

# Subacute Ruminal Acidosis: The Role of Nutrition in the Etiology of Bovine Laminitis

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## Introduction

For many dairies, high production is required for the business to be profitable. The demand for production necessitates approaching and testing the limits of energy and fiber feeding. Consequently, subacute rumen acidosis is an insidious problem in some dairy herds. The feedlot industry is well aware of the significance of rumen acidosis in cattle and its negative effect on profits. However, the dairy industry has been slow to recognize acidosis as a common and significant disorder. A description of subacute acidosis and background information on rumen adaptation as it relates to subacute acidosis are provided so that the etiology of the disorder can be better understood. One of the sequelae to ruminal acidosis is chronic laminitis, which leads to lameness by causing decreased or defective horn growth, which then leads to sole ulcers, white-line lesions and deformed claws. While ruminal acidosis is associated with laminitis, it is important to realize that chronic laminitis in the bovine has multiple risk factors that may act synergistically.

## Subacute ruminal acidosis

*Definitions: Acute vs. Subacute Ruminal Acidosis.* The term ruminal acidosis refers to any situation in which the pH of the rumen contents is abnormally low. A problem in the discussion of ruminal acidosis is defining what is normal for the rumen. It is difficult to precisely define ruminal acidosis because of variation in ruminal pH and in animal response to changes in ruminal pH. The pH of ruminal contents typically exhibits a diurnal variation in response to consumption of meals (Fischer et al. 1994, Fulton et al. 1979, Le Ruyet et al. 1992). The normal range of ruminal pH is quite large. Ruminal pH can vary 1.0 pH units and still be considered normal. Unfortunately, it is difficult to quantify a change in production or health status due to changes in ruminal pH because of other confounding variables that cannot be controlled.

Given these caveats, approximate bounds between normal, subacute ruminal acidosis and acute ruminal acidosis have been proposed. Acute acidosis is an extreme condition with ruminal pH values  $\leq 5.0$  and subacute acidosis is less severe with pH values ranging between approximately 5.0 and 5.5 (Blood & Radostits 1989, Garrett 1996). It is difficult to delineate precisely where subacute becomes acute or where normal becomes subacute. While difficult to differentiate precisely in terms of pH, subacute and acute acidosis are different clinical entities. The two clinical syndromes share a similar etiology in the over-consumption of rapidly degradable carbohydrate; however, in subacute acidosis, the rapidly degradable carbohydrate supply is depleted before sufficient quantities of organic

acids are produced to cause serious, immediate damage to the animal. As a result, the two syndromes have different clinical presentations and different physiologic effects on the animal.

An animal suffering from acute acidosis is depressed, ataxic or recumbent, anorectic, dehydrated, has a fluid-distended, atonic rumen and an elevated heart rate. Rumen pH is  $\leq 5.0$ , protozoa are absent, and only the most acid tolerant bacteria (e.g. *Lactobacillus sp.*) remain. Serum lactic acid levels are elevated, pCO<sub>2</sub> is increased, and blood pH is decreased indicating uncompensated systemic acidosis as a result of the absorption of excessive amounts of organic acids from the rumen (Blood & Radostits 1989, Britton & Stock 1987, Counotte & Prins 1981, Elam 1976). These animals have a guarded to poor prognosis. Even with treatment, death may be the end result.

Subacute rumen acidosis can be described as a temporary imbalance between the rate of acid production, the rate of acid absorption and buffering capacity, which causes an accumulation of volatile fatty acids in the rumen, a decrease in rumen pH and changes in the microbial population distribution; however, the aberration is not severe enough or of sufficient duration to cause overt clinical signs in the animal. Subacute acidosis is subtle in its clinical presentation with the primary symptom being reduced feed intake (Britton & Stock 1987, Fulton et al. 1979, Krehbiel et al. 1995, Slyter 1976). Other symptoms include mild dehydration, decreased rumen motility, and pH of the rumen contents less than 5.5 (Blood & Radostits 1989). Detection of these symptoms depends on when the animal is examined during the course of the acidotic episode. Animals may be off feed and have ruminal pH values less than 5.5, yet, unlike acute acidosis, ruminal and blood lactate remain at normal levels (Tremere et al. 1968, Leedle et al. 1995). The short-term prognosis for the animal is good; however, long-term sequelae such as laminitis, liver abscesses, and pulmonary abscesses may follow at a later date (Nordlund et al. 1995).

## Etiology of ruminal acidosis

To maintain a normal rumen environment, acid production, absorption and buffering must occur at balanced, appropriate rates. The ruminal microbial milieu and nutrient characteristics of the diet control the rate of acid production, the ruminal mucosa is responsible for the rate of acid absorption, and the fiber form and content of the diet primarily controls the amount of buffer entering the rumen.

*Periparturient or Transition Acidosis.* To avoid ruminal acidosis, the microbial milieu of the rumen must be allowed to adapt to changes in type and quantity of nutrient

substrates entering the rumen. A sudden increase in energy substrate can disrupt the competitive balance between microbial species. In an energy unrestricted environment, *Strep. bovis* replicates at a very fast rate and produces lactate, which contributes to instability in the ruminal environment (Russell & Hino 1985). The sequence of changes occurring in bacterial and protozoal numbers during adaptation to grain has been described (Mackie et al. 1978). In general, numbers of protozoa, amylolytic bacteria and lactate-utilizing bacteria increase as the proportion of grain in the diet increases. The importance of microbial adaptation is well illustrated by the work of Huber (1973) and Allison et al. (1964) who showed that inoculating the rumen of a forage-fed sheep with the rumen contents of a grain-adapted sheep would prevent acidosis.

The absorptive capacity of the ruminal mucosa must match the VFA production potential of the diet to prevent accumulation of VFA in the rumen and a decline in ruminal pH. The surface of the rumen is covered by papillae, which serve to increase the absorptive surface area of the rumen mucosa. The rumen papillae vary in size according to diet. Increased intake of fermentable feeds stimulates papillae development and conversely, a decrease in intake of fermentable feed causes papillary regression. As the surface area and mass of the ruminal mucosa increases, the capacity to absorb VFA from the rumen increases. (Dirksen et al. 1985, Sutton et al. 1963, Perrier et al. 1994) This relationship is important in preventing an accumulation of VFA in the rumen. Animals switched from a low-energy diet to a diet high in readily fermentable feeds will be prone to an increase in the size of the VFA pool in the rumen until the ruminal mucosa adapts. During this period of adaptation, animals may experience periods of subacute ruminal acidosis due to an inability to absorb VFA rapidly. Current diet affects ruminal acid load by controlling the rate and quantity of acid production through bacterial substrate availability, and previous diet affects ruminal acid load by influencing the VFA absorptive capacity of the ruminal mucosa.

Periods of dietary change to higher energy diets are potential risk periods for ruminal acidosis because the microbial populations and rumen must undergo simultaneous adaptations to maintain the balance between acid production and absorption. Inadequate adaptation may lead to an accumulation of organic acids in the rumen and ruminal acidosis. In dairy herds, such dietary changes occur during the transition from the pre-calving diet to the lactating diet. Transition diets intermediate in energy/fiber between the dry and lactation diets are recommended to minimize the risk of ruminal acidosis in early lactation.

**Diet Formulation Acidosis.** The National Research Council (NRC, 1989) has estimated the nutritional requirements of lactating dairy cows and provided guidelines for nutrient content for ration formulation. Dairy nutritionists strive to meet or exceed NRC requirements for protein, energy, minerals and vitamins; however, the inverse relationship between energy and fiber makes it difficult to meet the fiber requirements. Additionally, fiber

is a difficult entity to quantify. No single value sufficiently represents the fiber content of a ration; therefore, fiber is quantified using a combination of acid detergent fiber (ADF), neutral detergent fiber (NDF), NDF from forage (FNDF), and effective NDF. The primary objective of maintaining a minimum fiber level in rations is to stimulate rumination, which provides copious volumes of bicarbonate-rich saliva to buffer acids generated in the rumen during fermentation. Increasing NDF increases eating time, ruminating time and ruminal pH (Beauchemin 1991, Beauchemin and Buchanan-Smith 1989). Feeds of small particle size do not stimulate rumination to the same extent as large particle size feeds, therefore, the effectiveness of a fiber source must be considered when evaluating the fiber levels of a ration. Small forage particle size decreases rumination time, which decreases flow of saliva to the rumen and results in a decrease in ruminal pH (Woodford & Murphy 1988, Grant et al. 1990). Rations for lactating cows formulated to contain insufficient fiber cause repeated bouts of acidosis *throughout lactation*. Herds fed low-fiber, high-energy rations tend to be severely affected by the sequelae to acidosis. In these cases, the combination of poor rumen buffering and high levels of acid production cause rather severe acidosis

### **Clinical signs of subacute ruminal acidosis**

The clinical signs of subacute acidosis are subtle and often temporally separated from the inciting event, which makes the diagnosis difficult. Subacute acidosis is a *herd problem*; therefore, the clinical signs of the disorder are related to the herd. Lameness, cyclical feed intake, high cull rate and unusual or vague illnesses are the typical signs of a herd suffering from subacute acidosis. The symptoms listed are not specific for acidosis, therefore, an investigation must attempt to rule out other possible etiologies of the observed symptoms. More detailed discussion of the clinical syndrome and diagnostic rationale are available (Nordlund 1994, Garrett, 1996) but the discussion in this paper will focus on the symptom of lameness, which is due to chronic laminitis.

### **The role of nutrition (acidosis) in laminitis**

Chronic laminitis is a common clinical sign in herds experiencing subacute ruminal acidosis. Ridges in the dorsal hoof wall, sole ulcers or abscesses, white-line lesions, sole hemorrhages and misshapen hooves are the common clinical signs of chronic laminitis (Boosman et al. 1991). The etiology of bovine laminitis is multi-factorial. Risk factors for laminitis include housing/environment, nutrition, management, disease (e.g. mastitis, metritis), breed/genetics, body weight, stress and behavior (Nocek 1997, Bergsten & Frank 1996). In the author's opinion, among this list, housing/environment, nutrition and breed/genetics are the three most important factors. Nutrition is an important component in the pathogenesis of laminitis; however, the physiologic details leading from ingestion of feed to the onset of the vascular changes in the claw that cause laminitis are not completely understood. To complicate matters, an interaction of two or more of the

aforementioned risk factors may be required to cause laminitis when no single error in management is extreme. This is a liberal interpretation of an experiment by Bergsten and Frank (1996) where a 2 X 2 experiment comparing high- vs. low-concentrate diet and concrete vs. rubber-padded stall indicated that the stall was the most important factor in the severity of sole hemorrhages, but the highest score for sole hemorrhages was in the high concentrate plus concrete stall treatment group.

Ruminal acidosis probably causes laminitis by causing the absorption or release of vasoactive substances such as histamine and endotoxin that cause alterations in the hemodynamics of the claw region (Boosman et al. 1991, Boosman, Nemeth & Gruys 1991, Hunt et al. 1990, Nocek 1997). Histamine and endotoxin are found in the rumen and histamine has been associated with liver abscesses, which are sequelae to ruminal acidosis. Inciting agents may cause paralysis of arteriovenous anastomoses in the digit leading to increased local blood pressure and vascular damage (Greenough 1996). In equine research, endotoxin administration resulted in vasoconstriction and arteriovenous shunting, which would lead to poor perfusion of the digit and laminitis (Hunt 1990). Boosman et al. (1991) administered endotoxin to cattle and created histopathologic lesions in the digit consistent with laminitis, but the animals did not show clinical signs of laminitis. However, Anderson and Jarlov (1990) were unable to reproduce an increase in ruminal endotoxin or measure endotoxin in systemic circulation following experimentally induced ruminal acidosis. Thrombosis, congestion, edema, arteriosclerosis and hemorrhages are common histopathologic features of laminitic bovine digits (Anderson & Bergman 1980, Boosman et al. 1991, Singh et al. 1992). All of these vascular features culminate in chronic poor perfusion of the keratogenic tissues of the digit, which leads to decreased horn production or production of inferior quality horn. The disruption in horn production sets the stage for white-line defects, sole ulcers and abnormal hoof morphology.

While the specific mechanism by which nutrition leads to laminitis is lacking, there is strong circumstantial evidence that nutrition is involved in the pathogenesis. High-energy, low-fiber diets have been linked to laminitis in dairy cows (Moser 1987, Livesey 1984). Supportive evidence also comes from experiments that demonstrated feeding high-energy, low-fiber diets or diets with rapidly degradable starch sources increased the incidence, severity and duration of lameness in cows (Kelly 1990, Manson 1988).

## Summary

Diets deficient in fiber or effective fiber or with excess rapidly fermentable carbohydrate may cause ruminal acidosis. Cow variables such as dietary adaptation will influence the effect of the diet on the rumen environment. Periods of dietary change to higher energy diets are potential risk periods for ruminal acidosis because the microbial populations and rumen mucosa must undergo

simultaneous adaptations to maintain the balance between acid production and absorption. Inadequate adaptation may lead to an accumulation of organic acids in the rumen and ruminal acidosis. Ruminal acidosis can cause damage to the rumen epithelium and lead to a variety of health consequences for the animal. Chronic laminitis is one of the maladies associated with ruminal acidosis. Chronic laminitis results in a high incidence of lameness by causing decreased or defective horn growth, which then leads to sole ulcers, white-line lesions and deformed claws. While ruminal acidosis is associated with laminitis, an investigation of a herd with a high incidence of laminitis must also consider other risk factors for laminitis such as housing, disease, stress and genetics.

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