**Bovine Laminitis**

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**Introduction**

Laminitis, according to Merck Veterinary Manual, is a pathophysiologic disturbance of the microstructure of the dermis or corium of the claw and may in fact involve more than just the laminar region of the foot.  
Stokka et al 2001 described laminitis as a lameness condition commonly observed in dairy and beef cattle enterprises where aggressive high concentrate rations are used. They further state that it may be the number one cause of foot problems particularly in the subclinical form. Subclinical laminitis is considered to be of economic importance due to predisposing animals to sole ulcers, white line disease and toe ulcers.  
This article briefly reviews some points regarding laminitis and will hopefully draw attention to this erosive condition

**Laminitis**

Laminitis is defined by Stokka et al to be a disease that causes degeneration, necrosis and inflammation of the dermal laminae and epidermal lamellae of the hoof. SEE FIGURE 1  
The bovine hoof supports the animal’s weight, through a laminar suspension apparatus that maintains the structural integrity between the pedal bone and the hoof wall. Nutritional, metabolic and infectious diseases may damage the suspending laminae by disruption of the microcirculation via inflammatory mediators, such as histamine and endotoxins. The disruption of the microcirculation is postulated to activate arteriovenous shunts, resulting in localized ischaemia of the corium. Nutrient and oxygen deprivation of the laminae and the consequent oedema damages the integrity of the laminae suspension mechanism resulting in separation of the hoof wall from the pedal bone. In severe cases the pedal bone may rotate ventrally into the soft tissues of the sole and visible discoloration and bruising may occur.
Pathophysiology

Identification of the agents that play a part in laminitis is ongoing, however some of the current pathogenesis factors reported in Merck Veterinary manual are as follows: The basement membrane of the corium of the claw contain epidermal growth factor (EGF) receptors and EGF is liberated in large quantities from the GI tract when it is damaged (eg, rumenitis) and could be involved in the pathogenesis of laminitis. Inhibition of differentiation of keratinocytes of the hoof matrix is a dominant morphologic feature in the early stages of laminitis and it has been shown that EGF can inhibit this differentiation of keratinocytes in vitro. This suggests that laminitic histopathology results from an inadequate regulation of gelatinase activity, resulting in selective degradation of basement membrane components and laminitis due to failure of the basement membrane-epidermis attachment.

More recent investigations have studied the role of endotoxin activity in the pathophysiology of laminitis. It is not known precisely which endotoxins are involved in laminitis, however reports implicate the incidence of liver abscesses as a risk factor. Fusobacterium necrophorum is regularly isolated from liver abscesses and, with its several biotypes, could provide the suspected endotoxins. Cytokines and prostaglandins may play a part, and certainly hypoxia has a role in accelerating the pathology. The role of growth hormones and peripartal hormones such as relaxin could also have a complicating role.

Regardless of the initial cause there are two different types of tissues associated with laminitis: the papillary or solear dermis and the collagen fibers of the laminae. Two distinctly different pathologies affecting firstly the disruption of normal keratogenesis, and secondly, abnormal Matrix metalloproteinases (MMP) activity, results in sinkage and/or rotation of the digit. MMPs and matrixins are a family of secreted and membrane-bound zinc-dependent endopeptidases that have the combined capacity to degrade all the components of the extracellular matrix. The pathophysiologic process associated with compromised horn production starts when vasoactive toxins or other biologic agents reach the corium. Arteriovenous shunts may be paralyzed, pressure inside the claw rises, and the walls of the vessels are damaged. Blood or blood fluids escape and soak into the claw horn, staining it either pink or yellow. Hemorrhagic staining of the horn tubules of the sole give a “brush mark” appearance. Mural thrombi form, reducing blood flow and causing oxygen deprivation and an insufficient nutrient supply to the keratin-producing cells. Thrombus formation is a characteristic feature of laminitis and the resulting horn is soft and prone to damage, infection, and scar formation.

The second pathophysiologic process involves MMP release and stretching of the collagen fibers of the suspensory apparatus of the digit. This is the system that transfers weight-bearing from the pedal bone to the inside wall of the claw. As the collagen fibers stretch, the pedal bone is displaced. Occasionally, the pedal bone will rotate and the apex of the bone will prolapse through the apex of the sole. Perhaps more frequently, the whole bone will “sink,” causing the space between the flexor process and the sole of the claw to narrow and increasing the possibility of a sole ulcer developing. Frequently, young animals appear to recover from laminitis. This may be because new blood vessels develop to form collateral circulation and take over the function of those that have been
damaged. Nevertheless, each time an animal has a bout of laminitis, more scar tissue is formed and the animal is less able to recover from the next insult.

**Clinical Findings**

Merck Veterinary Manual states that laminitis can be acute, subacute, chronic, or subclinical, depending on the variables involved.

Merck states that acute laminitis is relatively uncommon while subacute laminitis may be seen in animals being fed carbohydrate rich diets. Chronic laminitis associated with a ridged and slipper foot appearance and is the result of a number of laminitic episodes. Stokka et al mention that in feedlot situations acute laminitis may affect up to 50% of animals in a pen.

In groups of cattle there may be varying degrees of response to the same set of causal factors varying from acute, subacute to sub clinical cases showing no symptoms.

Merck veterinary manual states that typically, in the most acute cases, there may be fever and an increased respiratory rate. The animal will be reluctant to walk and will stand abnormally with the feet drawn under the body. Frequently, the superficial veins of the hindlimbs may be turgid, the claws may be warm to the touch, and a pronounced digital pulse will be perceptible. Pain may be detected in the claws with the use of hoof testers. Some animals appear to walk in a deliberate, careful manner.

Erythema and edema of the skin above the coronary band and around the dewclaws in freshly calved cows may indicate a transitory laminitis-like insult and that the cows are being introduced to concentrate too rapidly.

Subclinical laminitis is typically undetected but animals may show an unnatural gait when walking or running. Hemorrhages in the sole and/or white line are consistent findings and concave and ridged dorsal walls of the hoof are also frequently observed. the Cow Comfort Index may be used as an indicator of the presence of this condition and if the appearance of sole and toe ulcers, white line disease, and double sole is >10% in a herd, it should be assumed that subclinical laminitis exists.

The Cow Comfort Index (CCI) is measured 1 hr before milking and calculated as the proportion of animals standing. If the CCI is >20%, risk factors affecting cow comfort should be reviewed. These include stall size, adequacy of bedding and bunk space, placement of water sources, and alley widths sufficient to avoid congestion or queuing.

Chronic laminitis is generally identified by the posture of affected animals and visual changes in the hoof. Varying degrees of gait change may be seen and animals may be reluctant to stand. When standing animals may carry more weight on their heels and hind legs may be brought forward to relieve the weight on the front feet. The normal dorsal angle of the front claw is 50° and the hind claw is 45° and in cases of chronic laminitis these angles may reduce as the hoof wall grows forward and appear flattened and ridged. PICTURE

Laminitis in breeding stock is serious because the tissues of the feet are permanently damaged, however, long term damage is of no importance to animals that will be slaughtered in the short term. However, the presence of pain affects weight gain.

In a study of undifferentiated lameness in feedlot animals Kruse et al evaluated feedlot health data sets for more than 30000 animals over a number of years and concluded that weight gain as
a result of undifferentiated lameness lost between 0.018 to 0.090 kg per day. They used undifferentiated lameness as the criteria due to the common misclassification of the reason for lameness. In dairy cows early culling due to hoof and claw disease is a likely significant loss.

**Aetiology and risk factors**

The classic theory for the cause of laminitis in cattle suggests that high levels of carbohydrate, either from a grain based ration or lush pasture, in the rumen invoke an increase of *Streptococcus bovis* and *Lactobacillus spp*, which induce a state of acidosis in the rumen. This causes gram-negative organisms to die and release vasoactive endotoxins.

Endotoxins are structural parts of the bacterial cell wall and in general comprise 3 major parts: O specific (oligosaccharide) side chain, the core polysaccharides and lipid A.

Beauchemin and McAllister described several theories that have been proposed to explain the link between acidosis and laminitis. They report that according to one theory, a reduction in systemic pH during acidosis activates a vasoactive mechanism that increases total blood flow to the hoof. Alternatively grain feeding increases the formation of histamine in the rumen which may be absorbed through rumen epithelium damaged during acidosis. Histamine is an inflammatory agent and vasoactive substance, and as such, may increase blood pressure and damage blood vessel walls causing inflammation and hemorrhaging within the hoof. However recent studies have produced compelling evidence for a different link between acidosis and laminitis based on the effects of bacterial toxins rather than vasoactive substances. They suggested that acute acidosis and repeated bouts of sub-acute acidosis damage the surface of the rumen wall and possibly the intestine thus allowing bacteria and bacterial toxins to enter the portal circulation, causing liver abscesses and an inflammatory responses.

Andersen, in a 2003 review, states that several authors have suggested that endotoxins play a significant role for the development of diseases such as laminitis, abomasal displacement, sudden death syndrome of feed-lot steers etc. In the review it is concluded that acidosis may increase the translocation of endotoxin from the gastro-intestinal contents to the systemic circulation. Certain animals may consequently suffer from a generalized endotoxin induced inflammatory response, which includes decreased motility of the fore stomachs, leukopenia, hypocalcemia and other imbalances. The clearance capacity of the liver plays important roles in the defense of endotoxin mediated diseases and in certain conditions animals may become tolerant to endotoxin levels. Tolerance is traditionally divided into 'early' and 'late' tolerance and occurs if an animal is exposed to endotoxin in sub lethal doses, either repeatedly or continuously, for a period of time. Endotoxin tolerance is a transient stage of hypo responsiveness during which the biological responses to endotoxin are diminished or absent. After tolerance is induced, endotoxin administered in even lethal doses does not elicit detrimental biological responses.

Andersen states that the nature of tolerance is not yet fully understood and several processes are involved including:

- Induction of a self-limiting inflammatory process
- A raised immune function and
- Increased production of mediators.
The early phase of endotoxin tolerance occurs within a few hours of the challenge, is transient and nonspecific regarding the type of endotoxin, is not associated with the occurrence of anti-endotoxin antibodies and cannot be transferred with plasma. The mechanisms of the early phase are complex, involving production of acute phase proteins which bind endotoxin, induction of endotoxin receptor blockage, and alterations of the macrophage activity. The priming endotoxin stimulus may lead to a production of immature monocytes, with only few active receptors available. The endotoxin challenge may also induce the production of anti-inflammatory serum factors, such as IL-10, type I and II soluble TNF receptors or cortisol, which down regulate the endotoxin response. The late phase occurs after days of repeated endotoxin challenge, lasts for longer and is related both to the development of antibodies against the different endotoxin elements. Antibodies against lipid A have a greater cross protective effect than antibodies directed against the more variable O-chain.

Rissoa et al reported that the bovine digital vasculature contractility has been implicated in the development of laminitis and studied the effect of hypoxia and re-oxygenation on the contractility of isolated peripheral bovine digital veins (BDVs). More research will be necessary before this can be fully implicated or understood.

Bell and Weary have stated that the link between laminitis and nutrition have long been recognized but they reviewed newer work identifying environmental and management factors that may also contribute to the development of laminitis. They suggested that factors affecting how long cows spend on their feet (standing or walking), and the quality of surface they are standing upon, are also associated with laminitis, however little work has been done in this regard to substantiate these views.

Burtt-Davy described feeding trials that confirmed the effect of laminitis due to Crotalaria Burkeana in as early as 1906. Botha and Penrith (2008) mention that severe laminitis may be caused by certain species of Crotalaria, primarily Crotalaria burkeana and Crotalaria barkae, while Crotalaria juncea has been associated with laminitis in cattle. The pathophysiology for this plant poisoning is not clear but like Senecio, at least some members of the genus Crotalaria contain pyrrolizidine alkaloids. Crotalaria spartioides has occasionally been associated with hepatotoxicity in cattle and dosing cattle with Crotalaria spartioides and Crotalaria dura has resulted in cirrhosis of the liver.

Treatment and control

Acute laminitis should be treated as an emergency using anti-inflammatory agents to reduce pain, re-establish normal circulation in the hoof and removal or correcting the cause. Stokka et al describe the use of NSAID’s and cortisone while other treatments may include antihistamines and anti-prostaglandins. They suggest that recovery rate is limited and severe cases require culling for humane reasons.

Merck Veterinary Manual suggest that if the cause is obvious, such as overeating grain, this should be corrected. Keeping the animal moving and the claws cool are reported to be helpful. Antihistamines may be useful if given within the first 48 hr after a known insult. Anti-
inflammatory drugs may be useful if given before the onset of acute signs. However, caution should be exercised in using corticosteroids later than 24 hr after signs appear.

Treatment for subclinical laminitis is impractical because diagnosis in an individual animal is not possible at the time of the causative insult(s). Controlling subclinical laminitis in a herd of high-production, intensively managed dairy cows requires a systematic management approach.

The Alberta Health Management: Lameness in Feedlot Cattle describes many points in risk factors causing or associated with laminitis. The general principles may apply to intensive dairy production as well as feedlots and are summarized below for the convenience of the reader.  

**Energy** - It is probable that animals between 8 to 12 months of age are more susceptible to high levels of energy intake than are animals from 12 to 16 months of age. High energy diets should be considered risk factors and rations formulated carefully. Highly digestible carbohydrate may also be a problem if it is introduced suddenly into the feed of animals and different grains have different starch digestibility. Finely ground maize, high moisture maize or highly digestible grain e.g. barley are potential risk factors. Gradual increments in high digestible components and more frequent smaller feed deliveries are recommended.

It has been postulated that compromising the foot health of heifers reduces the animal's lifetime productivity. Heifers calving for the first time prior to 28 months of age have a much higher incidence of lameness than those calving when they are older. Accelerating weight gains in order to meet mating criteria may be detrimental. It is suggested that weight gains exceeding 750 g/day are contraindicated before a heifer calves

**Forage** – The risk of laminitis increases as the percentage of forage in the ration decreases. The acid detergent fibre (ADF) component of the forage acts as a buffering agent which counteracts the acidity generated in the rumen by highly digestible carbohydrates. Usually hay and straw have reasonable buffering qualities but silages harvested during fast-growing stages may be deficient in buffering capabilities.

**Protein** - Generally protein is not reported to play a major role in the onset of laminitis. However, when crude protein (CP) levels exceed 16% in the ration, the excess may be converted to amino acids in the rumen and then metabolized as energy by the liver. CP in excess of 21% may be encountered in fast growing forage crops. When this is the case, the condition of the animals should be monitored carefully as conditions analogous to ‘grass founder’ in horses can occur.

**Particle Size** - Small particle size reduces the effectiveness of the fibre in the ration. The theoretical length of cut (TLC) should provide at least 25% of the particles greater than 5 cm long.

**Buffers** - The addition of buffers at 0.75% of the total dry matter may be useful with some forage. Alfalfa is generally more forgiving with regard to ruminal acidosis. The pH of alfalfa silage may be 4.5-5.0 as compared to corn or grass silage, which may be 3.5-4.0. When offered at levels higher than 1.0%, buffers tend to affect the appetite.

**Nitrate** - High levels of nitrate may be found in drinking water or in silage that has been harvested from fast-growing crops on heavily manured or fertilized land. Nitrate is converted to nitrite in the rumen and although nitrite toxicity is uncommon, it may occur occasionally and can exacerbate the other factors that may contribute to laminitis.

**Acclimatization** - It probably takes 28 days for the ruminal environment to adapt to a high concentrate ration and ad lib feeding within this period should be considered as a potential risk
factor. During the acclimatization period, it is prudent to increase the TDN content of the feed gradually.

**Discussion**

It seems that there is compelling evidence for the involvement of endotoxins in the pathogenesis of laminitis when considering the signs of endotoxicosis compared with clinical subacute and acute laminitis. Andersen states that clinical signs of endotoxicosis, in more severe cases, may include urination, defecation and salivation. Pulse and respiration rate frequencies increased following experimentally induced endotoxicosis. This was interpreted as an early vasoconstrictory response, which eventually results in cold ears and skin. The pulmonary responses in the cow seem to be quite marked in comparison to other species. Cattle have more smooth muscles in the pulmonary tree, compared to other species. Smooth muscle cells produce large amounts of prostanoid mediators when stimulated by endotoxin. The initial respiratory distress is probably caused by a thromboxane induced constriction of pulmonary arterioles. From other studies it is known that such episodes are associated with pulmonary hypertension. After 5-10 minutes the respiratory distress ceased, although the respiration rate in some instances may remain elevated for several hours, due to the formation of a pulmonary edema. Also low doses of endotoxin which do not result in shock are able to induce hyperventilation, possibly induced by vasoconstriction. Ruminal contractions decrease in number and strength and reticulo-ruminal stasis develop, often within 15-30 minutes. Reticulo-ruminal stasis is of particular interest in relation to the pathogenesis of production diseases associated with gastrointestinal atonia, such as displaced abomasum and 'off feed'.

It is a very old observation that the body temperature rises after administration of endotoxin. Significantly elevated rectal temperatures is not always observed in the bovine. It seems to be depending on the dose of endotoxin administered. Very high and very low doses do not elicit a fever response. This might be controversial, because traditionally fever has been a prerequisite for the clinical diagnosis of endotoxicosis. If fever can be absent from the clinical picture of endotoxicosis, the category of feeding-induced disorders is no longer excluded from the group of possible endotoxin related disorders.

The involvement of the liver and possible compromise to certain functions is supported by the plant type poisonings associated with laminitis when such plants are also often associated with liver damage.
The use of oligofructose in the induction of laminitis by Thoefner et al raises the question of the potential of different starch sources in the condition and the work from Bichalo et al in focusing attention on the digital cushion thickness and body condition score further complicates the concepts of laminitis.

**Conclusion**

The veterinary practitioner involved with herd work where lameness is a factor needs to review nutritional, management and other factors in advising their clients. In addition animal welfare
issues are being raised in this context and also requires careful strategies in minimizing such performance limiting and painful conditions.

Figure 1
References

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