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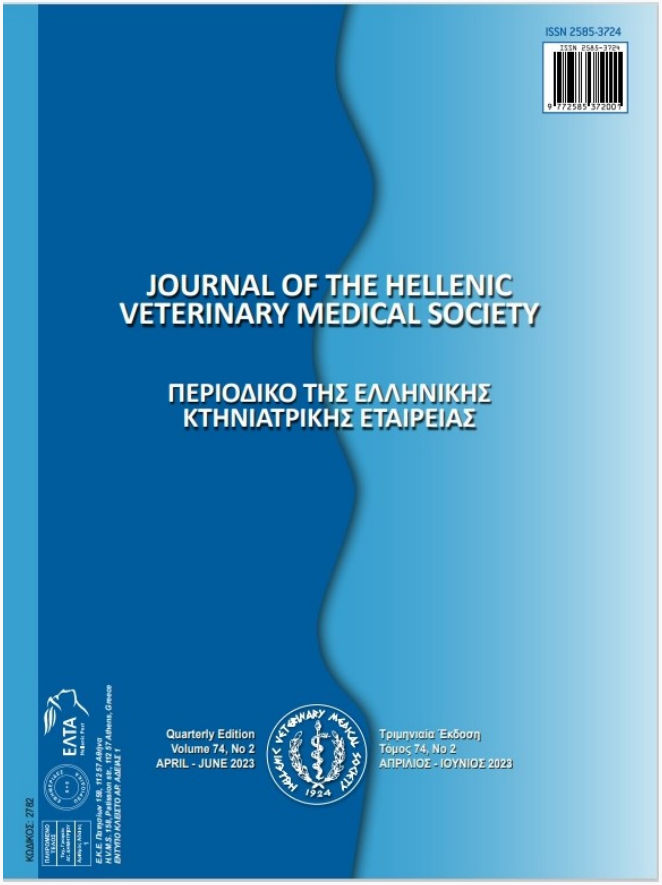

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Review article
Ανασκόπηση

Ruminal Acidosis
Part I: Clinical manifestations, epidemiology, and impact of the disease

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ABSTRACT: Ruminal acidosis is presented as the most considerable nutritional disorder of ruminants with severe impacts on animal health, animal welfare and considerable economics losses in small ruminant and bovine herds. The disease can be distinguished as acute (ARA) and subacute ruminal acidosis (SARA). Subacute ruminal acidosis constitutes the main nutritional disorder in intensive ruminant farming with several complications, such as liver abscesses, milk fat depression, reduced milk yield, and early culling. This paper constitutes the first part of a thorough review of ruminal acidosis. The first part focuses on the definition, types of ruminal acidosis, epidemiology, economic impacts and welfare implications.

***Keywords*:** ruminal acidosis; acute ruminal acidosis; subacute ruminal acidosis; milk fat; milk production; animal welfare

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INTRODUCTION

Ruminal acidosis has been introduced in the literature since 1959 with several names, such as rumen overload, toxic indigestion, grain engorgement, grain overload, grain poisoning, and acute indigestion (Krotkova et al., 1959; Dehkordi and Dehkordi, 2011). It is classified into two basic types, acute and subacute (also called latent or subclinical) based on the degree of ruminal pH reduction in the rumen and the clinical manifestations (Aschenbach et al., 2011; Reis et al., 2014). Acute Ruminal Acidosis (ARA) is usually observed in feedlots with a significant morbidity and mortality rate compared to subacute ruminal acidosis (SARA) which is frequently recorded in dairy farms and compromises farm profitability due to treatment costs, reduced herd productive and early culling (Kleen et al., 2003; Krause and Oetzel, 2006; Snyder and Credille, 2017).

Ruminal acidosis is considered to be one of the most frequent metabolic disorders especially for SARA, many authors have highlighted the considerable economic losses mainly due to reduced milk production and low body condition (Krause and Oetzel, 2006; Chaudhry et al., 2018). From an animal welfare perspective, SARA is considered a source of further complications in animal health due to consequent pathological situations such as abdominal pain and depression (Oetzel, 2003; Wang et al., 2019).

The aim of this part (I) of the review is to present a comprehensive aspect of the etiology, pathophysiology, and clinical presentation of ruminant acidosis in cattle and small ruminants.

DEFINITION

Ruminant acidosis is a ruminant nutritional disorder that emerge due to the consumption of feed with high carbohydrate and low digestive fiber content (Mahmood et al., 2013; Snyder and Credille, 2017). The pathophysiological definition of ruminal acidosis is the rapid reduction of ruminal pH due to extensive accumulation of small chain fat acids (SCFAs) (Kawas et al., 2007; Bramley et al., 2008).

TYPES OF RUMINAL ACIDOSIS

As mentioned, two main types of ruminal acidosis, ARA and SARA, have been suggested by the majority of the researchers.

In bovine medicine, physiological rumen pH varies between 6 and 7 for animals on a roughage diet (Gruenberg and Constable, 2009). In small ruminants, normal ruminal pH ranges between 5.8 and- 6.8 (Jasmin et al., 2011; Wang et al., 2019). A range of ruminal pH at 5.0-5.6 is suggestive for SARA. Ruminal pH below 5.0, approaching 4.5 or lower, characterizes the ARA (Krause and Oetzel, 2006). In small ruminants, rumen pH decreases below 5.2 in cases of ARA and between 5.2 and 5.8 in cases of SARA (Church, 1991; Commun et al., 2009).

Acute Ruminal Acidosis (ARA)

Acute Ruminal Acidosis called clinical ruminal acidosis or acute ruminal lactic acidosis (ARLA). Ruminal pH value lower than 5 is suggested as the main diagnostic feature for ARA (Commun et al., 2009; Kleen and Cannizzo, 2012). In bovine, ARA is characterized by a large accumulation of lactic acid (50-150 mM) (Blanch et al., 2009). In small ruminants during ARA, concentrations of lactic acid varying around 34 mM and total Volatile Fatty Acids (VFAs) around 94 mM (Wiryawan and Brooker, 1995).

In addition, several changes evoke in microbial populations, such as the decreased population of Gram-negative bacteria and lactic acid-utilizers, the increased population of Gram-positive bacteria (*Streptococcus bovis, Lactobacillus* spp.), the increased population of lactic acid-producers and the absence or reduction of ciliated protozoa. Furthermore, other microbial toxic products are present in cases of ARA such as ethanol, amines, and endotoxins (Wiryawan and Brooker, 1995; Nagaraja and Titgemeyer, 2007).

During ARA, blood profile is also altered with blood pH recorded below 7.35 (normal range 7.35-7.45), lactic acid increased (especially the D-lactic acid), bicarbonate decreased <20 mEq/L and packed cell volume (PCV) increased >40% (Nagaraja and Titgemeyer, 2007).

It has been well established that ARA causes damage to the epithelial barrier integrity in the ruminoreticulum and/or abomasum, which allows toxins (Lipopolysaccharide- LPS), biogenic amines and bacteria, such as *Fusobacterium necrophorum*, to infiltrate across ruminal epithelium (Tadepalli et al., 2009; Minuti et al., 2014).

Epidemiology

The acute form is considered rarer and mainly accidental compared to the subacute form (Vasconcelos and Galyean, 2008; Giger-Reverdin, 2018). The in-

cidence of ARA varies between 0.3-3% during lactation, with a considerably higher rate during the 1st-month post-calving (Krause and Oetzel, 2006; Lean et al., 2007).

Acute ruminal acidosis emerges after the significant consumption of rapidly fermentable carbohydrates such as starch, corn, wheat and barley in ruminants unadapted to a high-concentrate diet. The administration of wheat flour at a dose of 50 g/kg of body weight (BW) in sheep and a dose of 80-100 g/kg BW in goats can cause ARA (Aslan et al., 1995; Lettat et al., 2010). Similarly, ruminal acidosis can be provoked due to the unintended gross consumption of sorghum flour amounts especially for goats at a dose of 50 g/kg BW (Nour et al., 1998). Further, ARA is observed in the early feeding period when newly feedlot cattle are introduced to a primarily concentrate-based ration and stepped up too rapidly (Krause and Oetzel, 2006).

Feeding practices with a pasture of low neutral detergent fiber (NDF) and high non-structural carbohydrate (NSC) may induce acidosis (Wales et al., 2001). Diets that are deprived of forage (<10% of total Dry Matter Intake, DMI) significantly increase the risk of ruminal acidosis (Enemark et al., 2002). Ruminal acidosis may be invoked from the ingestion of small particle size (diameter less than 1.78mm) (Yansari et al., 2004; White et al., 2014). ARA is more frequent when animals are grouped than alone due to the psychological competition for overconsuming (Smith, 2009).

A great risk of acidosis for sheep is recorded when animals gain access to cropping regions especially after harvest. This risk is attributed to the availability of large amounts of grain, either remaining after harvest or split during the handling, in combination with the lack of any adaption stage. Similar issues are provoked by the rapid introduction of sheep to crops, particularly brassicas such as rapes, kales or turnips, that are highly digestible (Lean et al., 2007).

Clinical signs

Acute ruminal acidosis emerges as one of the most significant causes of increased morbidity and mortality in ruminant production systems (Plaizier et al., 2017).

The affected ruminants are presented with tachypnea, shallow respiration, diarrhea, profuse salivation, nasal discharge, abdominal respiration, and bilateral abdominal dictation. Behaviour alterations are manifested by anorexia, weakness, depression, lethargy, staggering, recumbency, flank watching, and lameness (Meyer and Bryant, 2017; Pupin et al., 2017). Clinical and laboratory testing reveal pyrexia in early stage and hypothermia in later stages, tachycardia, dehydration, congestion of mucous membranes and vessels of sclera, pneumonia, metabolic acidosis, hypocalcaemia, severe toxemia, acidified urine, and ruminal atony (Snyder and Credille, 2017; Reis et al., 2018).

Moreover, central nervous system symptoms may be presented such as ataxia, blindness, head pressing, opisthotonos, altered gait, teeth grinding, incoordination, tremors, seizures, prostration, and coma. The recumbent ruminants are perceived with their heads tucked in their flanks and similar presentation of parturient paresis (Meyer and Bryant, 2017; Sabes et al., 2017). Ruminants with a peracute form of acidosis emerge dead with few or no obvious clinical signs (Snyder and Credille, 2017).

In bovine medicine, profuse, watery foul-smelling or fetid diarrhea can be presented with a gray color and undigested grains or the presence of blood due to the increased passage and decreased digestibility (Meyer and Bryant, 2017; Snyder and Credille, 2017). In small ruminant medicine, diarrhea could be watery with yellowish color and acidic odor or greenish or soupy or just watery feces (Minuti et al., 2014; Pupin et al., 2017).

Pathoanatomical lesions

The hallmark of macroscopic examination of ruminant epithelium in ARA is easy detachment of ruminal papillae accompanied by patchy areas of sloughed papillae and multifocal erosion or ulceration (Ismail et al., 2010). In ARA and repeated ruminal acidosis episodes parakeratosis is also observed. Parakeratosis is the thickening of the *stratum corneum* and emerges with the dark color of ruminal mucosa (Enemark, 2008; Lopez-Campos et al., 2010). The ruminal papillae mucosa appears brown, friable, and easily detached. Parakeratosis often results in rumenitis due to impaired epithelial barrier caused by the decreased ruminal pH (Steele et al., 2009). Rumen content with undigested grain and sour smell and profuse amounts of fluid in the whole gastrointestinal tract are common findings in bovine with ARA (Meyer and Bryant, 2017). Nodules on papillae in both dorsal and ventral ruminal sacs, hyperaemia of papillae in *atrium ruminis*, epithelial necrosis, dyskeratosis, and acute super-

ficial ulcers have been referred in ARA cases, as well (Aslan et al., 1995; Fanning et al., 2018). On the other hand, Odongo et al., (2006) reported a reduced number of papillae on the ventral sac of the rumen during ruminal acidosis in lambs.

Ruminal fluid during ARA (intra-ruminal lactate concentration >30 mg/100 ml or 3.3 mmol/L) presents several aspects such as milky/greenish-brown or gray or yellow to orange color, sticky/sour or putrid odor and frothy consistency with contained pods and fragments. Laboratory analysis reveals watery or thin viscosity, no or fast flotation/sedimentation, pH 5.2-3.8, methylene blue reduction (MBR) test >5 min, reduced glucose fermentation test, absence or reduced protozoal population and dominance of gram-positive bacteria (Snyder and Credille, 2017; Ribeiro et al., 2020).

At the microscopical level, the most prominent lesions seen in rumen epithelium after ARA and repeated ruminal acidosis episodes are increased apoptosis of ruminal epithelium and parakeratosis (Steele et al., 2009; Minuti et al., 2014). Attenuation of the cell defense system due to acidosis is suspected to be the mechanisms that render epithelial cells more vulnerable to cellular damage and apoptosis (Steele et al., 2009; Hollmann et al., 2013). Ruminal parakeratosis is characterized by the accumulation of keratinized, nucleated squamous epithelial cells. Moreover, the cellular adhesion in the *stratum corneum* and *stratum spinosum* is weakened after ARA episodes. Large gaps among *stratum corneum* and *stratum granulosum* lead to microbial colonization of rumen epithelium and consequent infiltration of inflammatory cells (Steele et al., 2009). In small ruminants, parakeratosis in the rumen, omasum and reticulum is referred to as the main lesion with mild to severe detachment of the mucosa from the submucosa, ballooning degeneration of mucosal cells with cellular debris, swollen and vacuolated epithelia with intraepithelial pustules, necrosis, and presence of neutrophils (Bacha et al., 2017; Pupin et al., 2017).

Additionally, LPS, endotoxins, and histamine release evoke endotoxic shock, cardiovascular collapse, rumen ulceration, mycotic or bacterial rumenitis, renal failure, liver abscess, laminitis, enterotoxaemia, systemic acidemia, and thromboembolic respiratory disease (Snyder and Credille, 2017; Ribeiro et al., 2020). Lungs are characterized by congestion with severe diffuse edema (Lira et al., 2013). Liver failure, muscular weakness, and further rhabdomyolysis are aggravated due to recumbency during ARA (Constable, 2010; Fartashvand and Haji-Sadeghi, 2018).

Economic impacts

ARA emerges as a financial significant loss in the beef production industry, where the estimated treatment cost is 10 dollars/animal (Snyder and Credille, 2017). In Australia, the financial losses are estimated at 9 million dollars/year in beef feedlots (Shu et al., 2000).

A survey over six years about mortality and morbidity in feedlot cattle presents that SARA and ARA emerge with 3-7% morbidity and 14-36% mortality (Edwards, 1996). Rahman et al., (2014) reported morbidity rates at 10-50% in goats with ARA. The fatality rates are 90% for untreated animals and 30-40% for treated animals (Oliveira et al., 2009).

Subacute Ruminal Acidosis (SARA)

It is also called Subclinical Ruminal Acidosis or Latent Acidosis. The consumption of huge amounts of rapidly fermentable carbohydrates combined with inadequate fiber induce SARA, which is characterized by a low ruminal pH (5.0-5.5), an increase in VFA (150-225 mM) and a fluctuating lactate acid (10-40 mM) (Calsamiglia et al., 2012; Li et al., 2014). In the subacute form of acidosis, the clinical signs are not obvious. Several microbial changes are observed during the incident of SARA such as an increase of *Lactobacillus* spp., lactic acid-producing bacteria and lactic-acid utilizers, and the absence (or) reduction of ciliated protozoa (Nagaraja and Titgemeyer, 2007).

For definition of SARA in bovine medicine several cut-off values have been suggested based on the duration of decreased pH, such as 6.0, 5.8, 5.6, and 5.5, with the majority to agree with values below 5.6 for a period of 3h/day (Petzold et al., 2014; Bilal et al., 2016).

Several authors demonstrated as the main characteristic of subacute ruminal acidosis, in small ruminants, short periods with a pH range between 5.0-5.6 (Shen et al., 2019b). In caprine medicine, a range of 5.5 to 6.0 is used as rumen pH cut-off in SARA definition for at least 3 hours (Jiang et al., 2014; Taghipoor et al., 2020).

Furthermore, SARA is based on the presence of butyrate or propionate or both acids. The butyric form of SARA is defined as a consequence of increased corn consumption (1.2% of BW) and characterized

by a ruminal pH among 5.5-5.8, an increased butyrate percentage (from 13 to 22% of total VFA), the total value of VFA at 107 mM and a reduction of lactate concentration (<5 mM) (Doreau et al., 2001; Lettat et al., 2012).According to Lettat et al., (2010), the propionic form of SARA in small ruminants is caused by increased consumption of beet pulp (1.2% of BW) and is characterized by a mean ruminal pH among 5.8-6.2, a propionate percentage at 35%, a total VFA concentration at 110 mM and lactate concentration less than 10 mM. However, in bovine, a propionic form of SARA has as its characteristics, a mean pH of 5.67, a total VFA concentration at 114 mM, and a lactate concentration of less than 3 mM (Khafipour et al., 2009).

Epidemiology

SARA emerges commonly in intensive livestock production systems (Lopez-Campos et al., 2010; Gao and Oba, 2014). Episodes of low ruminal pH and consequent SARA in lactating cows are recorded among calving and 5 months post-calving (Kleen et al., 2013). However, the beef feedlot cattle could be exposed anytime to SARA, from the onset of their gathering into the feedlots until culling time, with pH <5.5 and prevalence of 14 to 42% (Nagaraja and Lechtenberg, 2007).

In dairy cattle, the occurrence of SARA is estimated at 11-26% with 11-20% in early and 18-26% in mid-lactation (Kitkas et al., 2013; Atkinson, 2014; Stefanska et al., 2016). A higher SARA frequency was recorded at a farm with history of high SARA prevalence in early- and mid-lactation [48.2%, 53.8% and 65.3% at 30, 90 and 150 days in milk (DIM), respectively] (Kitkas et al., 2019). The early lactation period is at high risk due to the diminished size and reduced absorption ability of rumen epithelium/papillae, fewer adapted rumen microflora and the rapid introduction to high energy-dense diets (DeVries et al., 2009). During mid-lactation, SARA emerges due to management errors such as automatic feeding misadjustment, wrong calculation of components in the preparation of total mixed ration (TMR), inconsistency in feeding time- schedule, and insufficient feed bunk space per cow (Kleen et al., 2003, Kitkas et al., 2013).

In sheep farming, similar findings were presented by Lira et al., (2013) in an extended survey in northeastern Brazil, where ruminal acidosis emerged at a frequency of 13.9% in goats and 12.4% in sheep. A cross-sectional survey of 19 dairy sheep farms in Argentina reported that 42.1% of flocks challenged presented ruminal acidosis (Suarez and Busetti, 2009).

Primiparous cows present a higher frequency of SARA (29% than 19% of multiparous cows) and occurrence earlier in lactation. The above emerged because primiparous cows are not yet capable of managing their feed intake during the consumption of a high-energy diet and are not able to have access to feed bunk for small and frequent meals due to socially domination (Oetzel, 2017; Kitkas et al., 2013).

Clinical signs

The clinical signs of SARA emerge after a delay of onset time or disguise by other common diseases, such as lameness and considerable deterioration of body condition (Colman et al., 2015).

The affected by SARA ruminants are usually presented with intermittent diarrhea, poor body condition and reduced or variable feed intake, and rumination. Behaviour alterations comprise mild transient anorexia, alternating mood, depression, and weakness. Clinical and laboratory examination present signs of dehydration, rumen stasis, abdominal pain, feces alterations, laminitis, metabolic disorders, lower fecal and urinary pH, reduced milk yield, and reduced fat and protein concentration in milk (Snyder and Credille, 2017; Shen et al., 2019a). In bovine, continuous tail swishing, cud drooping during rumination, and increased colic incidence are presented as further clinical signs of SARA (Abdela, 2016; Meyer and Bryant, 2017).

Feces may be presented brighter and of yellowish color with the sweet-sour smell. The consistency may be loose and frothy with fibrin casts, undigested particles (1-2 cm) and whole cereal grains. Significant body fecal soiling, especially in the hindquarters has been reported in the case of SARA (Meyer and Bryant, 2017; Oetzel, 2017).

Furthermore, SARA impact on bovine reproduction consists of infertility, abortions, stillbirths, premature births and being prone to environmental mastitis (Abdela, 2016; Snyder and Credille, 2017).

Pathoanatomical lesions

Macroscopic examination of the rumen wall in SARA reveals grey to dark brown mucosa and is considered a typical sign of parakeratosis and/or hyperkeratosis (Ploger et al., 2012). The major impacts of SARA are parakeratosis, thickening and detachment

of epithelium and rumenitis (Steele et al., 2009). While parakeratosis is considered a persistent finding, the thickness of the total epithelium, *stratum corneum*, *stratum granulosum* and *stratum spinosum* may appear debilitated during SARA cases as well (Steele et al., 2011; Zhang et al., 2020). The pathogenetic mechanisms responsible for these lesions are similar to those pertaining to ARA.

Furthermore, necropsy besides rumen lesions may reveal liver abscesses, subcutaneous abscesses, pulmonary bacterial emboli, peritonitis, or inflammation in organs such as kidneys (pyelonephritis), lungs (pneumonia), heart valves (endocarditis) and joints (arthritis) (Kleen et al., 2003; Abdela, 2016).

Economic impacts

According to literature cost estimate of SARA in the USA dairy industry, based on milk production losses, reduction efficiency of milk production, premature and high culling rate, rises up to 1.12 USD/day/cow. Economic losses of SARA are estimated up to 500 million - 1 billion dollars/year in total USA dairy industry (Enemark, 2008; McCann et al., 2016).

For the beef USA industry, Stock, (2000) reports a financial loss of 9.40 dollars/feedlot steer due to acidosis impact on the reduction of final cattle weight. Furthermore, the reduced DMI due to SARA leads to the decreased growth of beef calves with losses of 10-13 dollars/animals (Stock and Britton, 1996).

Effect of SARA on milk production

Administration of the HC diet (High Concentrate diet; concentrate 60%) may increase milk quality and milk yield in a short duration of time, but also may reduce rumen pH and increase the risk of SARA in dairy ruminants (Abaker et al., 2017; Shen et al., 2019a). SARA reduces milk production and milk fat concertation but has no effect on protein and energy levels (Chang et al., 2015; Xu et al., 2015). On the other hand, short-term SARA has no effect on milk fat or protein concentration (Li et al., 2012; Tian et al., 2017).

A ratio of milk fat: milk protein lower than 1 in animals feed with an HC diet, could be a useful diagnostic sign of SARA in ruminants (Enemark et al., 2002; Giger-Reverdin et al., 2014). According to Cook et al., (2005) in bovine medicine, the incidence of SARA should be suspected when 10% of cows have individual milk fat percentages of 2.5 or less. Also, Danscher et al., (2015) reported a reduction of milk fat content at 4.14% during SARA.

The estimated reduction in milk production, milk fat and true protein is 2.7-3 kg/cow/day, from 37 to 34 g/kg (0.3% points) and 29 to 28 g/kg (0.12%), respectively (Xu et al., 2016). However, the financial losses of reduced milk fat and milk protein are estimated at 400-475 dollars/cow without taking into account the early culling and veterinary expenses (Abdela, 2016).

The increase in milk Somatic Cell Count (SCC) is reported as a sign of SARA due to reduced milk production (higher cell concentration) and due to the greater inflammatory status in cases of HC diets (Shen et al., 2019a; Antanaitis et al., 2020).

WELFARE IMPLICATIONS

Several authors reported that ruminants affected with acidosis will be distinguished due to their feeding behavior, which is dependent on daily DMI, cumulative intake patterns, fractional intake rate, sorting behavior, and chewing behavior (Calsamiglia et al., 2012; Giger-Reverdin, 2018).

During SARA a higher rate of saturation or an aversion to diet can be mainly presented by reduced concentrated feed intake and increased roughage intake (Nocek, 1997; Giger-Reverdin et al., 2014). The "off-feed" periods during acidosis decrease and alter feeding intake patterns (Calsamiglia et al., 2012).

Ruminants present different patterns in dealing with ruminal acidosis based on chewing behavior for buffering rumen pH, which explains why several animals suffer acidosis more than others in a herd with the same diet. During SARA, the chewing/kg Dry Matter (DM) duration and saliva production is decreased (Giger-Reverdin et al., 2014). It is noteworthy that goats can browse rather than graze, which allows them to choose their feed in a feedstuff variety during SARA (Desnoyers et al., 2008).

Animals with acidosis prefer to nibble or eat small amounts of food. This can be explained as an effort to increase the daily intake rate and further reduce the movement of high rapidly fermentable carbohydrates amount into the rumen during acidosis (Giger-Reverdin, 2012). The total time of eating, chewing, and ruminating is decreased in goats with HC diets, due to the reduction of NDF (Maekawa et al., 2002; Desnoyers et al., 2008).

The episodes of ruminal acidosis may be manifest-

ed by a slightly total effect on cow behavior such as standing time, lying time, or feeding time (DeVries et al., 2009). Brzozowska et al., (2013) report that SARA causes discomfort which stimulates the cow to increase feeding time due to an increase in both meal frequency and duration. Lying time and rumination during SARA is decreased (by 1h/day) and standing time is increased. Furthermore, high acidosis risk (HR) cows sort their ratio to a greater level than low acidosis risk (LR) cows (DeVries et al., 2009).

Goats with SARA demonstrate a position curled up as a sign of sickness or pain mainly in the afternoon, especially after feeding hours (Desnoyers et al., 2008). On the other hand, sheep are more aggressive and less responsive, and they are devoting less time to recumbency and more time to standing with the head up posture and ears pricked up during SARA (Commun et al., 2012).

Water or salt consumption may be higher during the acidosis challenge. Salt supplementation in water causes 1.4 times more drinking water during acidosis (Commun et al., 2012).

CONCLUSION

It is generally accepted that ruminal acidosis and especially subacute ruminal acidosis emerges as one of the main restraining factors in livestock productivity. In ruminal research, topics concerning the definition and clinical manifestation are adequately investigated and presented.

On the other hand, crucial topics such as epidemiology, productivity implications, and financial consequences are still under research, especially in the small ruminant herd with still vital space in research and justification.

The necessity to define the actual prevalence and incidence of SARA in small ruminant herds is unquestionable. Methods and techniques applied in cow husbandry will provide useful information for understanding and addressing these metabolic processes in small ruminants. Additionally, the investigation of resource-based and management-based measures will provide beneficial information on the economic impacts and welfare status of dairy herds.

Undoubtedly, every sign of illness in a dairy herd could potential be an indication of acidosis and therefore as a scientific community, we should be vigilant and prepared for perceiving and assessing these metabolic issues.

CONFLICT OF INTEREST STATEMENT

There is no conflict of interest.

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