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Lactic Acidosis: A Major Metabolic Disorder in Cattle

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Abstract

For ruminants fed diets high in concentrate, acute and chronic acidosis conditions that result from consuming excessive amounts of rapidly fermented carbohydrates are major production issues. Chronic acidosis can persist throughout the feeding period and is frequently experienced during adaptation to concentrate-rich diets in feedyards. As acids and glucose build up in acute acidosis, ruminal acidity and osmolality rise noticeably; these can harm the intestinal and ruminal walls, lower blood pH, and result in potentially lethal dehydration. Acidosis is frequently accompanied by laminitis, polioencephalomalacia, and liver abscesses. The incidence of acidosis should be decreased by feeding more dietary roughage, digesting grains less completely, and restricting the volume of feed, however these methods frequently impair performance and economic efficiency. New approaches to lowering the frequency of acute and chronic acidosis should result from ongoing research on grain processing, dietary cation anion balance, narrow-spectrum antibiotics, bacteria that use glucose or lactate, and feeding management (limit or program feeding).

Introduction

Acidosis is defined as a reduction in the alkali (base excess) content of bodily fluids in comparison to the acid (hydrogen ion) content (Mansilla *et al.,* 2022). Because bicarbonate buffers the pH of bodily fluids, the degree to which bicarbonate compensation is feasible will determine whether or not the pH of bodily fluids is lowered during acidosis. Low bicarbonate concentrations can disrupt central nervous system function even in cases where blood pH is not lowered (Passos *et al.,* 2023). The standard diagnostic indicators of acidosis in feedlot cattle include ruminal pH, anorexia, fluctuating feed intake, diarrhea, and lethargy, even though a clinical diagnosis of acidosis needs blood pH to drop below 7.35. Harmon (1996) has provided comprehensive reviews on the etiology of ruminal and systemic acidosis. Highlights and particular

hazard control locations where changes could help avoid or lessen acidosis are shown below.

Etiology of Acidosis:

I) Concentration of Starch and Its Conversion to Glucose

The most common cause of acidosis is the engorgement of significant quantities of starch or another quickly fermented food. When animals are first acclimated to a high-concentrate (feedlot) diet or are transitioning from bulk fill to chemostatic intake management, they frequently consume excessive amounts of rapidly fermented starch. When grazing animals are given a lot of a starch-rich supplement, acidosis can also happen.

ii) Restricting the Availability of Glucose and Starch

Modulating carbohydrate consumption or diluting the diet with roughage are two popular treatment strategies that help prevent acidosis. Meal size and eating rate are reduced by dietary roughage. Saliva production and chewing time both increase with the proportion of dry roughage. Ruminal acids are neutralized and diluted by an increased input of salivary buffers from prolonged chewing or rumination, even though a greater degree of mastication may reduce the size of grain particles entering the rumen and hence enhance its rate of fermentation. By replacing cereal grains with starch-extracted concentrates (such as distilling or brewing co-products and middlings), the amount of starch in the diet can also be decreased. According to Preston (1995), a limited maximum intake feeding plan can also be used to control total diet intake.

iii) Glycolysis

When free glucose is present, anaerobic microorganisms usually flourish. However, the high levels of free glucose in the rumen during acidosis suggest that glycolysis may be partially inhibited. Less than half of the glucose incubated with ruminal contents (1% wt/vol) vanished within 6 hours in our ruminal fluid incubation investigations and those of others (A. Z. Leedle, personal communication); this supports the idea that free glucose is not being catabolized efficiently for reasons that are currently unclear.

iv) Production and Use of Lactate and Volatile Fatty Acids

Rumen bacteria are frequently categorized as "lactate producers" or "lactate users." Whether lactate builds up depends on how these two groups are balanced. Bacterial strains' final products can vary based on the availability of substrate and culture conditions (Russell and Hino, 1985). While most lactate manufacturers are not sensitive to low pH, the majority of lactate-using microorganisms are. Pyruvate is transformed into lactate in anaerobic environments in order to provide the NAD needed for glycolysis. Lactate does not build up in the rumen at amounts higher than 5 mM under "normal" circumstances.

v) Osmolality of Rumen

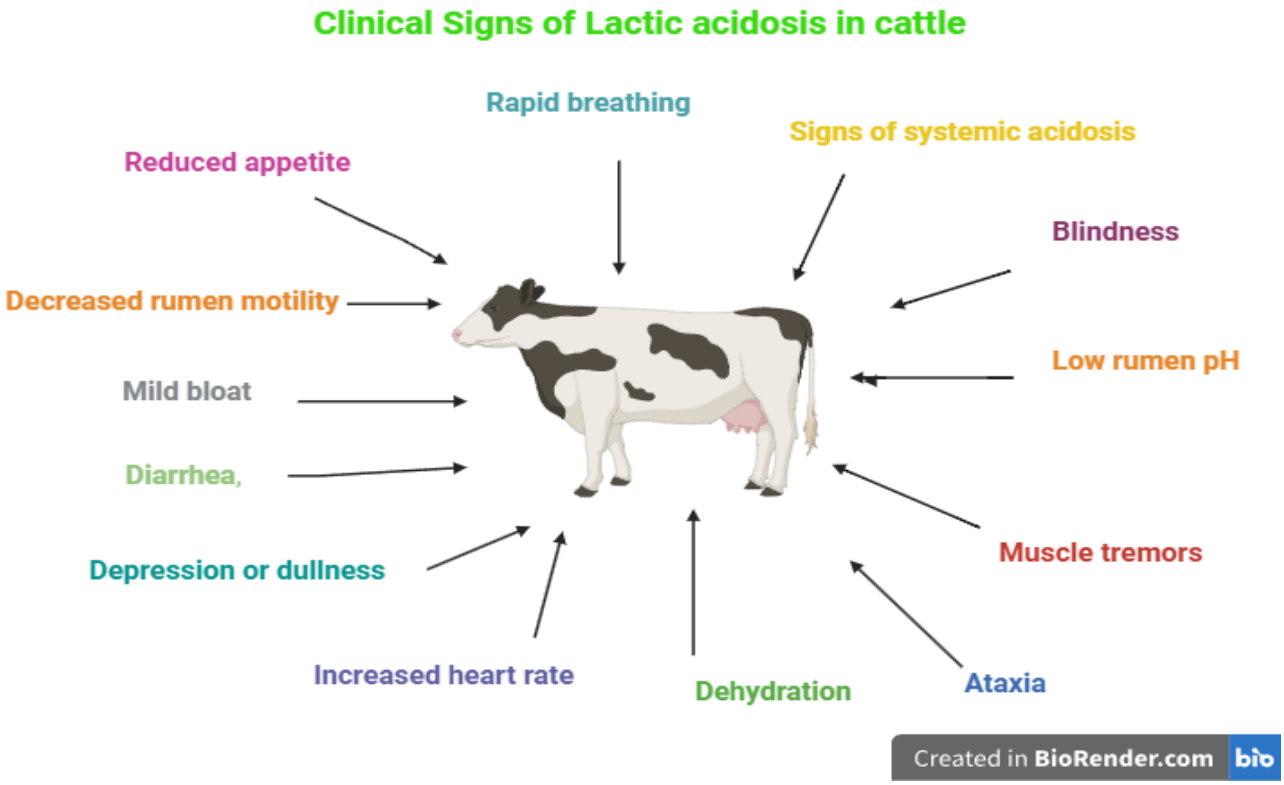
Depending on the relative amounts of dissolved elements, osmotic pressure either pushes or pulls water across membranes. According to Garza *et al.* (1989), ruminal osmolality typically varies from 240 to 265 mOsm/L with roughage diets and 280 to 300 mOsm/L with concentrate diets. The main solutes in ruminal fluid include minerals, VFA, lactate, and glucose. The osmotic pressure in blood, which typically ranges from 285 to 310 mOsm, is significantly influenced by dissolved protein. Ruminal osmolality has been measured as high as 515 mOsm under the acidotic conditions of engorgement experiments. Water from blood is quickly pulled inward through the rumen wall when ruminal osmolality is significantly higher than blood osmolality. Histological investigations by Eadie and Mann (1970) graphically demonstrate how rapid influx to neutralize osmotic pressure expands the ruminal papillae and can drag patches of the ruminal epithelium into the rumen by removing the internal surface layers of the rumen wall from the underlying layers.

vi) Acid Absorption

The rumen and intestinal epithelium passively absorb lactate and VFA. When concentrations are high, pH is low, and osmolality is normal, the rate of absorption is higher (Tabaru *et al.,* 1990). The percentage of

each organic acid in the non-dissociated (acid) state and the rate of absorption increase with decreasing pH. During absorption, glucose is partially transformed into D lactate and butyrate is partially digested as an energy source for the rumen wall. The overall lactate load for the liver may much exceed the lactate absorption from the rumen since lactate is also produced in and absorbed from the intestines (Godfrey *et al.,* 1992).

microorganisms, transfaunation rumen fluid from a healthy cow is sometimes administered. Under veterinary supervision, probiotics may also be used. To treat shock, metabolic acidosis, and dehydration, aggressive rehydration is essential. The right IV fluids and rates will be selected by a veterinarian. To adjust blood pH, veterinarians may need to inject systemic buffers (such as particular IV alkalinizing



Management & Treatment Principles (High-Level):

Stop consuming high-starch or highly fermentable feed (grain, finely-ground concentrates) right away. Veterinarians can select the right alkalinizing drugs and dosages to combat low pH through rumen alkalinization. draining the rumen. In extreme circumstances, a veterinarian may remove the fermenting diet and acid through rumen lavage or rumenotomy. To re-seed normal

drugs). Due to species-specific safety concerns, anti-inflammatory drugs can only be used under veterinary supervision.

Conclusion

New approaches to lowering the frequency of acute and chronic acidosis should result from ongoing research on grain processing, dietary cation-anion balance, narrow-spectrum antibiotics, bacteria that use lactate or glucose, salivary flow stimulants, and feeding management.

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