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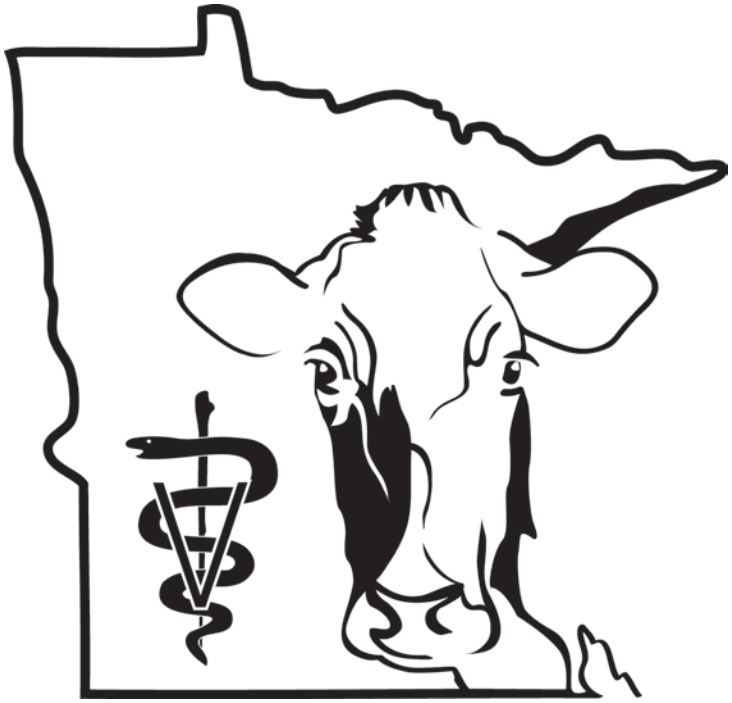

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# The Relationship between Nutrition and Management to Lameness in Dairy Cattle

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Lameness is an important disease of dairy cattle. It is the third most common reason for culling cows exceeded only by reproduction and mastitis as more common causes for removal. In addition to the costs associated with culling, less clearly defined economic losses are the costs associated with treatment, milk discarded because of antibiotic residues, adverse effects on dry matter intake, reduced milk yield, weight losses, and reduced reproductive performance. In addition, the high prevalence of lameness in some herds raises important animal welfare issues. In survey studies, the annual incidence of lameness in dairy cattle has ranged from 3 to 30% and claw disorders account for 75 to 90% of the diagnosed lameness in cattle. In a survey lameness in 427 cows and cohorts, the reproductive performance of lame cows was poorer than cohorts. The lame cows had a 6 day longer interval from calving to first service, a 14 day longer interval from calving to conception, and higher culling rate. The most common infectious cause of foot lamenesses are foot rot and hairy warts. The most common non-infectious causes of foot lamenesses are a consequence of chronic subclinical laminitis.

Chronic subclinical laminitis is the most important cause of non-infectious foot lamenesses in dairy cattle. Conditions associated with chronic subclinical laminitis include: 1) a softening of the sole with either a bloody or yellowish discoloration of the sole but without detectable lameness, 2) sole ulcers, 3) hemorrhage, widening, or separation of the white line with subsequent abscessation, 4) claw growth abnormalities resulting in increased growth rates of the anterior and abaxial hoof wall, and 5) development of false sole. The blood tinged discoloration of the sole may be mis-diagnosed as bruising. When the incidence of non-infectious foot lameness exceeds 10%, chronic subclinical laminitis should be suspected. In addition to laminitis, other health problems frequently associated with ruminal acidosis include intermittent diarrheas, poor appetites or cyclic feed intakes, high herd culling rates, cows with poor body condition in spite of consumption of high concentrate diets, cows that have either epistaxis or hemoptysis.

Hoof wall and sole are very specialized epidermis. As epidermis elsewhere on the body, the epidermis of the hoof has a limited number of means of responding to irritation. In humans, severe acute irritation of the epidermis and dermis may be manifest by the formation of blister. The analogous lesion in the hoof would be the hemorrhage and yellowish discoloration seen in the sole following acute clinical to subclinical laminitis with the subsequent formation of a false sole. In humans, if the irritation of the dermis-epidermis is of a milder and more chronic nature, the dermis responds with the formation of a callous. The equivalent response in the hoof is a increased production of hoof wall. Probably the more correct term for the condition characterized by increased proliferation of the hoof wall is coriosis rather than laminitis.

Sole ulcers or pododermatitis circumscripta are the most common lesion to develop secondary to chronic subclinical laminitis. To appreciate the pathogenesis of sole ulcers, one needs to understand weight bearing within the normal hoof. The third phalanx is firmly attached to the hoof wall by a laminar corium. The greatest contact area between the pedal bone and hoof wall is at the anterior surface and along the abaxial wall of the foot and decreases toward the heel area. This allows the papillar corium under the third phalanx to function as a cushion. Within a normal hoof that is well trimmed, there is a uniform distribution of weight from the third phalanx to the solar surface of the hoof. Following laminitis, there greater proliferation of hoof material from the papillar corium producing the hoof wall, especially at the dorsal hoof wall and along the abaxial wall of the hoof. Weight distributions from the pedal bone changes from a uniform distribution across the sole to a greater proportion of the weight transferred to caudal and axial surface of the sole. This change is weight distribution results in an ischemic necrosis to the papillary corium at the site of the sole ulcer.

Several factors predisposed cows to chronic subclinical laminitis. These factors include nutrition, confirmation, management practices, severe metabolic disturbances, heat stress, and genetics. Nutritional practices are recognized as the most important factor responsible for herd problems with non-infectious foot lameness. Several theories have been proposed to explain the relationship between ruminal acidosis and chronic subclinical laminitis. Irrespective of the mechanism by which changes in rumen pH cause laminitis, the relationship between nutrition and chronic subclinical laminitis frequently goes unrecognized because of the time lag between the nutritional insult and the subsequent lameness. Any nutritional practice which results in a decrease of rumen pH below 5.5 can result in chronic subclinical laminitis. Under normal circumstances, the primary products of rumen fermentation are acetate, propionate, and butyrate. Two factors can facilitate an increase in lactic acid production in the rumen by Streptococcus bovis. Either an increase in the concentration of substrate in the form of either starches or sugars or a decrease in rumen pH below 5.5 will result in a shift in the products of fermentation in the rumen from normal volatile fatty acids to lactic acid. Much of the adaptation of the rumen to high concentrate rations involves an increase in populations of lactilytic bacteria which convert lactic acid to propionic acid.

The role of feeding high concentrate diets to dairy cows as a factor predisposing cows to lameness has been well established in several experiments. Mason and Leaver, 1988, one group of cows a low concentrate ration and one group a high concentrate ration from weeks 3 to 22 of lactation (Table 1). The forage portion of the ration consisted of a grass silage. The concentrate mix was 17% crude protein and consisted of 24% barley and 20% wheat, both of which have rapidly starches. Ten cases of clinical lameness were observed in the group fed the low concentrate rations while 37 cases were observed in the group fed the high concentrate rations. The primary lameness observed in both groups was sole ulcers. Sole ulcers represented 7 of 10 clinical lameness cases in the low concentrate group and 28 of 37 cases in the high concentrate group. Clearly, feeding the higher concentrate ration resulted in a greater incidence of clinical lameness of which sole ulcers were the primary lameness.

Table 1.

| Effect of Forage:Concentrate Ratio on Lameness | | |
|---|---|---|
| Parameters | Low Concentrate | High Concentrate |
| Number of Cows | 24 | 24 |
| Dry Matter Intake, lb/d | 31.2 | 35.2 |
| Concentrate Intake, lb,d | 13.6 | 20.5 |
| Forage:Concentrate | 56:44 | 42:58 |
| Milk, lb/d | 45.1 | 52.1 |
| Number of Clinical Lamenesses | 10 | 37 |
| Number of Sole Ulcers | 7 | 28 |

In a subsequent trial, Mason and Leaver, 1989, evaluated the interaction of trimming hooves of cows fed either a low or high concentrate ration (Table 2). As in the previous trial, feeding a higher concentrate ration resulted in a greater incidence of clinical lameness and sole ulcers. However, whether cows were fed high or low concentrate rations, the trimming of hooves lowered the incidence of lameness and sole ulcers.

Table 2.

| Effect of Forage:Concentrate Ratio and Hoof Trimming on Lameness between Weeks 3 and 26 of Lactation | | | | |
|---|---|---|---|---|
| Parameter | Lo Conc Trim | Lo Conc Untrim | Hi Conc Trim | Hi Conc Untrim |
| Number of Cows | 12 | 12 | 12 | 12 |
| Dry Matter Intake, lb/d | 33.7 | 33.9 | 31.7 | 31.9 |
| Forage:Concentrate | 60:40 | 60:40 | 40:60 | 40:60 |
| Milk, lb/d | 45.8 | 45.3 | 46.6 | 49.5 |
| No. Clinical Lamemesses | 8 | 20 | 14 | 30 |
| No. of Sole Ulcers | 1 | 13 | 7 | 11 |

## DIAGNOSIS OF RUMINAL ACIDOSIS

Ruminal acidosis can be diagnosed by collecting samples of rumen fluid by rumenocentesis and measuring the pH of the fluid. Norlund has outlined a systematized approach to a herd-based diagnosis of ruminal acidosis. Samples of rumen fluid should be collected from a minimum of 6 early lactation cows and 6 mid-lactation cows. The samples of rumen fluid should be collected 2 to 4 hours after concentrates have been fed in component fed diets or 4 to 8 hours after total mixed rations have been fed. Nordlund has developed a system for classifying the results of rumenocentesis (Table 3). If 30% or more of the cows in either groups have ruminal pH's of less than 5.5, the group has a problem of ruminal acidosis. If more than 30% of the early lactation cows have ruminal acidosis, than there is a problem with adaptation of the cows to lactating cow rations. If more than 30% of the mid-lactation cows have ruminal acidosis, than there is a ration formulation or feeding problem.

Table 3.

| Rumen pH | Interpretation |
|---|---|
| < = 5.5 | abnormal |
| 5.6 to 5.8 | marginal |
| > = 5.9 | normal |

The two primary categories of nutritional problems that are responsible for ruminal acidosis are nutritional management practices which result in poor adaptation of the rumen to ration changes which occur in early lactation and rations which predispose the cow to ruminal acidosis. The poor ruminal adaptation of the post-calving cow is usually the result of failing to appropriately transition dry cows from dry cow rations to lactating cow rations. This adaptation failure has two consequences: 1) The microbial population of the rumen is poorly adapted to increasing concentration of grain in the diet resulting the production of lactic acid and a drop in rumen pH. 2) The rumen papillae are not stimulated to develop and increase the absorptive area of the rumen resulting in slower absorption of acids from the rumen and greater drops in rumen pH following feeding. Rations which cause ruminal acidosis can result from either errors in formulation or errors in feeding of rations.

The prevention of ruminal acidosis in early lactation cows is dependent upon developing and feeding a sound transition rations at least 2 to 3 weeks prior to calving. The transition ration allows for the adaptation of the ruminal microbes to the fermentation of concentrates without the accumulation of lactic acid and stimulates the development of ruminal papillae which increases the surface area of the rumen allowing for faster absorption of acids produced via fermentation. National Research Council (NRC;1989) estimates dry matter intake (DMI) of dry cows to be 1.8 to 2.0% of body weight. However, several trails have shown that DMI drops by as much as 30% the last week prior to calving. This prepartum decrease in DMI requires that a transition ration be developed with higher nutrient densities be fed to close-up dry cows to more closely meet their nutrient requirements.

The prevention of ruminal acidosis from the ration begans by formulating rations that meet or exceed the minimal NRC guidelines for acid detergent fiber of 21% of dry matter and neutral detergent fiber of 28% of dry matter. In addition, rations should contain a minimum of 18 to 21% of the NDF coming from forage. However, even when minimal fiber guidelines are met, other factors can contribute ruminal acidosis. Saliva is an important source of buffer for the rumen. The buffering capacity in one day's production of saliva is equivalent to 7 pounds of sodium bicarbonate. Any factor which reduces the amount of saliva produced will increase the risk of ruminal acidosis. If forage particles are too small, the forage losses effectiveness in stimulating rumination, less saliva is added to the feed, and ruminal acidosis may develop. Silages are frequently finely chopped to enhance packing and reduce the amount of trapped oxygen. However, finely chopped silages have a greater surface area than coarsely chopped silages which increases the rate of fermentation. In addition, silages are moist and require the addition of less saliva for ingestion than dry hay which further exacerbates the risk of ruminal acidosis. By chopping silages more coarsely, the silages will stimulate greater saliva production. Silages should contain at least 25% of the particles of greater than 1.5 inches in length. Changes in silage moisture can also contribute to the risk of ruminal acidosis. If the moisture content of a silage increases without awareness of the nutritionist or farmer, the amount of forage dry matter in the ration will decrease relative to the concentrate.

Dry matter content errors in total mixed rations can precipitate ruminal acidosis problems when the moisture content of silages increases without concurrent adjustment for the changes in the dry matter contribution to the TMR from the silage. If a ration meets minimal guidelines for fiber content with speicified quaintity of silage based on a given dry matter content, than a increase in silage moisture will reduce the amount of silage dry matter amd fiber contribution to the ration.

The nonstructural carbohydrate (NSC) represent the more rapidly fermenting starches and sugars in the ration. The level of nonstructural carbohydrate (NSC) in the ration should not exceed 40%. This recommendation is tempered by the source of the NSC. Rumen fermentability can be reduced by substituting corn for barley or wheat. Moist grains ferment more rapidly than dry grain.

Supplemental ruminal buffers such as sodium bicarbonate or sesquicarbonate fed in early lactation can reduce the risk of ruminal acidosis. The recommended feeding rate of sodium bicarbonate is .75 to 1% of the total ration dry matter.

**Heat Stress and Ruminal Acidosis**

Heat stress can lead to ruminal acidosis and subclinical laminitis with ration that function without development of ruminal acidosis in a non-heat stressed environment. Cows that are heat stressed pant as a mechanism to dissipate heat. As a consequence of the panting, the cows also drive off carbon dioxide from the blood and develop a respiratory alkalosis. The primary blood buffering system is dependent upon a constant ration between carbon dioxide and bicarbonate. Since the panting has driven off carbon dioxide, the cow attempts to correct the respiratory alkalosis by excreting more bicarbonate through the kidney. This attempts to return the ratio between carbon

dioxide and bicarbonate to normal by lowering the amount of bicarbonate in the blood. When the heat stress is alleviated enough that the cows quits panting, the level of carbon dioxide in the blood increases toward normal levels. However, because the cows had lowered the levels of bicarbonate in the blood, there is a relative deficiency of bicarbonate and the cows now develops a metabolic acidosis. There now a deficiency of bicarbonate to be incorporated in the saliva and the cow is incapable of adequately buffering the rumen. Also, when cows are heat stressed dry matter intake is depressed and what feed the cows does consume takes longer to transit the rumen. This results in feed spending more time in the rumen and producing more acid per unit of dry matter through the increased fermentation time while in the rumen. More acid is produced per pound of dry matter consumed while in the rumen and less bicarbonate is available in the saliva to buffering the acid produce through rumen fermentation. As a consequence, cows may develop ruminal acidosis during periods of heat stress. Frequently the recommendation is made to increase the energy density of the ration during periods of heat stress by increasing the proportion of concentrate in the ration. This can be a dangerous strategy for a cow that is already at increased risk of ruminal acidosis. The first strategy should be to increase the amount of rumen buffer in the ration of the heat stressed cow over that of non-heat stressed cows to compensate for the physiological factors that inducing ruminal acidosis. Rumen buffers for heat stressed cows should be increased by 50% over periods of non-heat stress.

Ruminal acidosis can develop in spite of the fact the ration has been formulated to meet minimal NRC fiber guidelines. When ruminal acidosis is suspected, the condition can be diagnosed through the technique of collecting samples of rumen fluid and determination of pH. Through the practice of monitoring rumen pH and prevention of ruminal acidosis, chronic subclinical laminitis and the subsequent lameness can be prevented.

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