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Acidosis in ruminants: Nutritional overview

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Abstract
Ruminal diseases, which occur during the transition period, can be prevented in part by nutrition. The ruminal disorders ketosis, acidosis, tympany, and milk fever have a negative impact on dairy animals' output. These issues arise in high yielding animals, and nutritional approaches can assist in resolving them. The purpose of this review was to investigate the connections between feeding practices and animal welfare, health, and production. Ruminants with metabolic disorders experience severe health and economic issues as well as significant financial loss. Despite significant management efforts, metabolic problems continue to be the primary cause of animal death, sluggish weight increase, and low milk supply. It may be concluded that controlling the transition cow to reduce metabolic problems can reduce ruminal disorder, which is not a pathogenic problem but a management issue during the transition phase.

Keywords: Ruminants, nutrition, ketosis, acidosis, bloat, milk fever

Introduction
Acidosis is a common digestive disorder in ruminants that are heavily fed on concentrated diets. Ruminal acidosis or increased accumulation of organic acids in the rumen reflects imbalance between microbial production, microbial utilization, and ruminal absorption of organic acids. Acidosis occurs when there is more lactic acid production than the capacity of ruminal microbes to utilize it. Amount and frequency of grain/concentrates feeding reflects the severity of acidosis. Two types of acidosis are observed in ruminants i.e., acute, and chronic acidosis. The response of animals is inconsistent with readily fermentable carbohydrates. Acidosis is a major problem affecting the production performance of animals. But the physiological basis of this has not been well understood. Rumen is a complex consortium of various microbes. It is a highly reduced and anaerobic ecosystem in which microbial digestion of feedstuffs converts fermentable substrates into organic acids, which are then removed primarily by absorption and utilized as a source of energy. These microbes depend on the availability of feed materials (substrates) from which they synthesis amino acid as per their requirements. Rate of fermentation in rumen depends on the pH which is maintained around 6.2-6.8. Ruminal pH is the function of feed intake especially carbohydrate and absorption of various organic acids and thus vary within a day. The topic of acidosis in ruminants has been reviewed extensively by Counotte and Prins, 1981; Britton and Stock, 1989; Nocek, 1997; Owens *et al.*, 1998; Krause and Oetzel, 2006 [6, 5, 19, 20, 16]. In the present article nutritional overview is summarized.

The major factor determining the severity of acidosis is pH, pH below 5.5 results in acidosis. In sub-acute or chronic acidosis rumen pH falls in the range of 5.0-5.5. Acute acidosis is the condition when pH of rumen fluid falls below 4.5 (Britton and Stock, 1989; Owens *et al.*, 1998; Krause and Oetzel, 2006) [5, 20, 16]. In acute acidosis, fall in ruminal pH is due to the excess accumulation of organic acids. Consequently, decreased concentration of total volatile fatty acids observed as normal rumen microflora diminished. Research suggests that non-dietary physiological factors, such as capacity for absorption of acid primarily regulate the pH in rumen (Aschenbach *et al.*, 2011) [3].

Etio-Pathogenesis
Common causes of acidosis are readily available sources of soluble carbohydrates, low fibre in rations, finely grounded forage, shifting of animals from a high fiber to high concentrate diet that is rich in fermentable carbohydrates (starches and sugars). In ruminants, fermentation of soluble carbohydrates produces lactic acid which is efficiently utilized by lactic acid utilizing bacteria. When soluble carbohydrates are speedily consumed by ruminants then the rate of lactic acid production exceeds the rate of utilization resulting in acidosis. At the same time hurriedly, consumption causes less amount of saliva entering in the rumen which has buffering

activity due to the presence of carbonates and bicarbonates. Thus, saliva is not passed into rumen at the proper rate resulting in increased acidity of rumen and pH of the rumen is not maintained around 6.2-6.8. Large amounts of starch and sugar stimulate the multiplication of lactic acid producing bacteria. Consequently, bacteria that normally utilize lactic acid are unable to utilize it as the rate of production of lactic acid is higher. This is adversely affecting the population of cellulolytic microbes and protozoa. The osmolality of the ruminal fluid is increased which is attributed to an increased concentration of lactic acid and stimulates the flow of water from the bloodstream to rumen resulting in dehydration. Rumen motility also decreases as rumen pH falls, sometimes motility even stops. This might be due to a higher concentration of butyric acid, but lactic acid probably exerts a reflex inhibitory action on entering the duodenum. A significant decrease in water absorption from colon results in diarrhoea (Lee, 1977) [2]. Consumption of grains causes proliferation of coliform and *Clostridium* species of microbes causing diarrhea. In mild lactic acidosis, the plasma buffering system works well, and animals may show recovery but in acute acidosis, all systems to neutralize acidity fail. The reduced pH of ruminal fluid is favorable condition for growing and multiplication of fungi such as *Mucor, Rhizopus, Absidia* sp. which further invades blood vessels and produces thrombosis and infarction and spread to liver (liver abscess) apart from severe ruminitis produced. Ruminitis along with toxemia is reported in acute acidosis which is fatal. In chronic acidosis, mild ruminitis along with hyperkeratosis may develop. Thus, it is recommended for dairy and fattening animals that concentrate mixture should be gradually increased over a period of 2 months and there should not be a sudden shift to high concentration diets.

The optimal rumen pH should be 6.2 - 6.8. Lactic acid is about 8-10 times stronger acid than the other rumen acids and causes the rumen pH to decrease (acidic). As the rumen pH drops below 5.8, bacteria that digest fiber begin to die and thus, fiber digestion is depressed. Because the end products of fiber digestion are used for milk fat synthesis, a drop in milk fat is a sign of acidosis. If the rumen pH continues to decline and falls below 5.5, many other beneficial rumen bacteria also begin to die. As lactic acid accumulates, it is absorbed and lowers the pH of the blood also. High levels of acid can also cause ulcers in the rumen resulting in infiltration of bacteria into blood that causes liver abscesses. Endotoxins resulting from high acid production in the rumen also affect blood capillaries in the hoof, causing them to constrict resulting in laminitis. Common symptoms of acidosis are dullness, falling on the ground, bulging of rumen with doughy sound on palpation, reduced rumination, diarrhoea, acidic pH of rumen contents, liver abscess, laminitis, and reduction in milk fat percent. Symptoms also include loss of appetite, even affected animals refused to drink water, soft and watery faeces with sweet-sour smell, undigested feed particles may also see in faeces. Subnormal temperature, increased heart and respiration rate are reported (Nocek, 1997) [19]. Recent research findings indicate that rumen acidosis negatively affects nutrient digestibility and animal health (Gressley *et al.*, 2011) [12] and endotoxins released can also cause systemic inflammatory responses (Andersen, 2010) [1] and laminitis. Some researchers reported that acidosis and laminitis are commonly seen the areas which have a prevalence of mycotoxins. This probably might be due to alteration in lactic acid metabolism which causes accumulation of lactic acid leading to acidosis. This needs to be validated through further research in this direction. Ruminal fluid is withdrawn with the help of needle and syringe from the flank region (rumenocentesis) and the acidity of rumen fluid can be measured either by pH strip or pH meter which indicates lower pH of rumen fluid, absence of rumen protozoa is seen microscopically. Some biochemical tests are used for diagnosis acidic urine, increased lactate, inorganic phosphorus and reduced bicarbonate in blood and marginal hypocalcaemia. Diagnosis should be based on clinical signs and history and confirmed by above mentioned tests. Acidosis can be differentiated from milk fever in which firm and dry faeces are seen than former which showed soft with fluid pockets in faeces.

Acidosis and microbial changes in rumen

In rumen, the rate of bacterial growth and fermentation is increased in response to availability of readily fermentable substrates leading to increased VFA production. When readily fermentable sources of carbohydrates are available abundantly, then bacterial changes occur. These are primarily starch and soluble sugar fermenting bacteria and lactic acid producing bacteria (Nagaraja and Titgemeyer, 2006) [18]. Lactate is an intermediate product of ruminal fermentation. This lactate is again metabolized to volatile fatty acids. The adaptation of the rumen to the high grain diet (concentrates) results in increased populations of bacteria capable of utilizing lactic acid (Huber *et al.*, 1976; Counotte and Prins, 1981) [14, 6]. Major lactate utilizing bacteria are *Megasphaera elsdenii* and *Selenomonas ruminantium* in concentrates fed animals (Huber *et al.*, 1976) [14]. Decline in pH of ruminal contents is mainly due to lactic acid produced by *Streptococcus bovis*, which in turn inhibits growth of other beneficial bacteria in rumen leading to acute acidosis. Thus, acid tolerant *Lactobacilli* becomes more predominant. Therefore, *Streptococcus bovis* is a major etiologic agent of acute acidosis. Intervention to prevent acidosis includes strategies such as antibiotics and vaccines are often targeted at controlling the growth of *Streptococcus bovis* in the rumen (Gill *et al.*, 2000) [10]. *Lactobacilli* are more resistant to low pH than *Streptococcus bovis*, which is why they are more dominant in the acidic rumen (pH < 5.6). A considerable increase in the population of ruminal *lactobacilli* is a common feature of both acute and sub-acute acidosis (Slyter, 1976; Nagaraja and Miller, 1989; Goad *et al.*, 1998) [22, 18, 11].

The number of ciliated protozoa is reported to increase on moderate feeding of concentrates (Dennis *et al.*, 1983). However, an increased amount of concentration results in complete elimination of protozoa (Towne *et al.*, 1990ab; Franzolin and Dehority, 1996; Goad *et al.*, 1998) [23, 24, 8, 11]. It is known that reduced pH in rumen causes destruction of protozoa (defaunation) as they are more sensitive to pH changes (Whitelaw *et al.*, 1984) [26]. Some ciliated protozoa *viz. Entodinium, Polyplastron, Isotricha,* and *Dasytricha* (Towne *et al.*, 1990a) [23] are resistant to low pH in rumen. The genus *Entodinium* is the most resistant to low ruminal pH. That is why the genus *Entodinum* is the most dominant (about 90 to 99%) of the total protozoal population, in cattle fed on concentrate diets (Towne *et al.*, 1990 ab; Franzolin and Dehority, 1996; Hristov *et al.*, 2001) [23, 24, 8, 13].

Laminitis

Decarboxylation of amino acids results in the production of histamines, tyramines, tryptamine etc. which are believed to

cause laminitis. Reduction in pH of rumen causes the accumulation of histamine (Van Der Horst, 1961; Irwin *et al*., 1979) [25, 15]. The major factor responsible for laminitis is the destruction of normal haemo-dynamic processes by histamines (Nocek, 1997) [19]. Brent (1976) [4] reported that histamine is a potent vasodilator that increases capillary permeability thus resulting in laminitis. Rodwell, (1953) [21] assumed that ruminal *lactobacilli* are the main producer of histidine decarboxylase. But recently, Garner *et al*. (2002) reported that *Allisonella histaminiformans* a gram-negative and ovoid species, is an important histamine producer in the rumen. This bacterium is acid tolerant and grows even at a pH of 4.5. The increase in absorption of histamine may be because of increased epithelial permeability or decreased catabolism in the epithelial cells.

Nutritional control measures

Control measures primarily include feed additives that can inhibit lactate producing microbes and stimulates the activity of lactate utilizing bacteria and ruminal protozoa. Inoculation of rumen with microbes prevents the accumulation of glucose and lactate, and they can also metabolize lactate at low ruminal pH. Maintenance of pH is very important to restore fiber digestion. This can be done by increasing buffering capacity of rumen, use of slowly fermentable polysaccharides and microbial feed additives. Incidences of acidosis can also be reduced by proper grain processing, salivary flow stimulants, feeding management and feed additives. Feeding more roughages and limiting the quantity of concentrates reduces the incidences of acidosis. Avoiding a sudden switch to more concentrated ration will be a good start for the prevention of acidosis.

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