A mini review on the Lactic Acidosis in goats and its remedial approaches

Asad Ali Khaskheli¹, Muhammad Ibrahim Khaskheli², Allah Jurio Khaskheli³, Arshad Ali Khaskheli⁴

¹Department of Animal Nutrition, Sindh Agriculture University, Tando jam, Pakistan
²Department of Plant Protection, Sindh Agriculture University, Tando jam, Pakistan
³Department of Biotechnology, Sindh Agriculture University, Tando jam, Pakistan
⁴Department of Poultry Husbandry, Sindh Agriculture University, Tando jam, Pakistan

ARTICEL INFO

Keywords:
Concentrates
Intestinal motility
Lactic acid
Metabolic disorder

Received: 14 May 2020
Accepted: 10 June 2020
Available online: 16 June 2020

DOI: 10.13170/ajas.5.2.16733

ABSTRACT

The current study was performed in order to investigate the threat of lactic acidosis in goats worldwide and explores the curative strategies. In this regards a detailed review was performed, however obtained facts were found to be much interesting and valuable. It was indicated by researchers that lactic acidosis is the most common problem in goats throughout the world. It represents significant economic loss due to direct and indirect effects. It was further stated that goats with lactic acidosis show decreased body temperature up to 98.1±0.89 °F, rumen and intestinal movement 0.23±0.48/m, rumen pH 4.8±0.07, blood pH 7.1±0.08, increased respiration rate 56.14±7.15/sec and heart rate, 136.28±4.71/sec. Affected goats also show signs of dyspnea, anorexia, inactivity, incoordination and recumbancy. The glucose level remains 190.14±36.49 mg/dl, total bilirubin 0.75±0.04 mg/dl, direct bilirubin 0.27±0.03 mg/dl, indirect bilirubin 0.40±0.03 mg/dl, alanine aminotransferase ALT 36.42±3.04 U/l and alkaline phosphatase increase with treatment of Cassia Fistula, serum biochemical changes rapidly return to normal compared to treatment with Sodium bicarbonate or Magnesium hydroxide. The ruminal juices changes are also significantly improved with the treatment. The changes in the ingesta color, odor and consistency and rumen pH return to normal with the use of Cassia Fistula, Sodium bicarbonate and Magnesium hydroxide. In conclusion, Cassia fistula, Sodium bicarbonate and Magnesium hydroxide could be used as valuable strategies against lactic acidosis in goats. These therapies have been proved to be effective for treating the acidosis in goats.

Introduction

Goats are globally reared mainly for meat and milk purpose (Escareno et al., 2012). To promote the efficient growth and achieve fast weight gain, the wheat grains or its’ by products are fed in pure or mixed form. These grains are highly fermentable in the rumen part of stomach (Kamra, 2005). Fermentable grains over feed always lead to the development metabolic disorders, particularly lactic acidosis. However, ruminal acute and sub-acute lactic acidosis have significant economic impact by causing animal or indirect effects like formation of liver abscess, laminitis and rumenitis (Penner et al., 2007).

Lactic acidosis can develop with excessive intake any highly fermentable diet. Many rumen bacteria are involves but especially Lactobacillus and Streptococcus bovis (acid resistant bacteria) cause rapid fermentation of carbohydrates and changes the rumen function through proliferation and an increased lactate and volatile fatty acids production which cause the fall in rumen pH to < 5.00 (Gozho et al., 2005; Gonzalez et al., 2010). The severity of lactic acidosis depends on the type and amount of carbohydrate-rich feed consumed (Gentile et al., 2004). There are two main causes of acidosis, first the ingestion of fermentable carbohydrates and sudden increase in ruminal microbial population, second the systemic and metabolic acidosis occurs due to absorption of acids into the blood stream (Radostitis et al., 2007). Morbidity rate of ruminal acidosis varies from 10 to 50 percent in clinically affected animals, characterized by sudden increase in respiratory rate,

* Corresponding author.
Email address: khaskheliars@gmail.com

Printed ISSN 2502-9568; Electronic ISSN 2622-8734
This is an open access article under the CC - BY 4.0 license (https://creativecommons.org/licenses/by/4.0/)
heart rate, decrease body temperature, distension of abdomen, pain, anorexia, constipation or pastrys diarrhea, depression, weakness, dehydration and death if not treated (Radostits et al., 2000).

Curing the lactic acidosis has always been challenging for the goat farmers. The recovery depends on the severity of condition. Treatment of less severe cases can be possible by withholding the dietary concentrates and feeding hay or dry grasses to stimulate saliva (Karapinar et al., 2008). However, in severe cases most of therapies fails and animal dies due cardiac arrest. Keeping in view these facts about the lactic acidosis in goats, current study was planned, whereby the main aim of study was investigate world impact of lactic acidosis on goats and exploring the curative measures.

**Threat of lactic acidosis in goats**

Lactic acidosis is a metabolic disorder of the ruminants that occurs due to the feeding of carbohydrates or highly fermentable feed that may be manifested in acute or sub-acute form, it causes a significant economic losses due to direct effects in alterations of the ruminal metabolism and changes in the ruminal micro-flora that could leads to death while indirect effects results rumenitis, liver abscess and laminitis (Oliveira et al., 2009). Acute ruminal lactic acidosis commonly is known grain engorgement which is an acute disorder of the rumen. This condition always arises due to the sudden and excess ingestion of carbohydrate-rich feed such as wheat grains, flour or wheat byproducts (Lean et al., 2000).

![Figure 1. Cascade of events in lactic acidosis (Hernández et al., 2014)](image)

The amount or type of highly fermentable carbohydrate rich feed intake can directly determine the severity by clinical signs (Haji et al., 2006). Small and large animals grain feeding aggravate the condition by feed competition (Piccione et al., 2010).

Experimentally lactic acidosis may be induced for kind of study. In a study, an experiment was conducted on the goats by Ullah et al. (2012). Each of 4 adult female goats was given sucrose orally at the dose rate of 18g/kg body weight twice. Changes in rumen pH, osmolality of plasma and blood were examined for 48hrs and it was found that the decrease in rumen pH (<5.00) was prominent (Figure. 1). Further, it has been studied that decrease of rumen pH, increases the ruminal mucosa to allow the body compartment fluid to enter into the rumen compartment from blood vessels that leads to severe dehydration with the passage of time (Aschenbach and Gabel, 2000). In lactic acidosis presenting goat rumen contents were examined under low power lens and seen 5-7 protozoa per lens field normally but at 5 pH or below these protozoa were absent in lactic acidosis (Annane, 2002). Table 1 is indicating the clinical signs of lactic acidosis in goat.

**Table 1. Clinical signs of lactic acidosis in goats**

| Sample No. | Clinical aspect | Average range |
|------------|-----------------|---------------|
| 1          | Body temperature | 98.1±0.89°F    |
| 2          | Heart rate/min   | 136.28±4.71/m  |
| 3          | Respiration rate/min | 56.14±7.15/m  |
| 4          | Ruminal motility | 0.23±0.48/m    |
| 5          | Rumen pH         | 4.8±0.07       |
| 6          | Rumen ingesta color | Yellowish     |
| 7          | Rumen ingesta odor | Soured         |
| 8          | Rumen consistency | Watery         |
| 9          | Appetice         | Anorexic       |
| 10         | Regurgitation    | Absent         |
| 11         | Behavior         | Dull           |
| 12         | Urination        | Absent         |
| 13         | Feces            | Absent         |
| 14         | Gait             | Staggering     |
| 15         | Blood pH         | 7.1±0.08       |
| 16         | Hb%              | 15.02±1.30     |
| 17         | Glucose (mg/DL)  | 190.14±36.49   |
| 18         | Total bilirubin (mg/DL) | 0.75±0.04 |
| 19         | Direct bilirubin (mg/DL) | 0.27±0.03 |
| 20         | Indirect bilirubin (mg/DL) | 0.40±0.03 |
| 21         | ALT (SGPT) (U/L) | 36.42±3.04     |
| 22         | Alkaline phosphatase (U/L) | 420±3.65 |

Acute lactic acidosis in goat causes rapid proliferation of acid resistant bacteria (Lactobacillus and Streptococcus) and directly an increase in the production of volatile fatty acids (VFA) and lactate, which causes drop in rumen pH to (<5.00) in most severe cases (Aziz et al., 2017). Neurologic symptoms in lactic acidosis goats including depression, anorexia, blindness, convulsions and incoordination have been recorded most common (Abeysekara et al., 2007). In carbohydrate engorgement other clinical which develops are abnormal distension of rumen, atony of
ruminants. The ruminal fermentation process, driven by a diverse microbial community, produces gases (e.g., methane, carbon dioxide) and byproducts such as lactic and organic acids.

Microscopic examination of rumen fluid revealed the presence of various microorganisms, including Gram-positive and Gram-negative bacteria, protozoa, and fungi. The pH of the rumen fluid is crucial for maintaining the health of the rumen ecosystem, as it affects bacterial growth and the fermentation process. A decrease in rumen pH below 5.5 can lead to acidosis, a metabolic disorder caused by feeding errors in ruminants. One of the most common causes of acute acidosis is dietary changes, particularly the inclusion of highly fermentable carbohydrates in the diet, which can lead to a rapid increase in lactic acid production.

The rumen is a complex ecosystem that is influenced by various factors, including diet, environmental conditions, and management practices. Maintaining a balance in the rumen microbiota is essential for optimal animal performance. The rumen microbiota plays a crucial role in the digestion of plant materials, producing essential volatile fatty acids and enzymes that aid in the breakdown of fibrous feed components. The rumen flora also influences the animal's immune system and plays a role in the production of certain vitamins and hormones.

Acute acidosis is a metabolic disorder caused by feeding errors in ruminants, characterized by a rapid increase in lactic acid production in the rumen. This condition can be triggered by dietary changes, such as the sudden addition of high-concentrate feeds, and is often associated with a decrease in rumen pH below 5.5. Acidosis can lead to a series of physiological changes, including increased heart rate, respiratory rate, and body temperature. The severity of the condition can range from mild to severe, with severe acidosis leading to acid-base disturbances, dehydration, and other complications.

The rumen microbiota is a dynamic community that responds to changes in the diet and environmental conditions. Understanding the interactions within this ecosystem is crucial for developing strategies to maintain rumen health and productivity. The rumen microbiota plays a key role in the digestion of plant materials, producing essential volatile fatty acids and enzymes that aid in the breakdown of fibrous feed components. The rumen flora also influences the animal's immune system and plays a role in the production of certain vitamins and hormones.
mean corpuscular volume (MCV) was recorded. The mortality rate of the disease was recorded with the severity than sub-acute ruminal acidosis (SARA). The similar study was conducted by Minuti et al. (2014) to investigate the induced acute rumen lactic acidosis in sheep. Sheep were kept off feed before the induction of lactic acidosis, after 24hrs of wheat flour 0.5 kg intake orally at 0h and 12hrs, after the experimental period of 96hrs, animals were offered dry grasses and hay. At 24hrs, rumen, fecal and blood samples were collected. Samples collected at 24hrs showed markedly (P < 0.01) reduce in lactic acidosis but rumen d- and l-lactic acid were increased. A significant level of fecal pH and (VFA) was observed. The increase level of lactulose in blood of acidosis animals showed increase gastrointestinal permeability after 2hrs and increased the lactate-producing bacteria Streptococcus bovis in rumen.

**Figure 2.** (A) Ruminal content composed of voluminous and moderate amount of white-gray liquid with a greasy appearance, (B) Hydropic degeneration of keratinocytes at the top of the ruminal papilla and a mild multifocal infiltrate of neutrophils, (C) Marked necrosis of the epithelium associated with marked inflammatory infiltrate that extends to the lamina propria, (D) Necrosis of the epithelium; inflammatory infiltrate of neutrophils, lymphocytes, and plasma cells (Bonadiman et al., 2018)

**Table 2.** Effect of Magnesium hydroxide, Sodium bicarbonate and Cassia fistula on the clinical signs of lactic acidosis

| Clinical signs | Magnesium hydroxide (400gm/50kg) | Sodium bicarbonate (400 gm/50kg) | Cassia fistula (30g/50Kg) |
|----------------|----------------------------------|----------------------------------|---------------------------|
| Rectal temperate / min | 101.41±0.76b | 102.97±0.41a | 101.36±0.72b |
| Heart rate/min | 99.28±5.46b | 83.14±5.17c | 100.62±6.32b |
| Respiration rate/min | 45.14±3.80b | 27.42±5.56c | 41.75±3.95b |
| Rumen motility/min | 2±0.40b | 3.07±0.53a | 2.06±0.41b |

**Figure 3.** Key reactions occurring during lactic acidosis in ruminants (Owens et al., 1998)
Curative strategies against lactic acidosis

The carbohydrate rich feeds cause severe clinical signs of lactic acidosis. The severity of ruminal acidosis can be decreased by using the alkalinosis producing agents or compounds that neutralize the acidic pH of rumen. Magnesium hydroxide is an effective antacid used in ruminants as alkalinizing agent (Ding and Yu, 2011). Magnesium hydroxide at the dose rate of 400gm can effectively treat lactic acidosis in goat. For achieving the therapeutic effects, Magnesium hydroxide is dissolved in 10 liters water and the solution is infused into rumen by using stomach tube. Given solution remains very effective in neutralizing the acids (Bashir et al., 2004). The vitamin B1 (Thiamine) is an important vitamin for the normal body functions (Harper, 2006). Oral administration of vitamin B1 (Thiamine) restores the function of the ruminal cells and increase the tissues replenishing thiamine deficiency and promotes metabolism of excess lactic acid of rumen by reducing acidosis (Cottee et al., 2004). In another study, it was found that sodium bicarbonate is an important buffering agent. It is used in veterinary medicine to increase the ruminal pH and it is also used as additives in many products as a buffering agent for the rumen that prevents ruminal acidosis the animals which consume high-grain diets. It also improves the productive performance of feedlot animals (Chand et al., 2016). Sodium bicarbonate orally drenched twice daily for two days at the dose rate of 400 gm/50kg has shown more effective results in goats to recover from acute phase of lactic acidosis (Joaquin et al., 2014). Further animals observed on intake of roughages diet show a range of ruminal pH 6 to 7, while animals on grain or mixed concentrated feed have been observed with pH 5.5 to 6 (Yang et al., 2012). Number of protozoa varies from animal to animal and changing in rumen protozoa depend on feed intake, seasonal variations of the year, time of the day, the number of normal protozoa and the frequency of healthy ruminant, the normal goats has been found with total number of protozoa 6.50±0.96, 105/ml in rumen fluid and differential sum of Entodium 75.09±7.60%, Diplodium 13.01±3.99%, Isotricha 5.61±2.08%, Dusytricha and Ophryoscolex 5.68±2.36%, the pH of rumen liquor decreased in acidosis, low rumen pH leads in changing rumenal protozoa (Lascano et al., 2011). Callaghan et al. (2016) conducted an experiment to observe the total serum proteins in SARA (Sub-Acute Ruminal Acidosis). They evaluated the variations of Acute Phase Proteins (APPs), blood parameters and pathological lesions. A total of 108 cows from 12 farms were randomly selected and divided into three groups comprising of 36 animals in each group, all cows were exposed to a rumenocentesis. Group “A” was selected with a rumen pH>5.8, group “B” was presented with a rumen pH ≤5.5≤5.8 and group “C” was observed with a rumen pH<5.5. Blood serum samples were evaluated for serum amyloid A (SAA), total proteins (TP), albumin (Alb), haptoglobin (Hp), and white blood cells (WBC). The selected time data analyzed on one way ANOVA showed a statistical significance on rumen pH, SAA and Hp. The APPs production from liver was not stimulated in SARA. Table 2 indicates the comparative influence of Magnesium hydroxide, Sodium bicarbonate and Cassia fistula on the clinical signs of lactic acidosis.

In another study, Zein-Eldin et al. (2014) observed the clinical, haemato-biochemical and ruminal changes from the induction of lactic acidosis to recovery in sheep. Five sheep were induced lactic acidosis with sucrose and treated with Sodium bicarbonate and ruminal yeast as probiotics and gential root powder. Sheep showed decrease in body temperature and increase in the respiration and pulse rate significantly (P<0.05), while other signs were recorded like anorexia, depression, reluctance to move, acute general weakness, holding their heads at lower position and passing soft feces. All sheep were improved after the treatment with Sodium bicarbonate and amino yeast mixture and there was slow improvement by the treatment of gential root powder. It was concluded that treatment of lactic acidosis in sheep using sodium bicarbonate and yeast help rapidly recovery from lactic acidosis.

Conclusions

Present study concludes that lactic acidosis is common threat to the goat worldwide. It results substantial economic loss to famers either by impairing the normal physiology of animal or by posing the animal to the death. It is further concluded that Cassia fistula (commonly known as Amaltas), Sodium bicarbonate and Magnesium hydroxide could be used as effective strategies against lactic acidosis in goats. Cassia fistula administration in lactic acidosis increases the ruminal and intestinal motility and rapidly restore all physiological parameters to normal level of the. Cassia fistula is effective laxative to expel the ruminal ingesta containing grain and protect liver. On other hand Sodium bicarbonate and Magnesium hydroxide are also helpful in restoring normal rumen and blood pH.
Declarations of interest

The author(s) declare that there is no conflict of interest with regards to the research, authorship and/or publication of this article.

References

Abcsselera, S., J.M. Naylor, A. Wassef, U.V. Sak, G.A. Zello. 2007. D-Lacticacid-induced neurotoxicity in a calf model. American Journal Physiology and Medicine, 293: 558-565.

Anjane, D., V. Sehilde, C. Charpentier. 2002. Effect of treatment with low doses of hydrocortisone and hydrocortisone on mortality in patients with septic shock. American Journal Veterinary Medicine, 7: 862-871.

Aschenbach, J., R.G. Gabel. 2000. Effect and absorption of histamine in sheep rumen significance of acidic epithelial damage. Journal Animal Science, 78: 464-470.

Aziz, M., A. Khan, A.H. Adnan, M. Izaullah. 2017. Traditional uses of medicinal plants reported by the indigenous communities and local herbal practitioners of Bajaur Agency, Federally Administered Tribal Areas Pakistan. Journal of Ethno Veterinary Medicine, 19: 268-281.

Bashir S., T. Hussain, M.A. Dar, A.A. Dar, A.A. Bhat, J. Farooq. 2004. Ruminal lactic acidosis in cow clinical assessment. Journal of Livestock Science, 6: 73-76.

Bonadiman, H.B., C.I. Schwertz, S.H.D. Sousa, R.M. Bianchi, R.A. Caprioli, A.G.C. Dalto. 2018. Acute rumenitis due to lipid overload in a bovine. Ciencia Rural, 48(9): 1231-1244.

Callaghan, M., M.J. Auliffe, P. Rodgers, R.J. Hernandez-Medrano, V.E.F. Perry. 2016. Sub-acute ruminal acidosis reduces sperm quality in beef bulls. Journal of Animal Science, 8: 3215-3228.

Chand, R., R. Kaur, A. Kaur, V. Kumar, C. Nirmala, A.N. Singh. 2016. Assessment of ethnomedical plant diversity of Una and Hamirpur district of Himachal Pradesh, India: an ethnocoological approach. Journal of Animal and Plant Science, 12: 1475-1490.

Cottee, G., I. Kyriazakis, T.M. Widowski, M.I. Lindlinger, J.P. Cant, T.F. Duffield, V.R. Osborne, B.W. McBride. 2004. The effects of subclinical ruminal acidosis on Sodium bicarbonate supplements water intake for lacting dairy cows. Journal of Dairy Science, 7: 2248-2253.

Dehklordi, A., Z.K. Dehklordi. 2011. Occurrence of metabolic alkalosis in rumen lactic acidosis a review article. Journal of Clinical Pathology, 1: 1-3.

Ding, Z., Y. Xu. 2011. Physiological, biochemical and histopathological effects of fermentative acidosis in ruminant production: A minimal review. Spanish Journal of Agriculture Science, 2: 414-422.

Escareno, L., H. Salinas-Gonzalez, M. Wurzinger, L. Inguez, J. Sulkner, C. Meza-Herrera. 2012. Dairy goat production systems. Tropical Animal Health and Production, 1: 17-34.

Gentile, A., S. Scona, I. Lorenz. 2004. D-Lactic Acidosis in Calves as a Consequence of Experimentally Induced Ruminal Acidosis. Journal of Veterinary Medicine, 51: 64-70.

Gonzalez, F., J.M. Ruizper, J.C. Sanchez, S. Souza, M. Martinez-Sabiula, J.J. Ceron. 2010. Haptoglobin and serum amyloid A in subacute ruminal acidosis. Reviews of Veterinary Medicine and Zoology, 57: 159-167.

Gozho, G., N.J.C. Plazier, G.O. Krause, A.D. Kennedy, K.M. Witternberg. 2005. Sub-acute ruminal acidosis induces ruminal lipopolysaccharide release and triggers an inflammatory response. Journal of Dairy Science, 88: 1399-1403.

Haji, H., M.R.M. Kolace, F. Nouri, A. Saberi, A. Jafari, M. Dehklordi. 2006. Effects of Experimentedly Induced Ruminal lactic acidosis Blood pH, Bicarbonate and in the sheep. Pakistan Journal of Bioscience, 4: 9-10.

Handekar, P., B. Kolte, A.Y. Mendle, H.C. Puri, R.M. Ravikanth, S. Maini. 2010. Effect of Polyherbal formulations on ruminal digestion in goat. Veterinary World, 5: 230-233.

Harper, C. 2006. Thiamine (vitamin B1) deficiency and associated brain damage is still common throughout the world and prevention is simple and safe. European Journal Neuroscience, 10: 1078-1082.

Hernández, J., J.L. Benedito, A. Abuelo, C. Castillo. 2014. Ruminal acidosis in feedlot: from aetiology to prevention. World Journal, 10: 123-130.

Hussain, S., R.N. Malik, M. Javid, S. Bibi. 2008. Ethnomotanical properties and uses of medicinal plants of Morgah biodiversity park, Rawalpindi. Pakistan Journal of Botany, 5: 1897-1911.

Joaquin, H., L.B. Jose, A. Angel, C. Cristina. 2014. Ruminal Acidosis in Feedlot from Aetiology to Prevention. Journal of Veterinary Medicine, 4: 2018-2028.

Kamra, D.N. 2005. Rumen microbial ecosystem. Current Science, 1: 124-135.

Karapinar, T., M. Dalab, O. Kizil, E. Balikci. 2008. Severe thiamine deficiency in sheep with acute ruminal lactic acidosis. Journal of Veterinary Internal Medicine, 3: 662-665.

Kawas, J., R. Garcia-Castillo, H. Fimbres-Durazo, F. Garzacazaeres, J.F. Hernandez-Vidal, E. Olivares-Saenz. 2007. Effects of Sodium bicarbonate and yeast on nutrient intake, digestibility, and ruminal fermentation of light-weight lambs fed finishing diets. Small Ruminant Research, 67: 149-156.

Kleen, J., G.A. Hooijer, J. Reyhage. 2003. Subacute ruminal acidosis (SARA): A review. Journal of Veterinary Medicine Series, 50: 406-414.

Lascano, G.J. 2011. Optimizing nutrient utilization of a precision feeding system for dairy heifers using low and high forage diets. Veterinary Medicine Series, 40: 409-419.

Lean, I.J., L.K. Wade, M.A. Curtis, J. Porter. 2000. New approaches to control of ruminal acidosis in dairy cattle. Asian-Australasian Journal Animal Science, 13: 266-269.

Minuti, A., S. Ahmed, E. Trevisi, F. Piccioli-Cappelli, G. Bertoni, N. Jahan, P. Bani. 2014. Experimental acute rumen acidosis in sheep: Consequences on clinical rumen, and gastrointestinal permeability conditions and blood chemistry. Journal of Animal Science, 9: 3966-3977.

Oliveira, D.M, J.M.A. Medeiros, A.C.O. Assis, A.B. Neves, G.J.N. Galiza, S.V.D. Simoes, A.F.M. Dantas, F. Riet-Correa. 2009. Acidose lática ruminal aguda em caprinos. Ciência Animal Brasileira, 117-122.

Owens, F. N., D.S. Secrist, W.J. Hill, D.R. Gill. 1998. Acidosis in cattle: a review. Journal of animal science, 76(1): 275-286.

Permer, G., B.K.A. Beauchemin, T. Mutsvangwa. 2007. Severe of ruminal acidosis in primiparous Holstein cows during the periparturient period. Journal of Dairy Science, 90: 365-375.

Piccione, G., S. Casella, L. Luriti, I. Vazzana, V. Ferrantelli, G. Caola. 2010. Reference values for some hematological, hematochemical and electrophoretic parameters in Girgentana goat. Turkish Journal of Veterinary and Animal Science, 34(2): 197-204.

Radostits, O., M.C.C. Gay, K.W. Hinchliff, P.D. Constable. 2007. Veterinary Medicine, A textbook of the diseases of cattle, horses, sheep, pigs, and goats. Saunders-Elsevier (USA). pp: 898.

Radostits, O.M., C.C. Gay, D.C. Blood, K.W. Hinchliff. KW. 2000. Rumen Acidosis. In: Veterinary Medicine, 9th Edition: Saunders, Elsevier, London, pp. 293-303.

Ullah, H.A., J.A. Khan, M.S. Khan, U. Sadique, M. Shah, M. Idrees, Z. Shah. 2013. Clinico-therapeutical trials of lactic acidosis in small ruminants. Journal of Animal and Plant Sciences, 23: 80-83.

Yang, W.Z., Y.L. Li, T.A. McAllister, J.J. McKinnon, K.A. Beauchemin. 2012. Wheat distillers grains in feedlot cattle diets: Feeding behavior, growth performance, carcass characteristics, and blood metabolites. Journal of Animal Science, 4: 1301-1310.

Zein-Eldin, M.M., M.M. Ghanam, A.Y. El-Raof, H.M. El-Attar, H.M. El-Khiait. 2014. Clinical, haematobiochemical and ruminal changes during the onset and recovery of induced lactic acidosis in sheep. Journal of Biology and Animal Husbandry, 4: 647-659.