaguda ruminal (ASR), donde no hay manifestaciones clínicas, el pH del rumen disminuye en un rango de 5,5 a 5,0 y se mantiene durante 111 a 180 minutos en 24 horas, este descenso provoca un desequilibrio tanto en el metabolismo ruminal como en el balance de líquidos. La acidosis ruminal se puede prevenir por la adición de bicarbonato de sodio, aditivos zootécnicos como la *Sccharomyces cerevisiae y Megasphaera elsdenii,* y aceites esenciales (cinamaldehído y eugenol). Es importante considerar la relación forraje/concentrado así como el tamaño de partícula del forraje, el contenido de FND de la ración, y la degradación del grano de cereal en el rumen. El objetivo de la presente revisión fue abordar la fisiopatología de la acidosis ruminal, los efectos que esta causa en el bienestar animal y las estrategias para su control, para minimizar su impacto detrimental en la producción animal.

Palabras claves: trastorno metabólico, acidosis subaguda ruminal.

INTRODUCTION

The functional health of the reticle-rumen is an essential requirement for productive behavior, encompassing livestock health and animal welfare (van Vuuren *et al* 2012). Under modern animal production systems, ruminants are fed high grain diets to maximize energy consumption and hence productivity (Oba and Wertz-Lutz 2011). However, ruminants are adapted to digest and metabolise forage diets (Krause and Oetzel 2006). Intensive fattening systems require rations containing high proportion of grains (corn, barley or sorghum), that upon reaching the rumen break down quickly producing large amounts of lactic acid that predispose to metabolic disorders (Smith 1998). One of the limitations in intensive ruminant production is acute ruminal acidosis and subacute acidosis ruminal (SARA); the latter is difficult to diagnose, because the only thing that can be observed is a decrease in feed consumption and consequently an increase in feedlot time and increased production costs.

The objective of this review was to address the ruminal acidosis pathophysiology, its effects on animal welfare and control strategies to minimise the detrimental impact on animal production.

ACIDOSIS PATHOPHYSIOLOGY

Ruminant lactic acidosis is a digestive disorder caused by the sudden intake of easily digestible carbohydrates in the rumen, mainly cereal grains (Haji-Hajikolaei *et al* 2006, Aschenbach *et al* 2011). Cereal starch is rapidly degraded in the rumen to become lactic acid at a very high rate that exceeds the rumen absorption capacity (Haji-Hajikolaei *et al* 2006). The lactic acid production is affected by the substrate, being higher for wheat than for maize (Lettat *et al* 2010). The accumulation of this acid in the rumen, favours pH decrease (Wenping *et al* 2007, Aschenbach *et al* 2011). The absorption of lactic acid in the circulatory system, alters the systemic and ruminal acid-base balance, as well as the water balance and food intake (Nocek 1997).

Food intake and chewing during ingestion and rumination, has a great effect on the acid-base balance of the

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ruminal fluid. The characteristics of the intake associated with decreased ruminal pH are: high intake of dry matter in a short period of time, promoting increased production of acid in the rumen, because saliva production decreases along with chewing time. The acid-base balance in the rumen requires time synchronisation between the production of acid, neutralisation of this through saliva and absorption through the ruminal epithelium (Gonzalez *et al* 2012). Giger-Reverdin *et al* (2006) mention that saliva production increases along with the chewing rate per kilogram of dry matter, and with the increase of saliva production there is a greater buffer input into the rumen increasing the pH to 5.94.

The primary system to counteract the development and progression of acidosis is the HCO_3- reaching the rumen via the saliva, which is the medium used to maintain stable ruminal pH. Estimates in high producing dairy cows indicate that HCO_3- is introduced into the rumen approximately in equal parts via saliva and secretion through the ruminal epithelium. Most secreted HCO_3- binds directly to the absorption of short chain VFA (Volatile fatty acids), which are absorbed by lipophilic diffusion in the ruminal epithelium (figure 1), both HCO_3- secretion and VFA absorption,are the key to maintaining stable the ruminal acid-base balance (Aschenbach *et al* 2011, Dijkstra *et al* 2012).

Volatile Fatty Acid produced during microbial fermentation should be neutralised o eliminated from the rumen to prevent ruminal pH decrease. VFA are weak acids and establish a balance between acid (H acid) and the base conjugate (acid-). Ruminal pH depends on the Henderson-Hasselbach balance $pH=pKa+\log[(acid^-)(H\ acid)^{-1}]$ where pKa is the negative logarithm of the acid constant, Ka. Lactic acid has a lower pKa (3.80) than acetic acid (pKa=4.76), propionic acid (pKa=4.78) or butyric acid (pKa=4.82). Due to the fact that the three major VFA, acetic, propionic and butyric acids have a similar pKa value, the production of 1 mole of butyric acid from 1 mole of hexose can reduce the pH relatively less than the production of 2 moles of propionic or acetic acid from 1 mole of hexoses. During ruminal acidosis, lactic acid concentration is low but as the pH drops below 5.7, the rate of lactic acid formation exceeds the absorption capacity, mainly through fermentation by acid user microorganisms, therefor lactic acid is accumulated in the rumen, which causes a drastic decrease in pH (Dijkstra *et al* 2012).

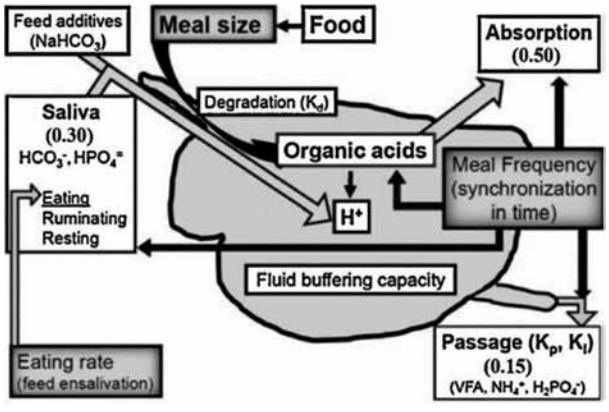

Figure 1. Factors involved in acid-base balance in the ruminal fluid. The main routes of elimination of rumen acids is absorption, neutralisation by buffer substances in saliva and the passage of food and liquid substances from rumen to the small intestine. Eating behaviour affects time synchronisation between the production of acids and food elimination (Gonzalez *et al* 2012).

Rumen lactate accumulates in two isomers: D-lactate produced in lower amount than L-lactate, being larger the absorption of the first one; an increase of D-lactate ruminal absorption to the blood system that can exhaust the buffering capacity of HCO_3- and produce a generalised acidosis (Harmon *et al* 1985). The ruminal microbiome is determined by the type of feed ingested by the ruminant, and rumen bacteria (*Ruminococcus*) predominate at pH 6.8 in cellulolytic diets.

However, increasing the concentrate in the diet and reducing the forage modifies the microbial population change the ruminal pH (5.5 and 6.5) and increase the amylolytic bacteria such as *Streptococcus bovis* (Lean *et al* 2007).

Health disorders, which result from acidogenic diets are classified as acute acidosis and SARA, this ranking is based on the degree of pH ruminal decrease (Aschenbach *et al* 2011).

Acute ruminal acidosis is a metabolic state defined by the decrease of blood pH and bicarbonate (Hernandez 2014), the ruminal pH is less than 5.0 and the animals have depression, loss of appetite, reduced rumination, diarrhea and dehydration (Bolton and Pass 1988, Laskoski *et al* 2014); microbial metabolism and food digestion are also affected, inflammatory processes occur and milk fat decreases (Dijkstra *et al* 2012).

Subacute ruminal acidosis (SARA) is a common health and production problem occurring en rumiants and is often triggered by low ruminal pH from a low-fiber diet (Zhao *et al* 2011). Subacute ruminal acidosis is defined as the time that rumen pH drops from 5.5 to 5.0, and is maintained in that range for 111 to 180 minutes in a 24 hour period (Krause and Otzel 2006, Rustom *et al* 2006, Penner *et al* 2010, Castillo-Lopez *et al* 2014). SARA is a state of temporary disruption of rumen amending fermentation patterns; however, the intensity and duration are not sufficient to cause immediate clinical signs (Nocek 1997, Maulfair *et al* 2013). Technological advances have greatly improved the understanding of the mechanisms of interaction between the fermentability of the diet, the intake and rumen pH (Penner *et al* 2009). The low ruminal pH characteristic of SARA may be due mainly to changes in the buffering capacity of carbonic acid and VFA. The effect of a long term acid diet over the alkaline reserve in blood suggests a long recovery period which is reflected in the ruminal parameters (Brosard *et al* 2003).

Another consequence of the reduction in pH is the decrease in digestible organic matter ingested and VFA production, which may be part of the rumen ecosystem self-regulatory mechanisms against ruminal acidosis (Calsamiglia *et al* 2008). SARA is not only a pH dependent disorder, but is the result of changes in secondary microbial population and the type of diet fed (Calsamiglia *et al* 2012).

CONSEQUENCES ON HEALTH, ANIMAL WELFARE AND PRODUCTION

Animal welfare implies that the animal will develop physically and mentally in good conditions, and that nutritional, social, management, health and comfort factors do not adversely affect production (Hewson 2003, Carenzi and Verga 2009).

Health disorders which result from acidogenic diets, in acute acidosis are based on the degree of pH ruminal decrease (Garret *et al* 1999, Aschenbach *et al* 2011, Kleen and Cannizzo 2012, Minuti *et al* 2014). The presence of large amounts of lactic acid in the ruminal fluid and its rapid entry into the blood system demand a great challenge from the buffering capacity of the ruminal intake and body fluids. Lactic acid remaining in the digestive system also contributes to lactic acidosis syndrome (Nocek 1997), affects metabolism (Galyean and Rivera 2002), change the population of microorganisms, rumen motility and the systemic fluid balance. As the pH of rumen intake severely diminishes towards pH 5.0, the amplitude and frequency of rumen contractions progressively decrease until it reaches rumen stasis (Huber 1976).

Three mechanisms may be responsible for ruminal atony after acidification of the rumen: 1) the effect of hydrogen ion on receptors in the gastrointestinal system; 2) central inhibition by acid absorption and 3) inhibition by amines or absorbed toxins (Huber 1976). Ruminal atony is manifested as poor appetite, because if the rumen is not functioning, degradation and nutrient absorption through the ruminal epithelium is not performed, so animals stop eating (González *et al* 2012).

In acute acidosis the rumen epithelium alters its function and structure; with diets high in grains the rumen papillae show a large detachment of corneum stratum and affects the adhesion of cells (figure 2) with the appearance of large gaps between cells across the layer (Steel *et al* 2009). As the ruminal epithelium is damaged, an inflammatory process that causes pain in ruminants is generated and manifests as the clenching of teeth, loss of appetite and ruminal atony (Odongo *et al* 2006[a]).

The ruminal barrier is responsible for maintaining the concentration gradient required for the absorption of ions, prevent translocation of lipopolysaccharides and other toxins, as well as bacterial entry into the systemic circulation. Rumen acidosis is the most important factor that can damage the rumen wall (Penner *et al* 2011, Plaizier *et al* 2012), decreasing the count of papillae on the ventral sac of the rumen (figure 3) (Odongo *et al* 2006[a]). Pharmacologically active amines have been detected during the intake in rumen from animals with acidosis, such as histamine that comes from histidine decarboxylation when rumen intake approaches pH 5.0 or less (Huber 1976). The histamine release in the rumen and its absorption into the circulatory system, allow these compounds to reach the extremities where they cause inflammatory reactions such as laminitis, which causes pain when moving.

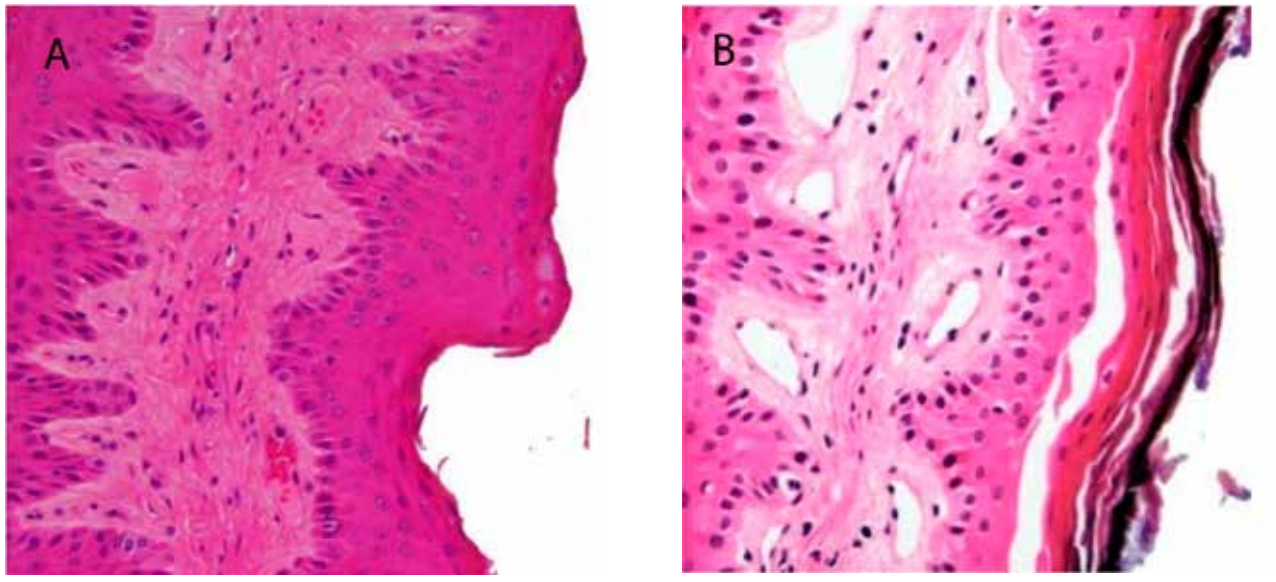

Figure 2. Photograph of two ruminal papillae: A) Ruminal papilla of a diet high in forages, with the corneum and granular stratum intact. B) Rumen papilla of a diet high in grains, where swelling of the corneum stratum and demarcation of cells throughout the epithelial layers is observed (Steel *et al* 2009).

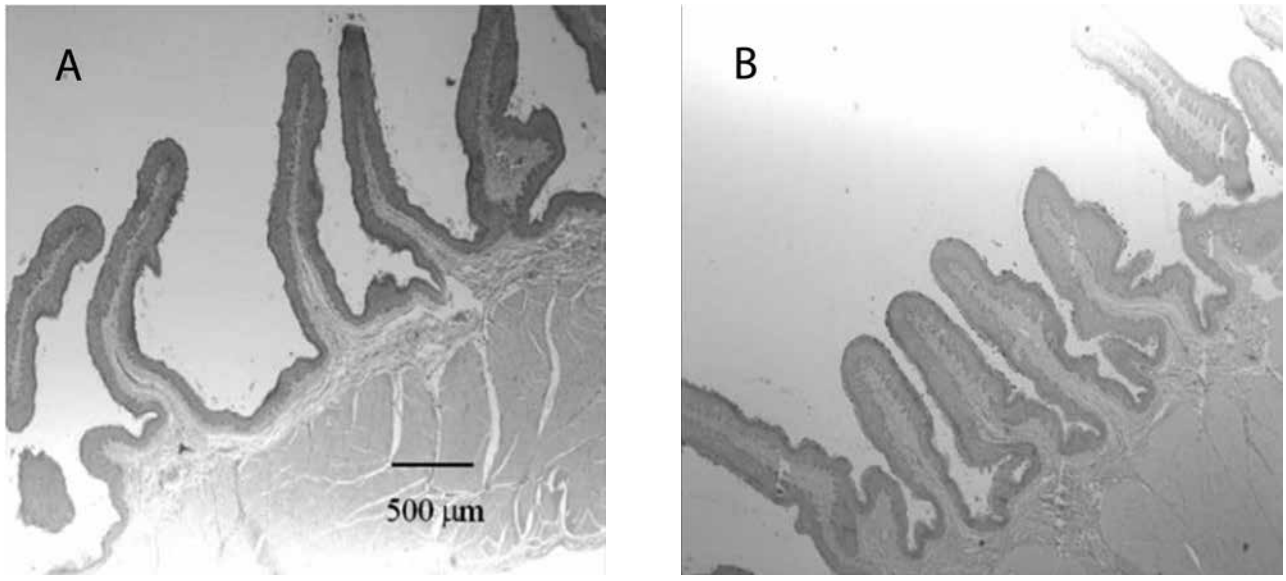

Figure 3. Ruminal papillae morphology of lambs on two diets. A) High in grains; B) Control group (Odongo *et al* 2006[a]).

Another consequence of ruminal acidosis is that fluid balance is affected. The animals that develop ruminal acidosis may experience diarrhea and dehydration, consequently there is fluid loss and therefore increases the hematocrit, decreasing the elasticity of the skin. The rumen fluid becomes hypertonic to plasma, therefore water is absorbed from the blood system into the rumen, and consequently the water lost in the body and blood enters the rumen. Rumen osmolarity increases from 255 to 401 milliosmoles. Lactic acid is responsible for the increase (61%) of the osmolarity. The fluid loss through faeces is high during diarrhea and occurs in the time period when the rumen motility is inhibited, suggesting a net movement of body water towards the intestine (Huber 1976).

Prolonged exposure to a low ruminal pH, results in a systemic acidosis which imposes a chronic stress on the physiological mechanisms of the animal and the ability to maintain the acid-base balance (Odongo *et al* 2006[b]). Chronic metabolic acidosis alters the acid-base parameters (Haji-Hajikolaei *et al* 2006) and the amino acid concentration in blood (Odongo *et al* 2006[b]).

In the SARA, dry matter intake decreases, which favors an imbalance in the ruminal metabolism, having a lower nutrient uptake (Kleen and Cannizzo 2012). There is a possible link between the decrease in rumen pH and voluntary feed intake (Fulton and Klopfenstein 1979).

To measure the blood acid-base balance in acute acidosis and SARA, the Henderson-Hasselbach equation is used, wherein the ratio of bicarbonate and carbon dioxide must decrease as blood pH decreases; however, when the tension of carbon dioxide from blood is increased and blood pH is decreased, the respiratory center is stimulated, leading to an increase in CO_2 removal and thus the buffer is restored to the desired level of 20:1. Therefore, as the input of lactic acid to blood increases, carbon dioxide tension increases, respiratory rate decreases, blood pressure decreases and pH quickly drops (Huber 1976). The acute decrease in ruminal pH is associated with the decrease of blood pH (Brown *et al* 2000), the increase of total lactic acid in the rumen fluid and blood, the increase of blood levels of sodium and inorganic phosphorus, and the decrease of potassium and calcium levels (Patra *et al* 1991).

Biochemical pattern changes initiate within the first twelve hours of ruminal acidosis and take 48 to 120 hours to reset (Patra *et al* 1991).

STRATEGIES TO REDUCE RUMINAL ACIDOSIS

BUFFER SUBSTANCE

A buffer is a material that when is present in an aqueous solution causes an effective resistance to changes in pH of the solution, when it is strongly acid or base is added. For a compound to act as a buffer the following requirements must be met: 1) must be soluble in water; 2) must be a weak acid or base; 3) the equivalence point (pK) must be near to the physiological pH of the system to buffer. Buffering agent requirements in the diet are based on the buffer capacity of the saliva secreted, buffer capacity of the food and diet acidity (Erdman 1998).

Buffer substances supplementation can be a way to limit the adverse effects of ruminal acidosis and can be added in whole rations from 0.5 to 2.5%. The following compounds have been used: sodium bicarbonate, disodium carbonate, magnesium oxide, potassium carbonate and anhydrous limestone (Gastaldello *et al* 2013). In dairy cows, a meta-analysis of 40 publications of the following buffer substances was made: sodium bicarbonate, disodium

carbonate, magnesium oxide, potassium monoanhydro carbonate, monosodium carbonate and magnesium oxide; the dry matter intake was improved in 0.15 kg/day, milk production 0.52 kg/day and fat percentage 0.15%. Rumen pH increased by 0.07 units, so buffer substances are more efficient in SARA (Meschy *et al* 2004).

The addition of sodium bicarbonate results in the release of CO_2 from the solution and eventually from the rumen via belching. This process directly neutralises ruminal acidity. The extent to which the process proceeds depends on the partial pressure of CO_2 in the gas phase, the pH and a constant (7.74), according to the Henderson-Hasselbalch equation: pH= 7.74+log[(HCO_3^-)]/CO_2 pressure in atmospheres (Kohn and Dunlap 1998).

Sodium bicarbonate can increase the osmolarity, water consumption, the production of saliva and therefore increases the dilution rate of the VFA in the rumen and the passage rate of the liquid phase (Gastaldello *et al* 2013). Sodium bicarbonate acts in two ways: as a source of sodium to meet the requirements and help provide a positive balance in the cation-anion ratio in dairy cows (Hu and Murphy 2005, Lean *et al* 2007) and as a buffer in diets based on corn silage (200 to 300 g/animal/day) (Lean *et al* 2007). In addition to baking soda, sodium sesquicarbonate in the diet can be used and its effect is more pronounced on diets that are used at the end of lactation (Clark *et al* 2009). The addition of sodium bicarbonate in dairy cows is limited by the elimination of sodium through urine and subsequent alkalization of the soil (Rauch *et al* 2012). In weaned lambs fed barley grain and a supplement of granulated protein, sodium bicarbonate did not buffer the pH of the ruminal fluid, nor improved dry matter intake and daily weight gain, but modified the fermentation patterns towards greater ratio of acetate to propionate (Boda 2004, Askar *et al* 2011).

Another buffer used in the prevention of ruminal acidosis is limestone; in lambs the addition of 1.3% in the diet maintained rumen pH between 5.7 and 6.0 and dry matter digestibility improved, so it can be used in diets with high concentrations of corn (Gastaldello *et al* 2013). For beef cattle in finalisation (Anderson 1982) three levels of limestone 1, 2 and 4% were used. The productive performance was better in diets with 1 and 2%, higher levels inhibited appetite and reduced efficiency. The addition of magnesium oxide 0.05% in the concentrate improved daily gain, feed intake and final weight of Lori-Bakhitiari lambs in finalisation (Hashemi *et al* 2012).

ZOOTECHNICAL ADDITIVES AND VACCINES

In 2003 the European Union banned the use of antibiotics as additives in food for domestic species. The old category of 'microorganisms' and the term 'probiotics' disappears for being too general and is replaced by that of 'zootechnical additives' in which microorganisms and enzymes are included. One of the points of interest of the use of zootechnical additives in ruminants, is to control the accumulation of lactate in the rumen and stimulate the synthesis of propionate; the most commonly used in ruminants are the yeasts (*Saccharomyces cerevisiae* and *boulardii*) as well as *Aspergillus niger* and *oryzae* (Caja *et al* 2003).

In the supplementation of live yeast to lactating cows (5 g/day) equivalent to 10^{10} CFU of *Saccharomyces cerevisiae*, the average ruminal pH was higher in cows supplemented than in those who received no yeast (6.05 vs 5.49). The effect of yeast supplementation on ruminal pH is evident one week after the supplementation initiated (Bach *et al* 2007). Yeast supplementation is effective in reducing the amount of time in which rumen pH is below 5.8 (Vyas *et al* 2014[a]). The use of yeast in ruminants reduces subclinical acidosis (Bach *et al* 2007, Vyas *et al* 2014[a]), however, the supplementation of yeast had no effect on the pH during the experimental induction of acute acidosis in cattle (Vyas *et al* 2014[b]). Uyeno *et al* (2017) suggest that effects of supplementing live yeast to cows in mid-to-late lactation may be limited to microbial composition and fermentation characteristics in the rumen.

The administration of *Megasphaera elsdenii* 10^{11} CFU, strain NCIMB 41125 in sheep, can control ruminal acidosis during the transition period from forage to grain, because *Megasphaera elsdenii* is able to use the ruminal lactic acid and keeps it constant at <10 mmole/L (Henning *et al* 2010[a], Henning *et al* 2010[b]).

Not only yeasts are used in the control of ruminal acidosis, polyclonal antibodies preparation (PAP) can also be used which are a mixture of immunoglobulins secreted against a specific antigen that may be effective in reducing the incidence of acidosis during transition from high forage diets to high grain diets. The major target bacteria for the PAP is *Streptococcus bovis*, main cause of ruminal acidosis, but is not relevant in practice (Blanch *et al* 2009). Intramuscular immunisation against *Streptococcus bovis* strain SB-5 and a booster dose at 28 days, improved ruminal pH and dry matter intake, reduced the incidence of diarrhea and increased the blood cell pack volume in sheep fed a ration containing 90% wheat grain and 10% alfalfa (Shu *et al* 2000). Besides *Streptococcus bovis* in ruminal acidosis research, the importance of *Pediococcus acidilactici* has also been demonstrated (Cobos *et al* 2011).

ESSENTIAL OILS AND ANTIOXIDANTS

There is a potential benefit for using some plant extracts to improve the profile of ruminal fermentation in beef production systems when the ruminal pH is low (pH 5.5). *Allium sativa*, *Capsicum anuum*, *Yucca schidigera* and *Cinnamonum cassia* extracts alter the fermentation of rumen microorganisms in favour of propionate (Cardozo *et al* 2005). *In vitro* studies with essential oils (EO) proved that microbial activity is modified in the rumen and can be a natural alternative to modify the rumen microbial

fermentation. The EO *Syzygum aromaticumm* at a dose of 500 mg/L increases the total of VFA (Castillejos *et al* 2008). Likewise, the mixture of cinnamaldehyde (0.18 g/d) and eugenol (0.09 g/d) proved to be useful as food additives in beef heifers fed high grain diets, by modifying the rumen microbial fermentation (Cardozo *et al* 2006). The effect of a garlic oil chemical compound, propyl-propane thiosulfonate (PTSO), in an effective dose between 50 a 100 mg/L modify ruminal fermentation in a direction consistent with higher propionate molar proportion (Foskolos *et al* 2015).

The general term vitamin E is used to designate a group of eight natural species of tocopherols and tocotrienols (α, β, γ and δ) (Sayago *et al* 2007). The main function of vitamin E is to be an antioxidant that reacts with free radicals soluble in lipid membranes (Cespedes 2012). In the ruminal lactic acidosis there is an oxidative stress (Kirbas *et al* 2014) so vitamin E can be used in the prevention of the harmful effects of lactic acidosis, because it helps keep the acid-base status, at mitochondrial level, as demonstrated (Morán *et al* 2013) in feedlot lambs using 0.6 g/kg of vitamin E concentrate.

Carnosic acid is not an EO, but a phenolic diterpene extracted from *Rosmarinus officinalis* that acts as an antioxidant, it can be used in lamb diets high in concentrates to correct metabolic acidosis, because carnosic acid helps to maintain a stable acid-base balance at cellular level (Morán *et al* 2012, Morán *et al* 2013).

CONCENTRATED FORAGES

Finalisation of cattle with high energy diets reduces the cost per animal and reduces the feeding time, high weight gains are also obtained. However, these rations increase the incidence of acidosis (Anderson 1982), therefor it is necessary to include forages in ruminant finishing diets.

The decrease in the percentage of concentrates highly fermentable in feedlot diets, and when the level of forage is increased, the incidence of ruminal acidosis can decrease but these strategies are usually impractical for economic reasons, due to a decrease in feed efficiency (Krause 2002).

Ruminants need an adequate amount of fibrous texture food to prevent metabolic disorders. The level of fiber and large particles of food necessary to maintain rumen health depend on both the source and the size (NRC 2001). To prevent acidosis in sheep, a 30% of NDF is recommended (Lean *et al* 2007) and for dairy cattle at least 25% of NDF is recommended in the total diet on a dry basis, and 75% of this contribution must come from roughage to maintain a functional and healthy rumen (Li *et al* 2014). In dairy goats, between 373 and 384 g/kg NDF of dry matter must be used to prevent acidosis problems (Sudweeks *et al* 1981). In lambs fed high concentrate diets, the use of 10 to 34.5% soybean hulls in the ration maintain a stable ruminal pH of 5.96 to 6.18, being a major source of FND (Ferreira *et al* 2011).

In goats, when making the change from forage to concentrate from 70:30 to 30:70, the nutrient digestibility was better for rations with grains, compared to legumes. The ruminal pH of the 70:30 diet was 6.43 and the 30:70 diet was 6.21 (Cantalapiedra-Hijar *et al* 2009). In goat kids on finalisation fed diets with forage concentrate ratios of 60:40, 45:55, 30:70 and 15:85, ruminal pH was 6.92, 6.15, 6.32 and 6.09, respectively, therefore the proportion forage-concentrate maintaining adequate ruminal pH without affecting productive performance was 30:70 (Haddad 2005). In dairy goats fed a 50:50 forage-concentrate ration, in which three food particle sizes (1.81, 1.43 and 1.2 mm) were evaluated and the NDF was effective, the ones fed 1.43 mm particles, VFA concentration was increased without affecting NDF degradability and cellulolytic bacteria growth, rumen pH was 6.04 (Li *et al* 2014).

In highly productive dairy cows, it is recommended to use 31.2% effective NDF and particle sizes greater than 1.18 mm, or 18.5% effective NDF with particle sizes larger than 8 mm (Zebeli *et al* 2012).

Ruminal acidosis can be reduced by increasing the content of NDF physically effective in the diet, either by the use of long chopped forage or fodder proportion increase in the diet (Yang and Bauchemin 2006). Forage large particles promote rumination and secretion of saliva, which help to buffer acidity resulting from starch fermentation (Dijkstra *et al* 2012, Gonzalez *et al* 2012.). In cows, it has been demonstrated a close relationship between ruminal pH with respect to NDF content of the diet (32.2%) and the average size of the food particles (2.59 mm), decreasing the content of FND below 32.2% and particle sizes smaller than 2.59 mm the rumen pH decreases and the joint effect of NDF and the particle size is increased when the rumen pH is lower than 6.0 (Sauvant and Mertens 2001).

Beet pulp is a byproduct of industrial sugar extraction and the main components are carbohydrates. It contains 25 to 45% NDF, barely lignified (1.7%) plus appreciable quantities of pectins (20%) and sugars (6%)[1]. Pectin fermentation in the rumen does not generate lactic acid; galacturonic acid in its structure provides a buffer potential through the cation exchange and the bonding with a metal ion, so that the pulp helps regulate ruminal pH in concentrated rations of highly productive animals (Fernandez 2006, Bodas *et al* 2007).

The inclusion of pulp in cereal based diets of feedlot lambs seems to improve the ruminal environment and prevents ruminal acidosis, but has a negative effect on feed intake and animal behaviour (Fernandez 2006).

The fibrous texture of the food is quantitatively reflected on the ruminant mastication activity, stimulating the production of saliva which contains buffer substances, promoting the stability of the ruminal ecosystem. The appropriate size of feed particles for cows to maintain

[1] FEDNA. 2012. Pulpa de remolacha (actualizado) nov. 2012, www.fundaciónfedna.org; fecha de acceso 15.04.2014.

unchanged rumination should be between 2.83 and 3.29 mm (Grant *et al* 1990).

Ruminal papillae are modified according to the ration ingested by dairy cows, decrease their size when receiving high forage (dry period), and increase in size when increasing the amount of fermentable carbohydrates in the rumen, which corresponds to the transition period, the increased size of ruminal papillae favours the absorption of volatile fatty acids and is involved in the regulation of ruminal pH (Liebich *et al* 1987).

The effect of the inclusion of forage and concentrate in granules of a size 4x10 mm in whole rations for lambs in finalisation and its effect on the productive performance was demonstrated by Blanco *et al* (2014[a]) and Blanco *et al* (2014[b]) who found a better productive performance in lambs receiving the whole ration with the inclusion of up to 250 g/kg dry matter of barley straw in the granule, than those who received separately barley straw and concentrate.

TYPES OF CEREALS AND HOW TO MANAGE THE GRAIN

The starch of grains is the most abundant energy source for productive livestock (Svikus *et al* 2005). Chemical structure has a linear (amylose) and a branched fraction (amylopectin) as different molecular types: amylose with α1,4 links and amylopectin with α1,6 links (French 1973, Huntington 1997). Starch is the major component of energy in grains: wheat contains 77% dry matter as starch, corn and sorghum 72%, and barley and oats 57 to 58% (Waldo 1973).

The starch in the rumen is first degraded by microorganism fermentation to produce VFA; also a small portion is degraded in the small intestine (Theurer *et al* 1999). The highest total starch digestibility of the diet occurs when the portion digested in the rumen is maximized and the fraction digested in the small intestine is minimized (Boyles *et al* 2015).

Starch digestion in the rumen differs between cereals, grouped from high to low digestibility as follows: oats, wheat, barley, corn and sorghum (Herrera-Saldana *et al* 1990, Tomankova and Homlka 2004) and the average ranking of grains for the risk of acidosis is: wheat, tritricale, barley, oats and sorghum, the risk factor was obtained by the disappearance of *in saco* starch, the production of lactic acid *in vitro*, as well as the starch content in the grain (Lean *et al* 2013).

Barley has a relatively fast ruminal fermentation of starch, and for the maximum efficiency rumen acidity must be maintained at an optimum pH range of 5.8 and 6.0 (AARD 2000, Nikkah 2012). Digestibility of the whole barley grain can be 10 to 25% lower than the one crushed by dry air. The one crushed by steam, uses moisture, heat and pressure to gelatinize the starch granules (Dehghanbanadaky *et al* 2007). The treatment of barley grain with 10 g/lactic acid/L of water and heated at 55 °C has multiple beneficial effects: rumen pH is > 5.8 in the most intensive stage of fermentation, this pH stability prevents SARA and increases the fat content in milk (Iqbal *et al* 2012).

The particle size of the flakes steamed did not affect cattle behaviour; density of the flakes is the most important factor affecting the availability of starch in corn, the best density is 335 g/L (Sendi *et al* 2006, Hales *et al* 2012). The flake density reduction decreases linearly the amount of starch flowing into the small intestine (Boyles *et al* 2015). Cornflakes with a density of 335 g/L, when combined with 6% alfalfa, can produce optimal behaviour of the animal with a limited risk of altering the acid-base balance; in feedlot cattle adapted to high grain diets, ruminal pH increases with the increasing bulk density of the flakes (Hales *et al* 2012).

ADAPTATION PERIOD AND FEEDING FREQUENCY

Another way to prevent lactic acidosis in ruminants is through a period of adaptation to the new diet from forage to concentrate. Feed changes from forage to concentrate or to whole diets must be done gradually and preferably within 2 to 3 weeks (Pugh 2002) to allow rumen microorganisms to adapt to new substrates, the ruminal epithelium to modify and ruminal homeostasis maintained (Liebich *et al* 1987). The adaptation of feedlot cattle to percentage increases of the concentrate in the diet, about 55-90% of the dry matter of the diet, can be carried out in 14 days while the *ad libitum* food intake usually results in a reduction of the behaviour during the adaptation or in the entire period of finalisation (Brown *et al* 2006). In intensive lamb fattening systems, feeding is performed without restriction, lambs should always have food available in the feeders; if it is not possible to supply the food *ad libitum* it is advisable to feed twice a day, once in the morning and another in the afternoon.

When comparing two periods of adaptation, a long period of adaptation of 34 d and a short one of 8 d, ruminal acidosis was induced by restricting food intake to 50% of the dry matter intake according to body weight for 24 hours, followed by intraruminal infusion of ground barley. Regression analysis confirms that the heifers exposed to the long period of adaptation, experienced a rapid linear recovery of ruminal pH with respect to time of the induced acidosis (Schwaiger *et al* 2013).

Sheep that are fed *ad libitum* spend more time with ruminal pH <5.6 than when food was provided on a schedule at 8:00 and 16:00 (7.77 vs 3.05 hours/day). The time spent with ruminal pH <5.6 was mainly linked to the amount of food intake. The time to recover the ruminal pH to optimum condition was longer in the *ad libitum* diet (Communs *et al* 2009).

The period of adaptation of ruminants to a high concentrate diet should be managed gradually to prevent acidosis. In finishing steers two periods of adaptations were evaluated rapid *vs* gradual (3 *vs* 15 days). In the gradual adaptation, the percentage of concentrate in the diet increased from 40 to 90%, starting with values: 48.3, 56.7, 65.0, 73.3 and 81.7 for three days each of the percentages and at the end of 15

days, they were given the finishing diet of 10% forage and 90% concentrate, proving that the gradual change in diet prevents cases of acidosis (Bevans *et al* 2005).

CONCLUSIONS

Ruminal acidosis is a metabolic disorder of ruminants fed high grain rations, with small particle size and deficient in fiber, it can also occur because of errors in nutritional management. It can be prevented by the inclusion in the diet of buffer substances, zootechnical additives, essential oils and antioxidants. Rations must be whole with a minimum amount of 32% NDF, food particle size suitable from 2.83 to 3.29 mm. Slow degradation cereals should be used in the rumen as corn and sorghum, the grain must be processed by steam in the form of flakes. The change of forage to concentrate in the diet should be carried out gradually for at least 15 days. Animal welfare and production is improved, by preventing ruminal acidosis.

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