

Causes, Risk Factors, and Prevention of Laminitis and Related Claw Lesions

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Introduction

Laminitis is an inflammation of the laminar corium of the hoof wall. In general, the term laminitis is used to describe a systemic disease affecting not only the hooves, but also the general condition of the animal. Researchers believe the inflammation is primarily associated with a dysfunction of the digital vasculature system that results in hypoxia and malnutrition of the sensitive laminar structure in the hoof wall. The etiology of the circulatory disturbance is not fully understood and there are some possible explanations often related to nutrition, which I will discuss further. Due to mechanical stretching of the attachment between the inner and outer laminar structures of the hoof wall, which has been affected by the inflammation (i.e. laminitis), the claw bone can rotate and or sink inside the hoof. Depending on the severity of the laminitis, the mobility of the claw bone inside the capsule and the counter pressure on the sole from hard floors, the sole corium can be contused and secondary lesions of the sole area can develop (*Ossent & Lisher 1998*).

The pathological alterations inside the rigid hoof capsule cause considerable pain and, therefore, results in lameness, which is an animal welfare issue. A United Kingdom survey (*Clarkson et al. 1996*) revealed a yearly lameness incidence of 55%, of which sole ulcers (40%) and white line lesions (29%) were the most prevalent lesions (*Murray et al. 1996*). Such laminitis-related lesions have a greater

economic effect than infectious diseases, for example, (*Esslemont & Spincer 1993*), due to severe influence on reproduction and consequently a higher risk for culling (*Sprecher et al. 1997*). Other common lesions associated with laminitis are: hemorrhages, fissures and abscesses of the white line, sole hemorrhages and double soles, toe ulcers and toe necrosis, and under-running of the heels. Thus, laminitis has a great economic impact on dairy operations, and all over the world dairy producers are moving towards housing cows in intensive, confined conditions. As we understand the causes of laminitis and identify the risk factors better, it is possible to prevent and reduce the effects of the laminitis syndrome.

History

Laminitis has been described in many species, but is most common in equine and bovine. Although bovine laminitis is most common in dairy cows, it has been reported in all ages and sexes.

The Greeks (Aristotle) associated equine laminitis with indigestion. The Greek name of the disease, Kritiatis, relates to overfeeding of barley. Xenophon noticed the characteristic symptoms, the hooves started to bleed and the horse became recumbent, which were due to pain in the hooves. These are the same symptoms observed in cattle today. During the last centuries, people have also considered that trauma significantly contributes to the disease

development. Later, toxic or chemical agents through the feedstuff or metastatic spread from infections have also been proposed in the etiology.

Bovine laminitis has a shorter history in the literature than equine. The Swedish Veterinary Journal in 1896 reported, "Inflammation of the claw corium is the most common cause of lameness and the often misshapen claw can be secondarily affected by a purulent process and/or the claw capsule could fall off. The affected animal is useless for work after it has been affected". Rusterholtz disease, i.e. sole ulceration, was described in the 1920s (*Rusterholtz* 1920), but was not associated with laminitis at that time. The first Ph.D. thesis on bovine laminitis was published in the 1960s (*Nilsson* 1963). It included a thorough description of the symptoms and the pathology, which are still relevant today. Since the same clinical symptoms are also observed in equine laminitis, it is natural that research on the etiology of equine laminitis is used to explain bovine laminitis.

Nutritional influence on laminitis

In equine experimental models for research, laminitis can easily be provoked by feeding excess quantities of carbohydrates, voluntary or infused. The same methods have not been successfully used in bovine experiments, but challenging diets have provoked more or less laminitis symptoms and claw horn lesions. Researchers have used claw horn lesions as retrospective tools to estimate the influence of various laminitis risk factors (*Bergsten* 1993, *Peterse* 1980). Most strikingly *Livesey & Fleming* (1984) showed that a separate fed diet with restricted forage resulted in 68% of the cows with clinical laminitis symptoms at calving, followed by 64% of them having sole ulcers two to three months afterwards. The control group fed the same amount of concentrates, but provided free access to forage, and it had 8%

with clinical laminitis and 8% with sole ulcers. The study also showed the association between clinical laminitis and sole ulcers.

In a series of metabolic studies, a higher concentrates-forage ratio (*Manson & Leaver* 1987), a higher concentrate amount (*Manson & Leaver* 1988a), and a higher dietary protein intake (*Manson & Leaver* 1988b), all resulted in higher lameness scores than the controls that were fed less intensive diets. The lesions associated with the lameness were sole ulcers and sole hemorrhages. Sole hemorrhages are considered to be symptom of subclinical laminitis (*Greenough* 1985).

Peterse et al. (1984) showed in a two-year cross over trial that separate fed concentrates at a higher concentration caused significantly more sole ulcers. In a parallel study with mixed diets, a higher concentrate ration also resulted in more sole ulcers, although the difference was only significant the second year.

Not only the diet composition, but also the way it is prepared and fed, and the feeding behavior of the animals are important risk factors for laminitis. In a Swedish study in tie-stall herds with separate concentrate feeding, there was no significant association between sole hemorrhage scores and the diets fed. On the other hand, high sole-hemorrhage scores were significantly correlated with feeding concentrates less than four times daily, less time to feed at the manger and feeding concentrates before roughage (*Bergsten* 1994). These results indicate an interference with the rumen metabolism due to improper feeding management. Recent U.K. studies compared wet and dry forages fed to heifers before calving under the same housing conditions. Wet diets (grass silage DM 19%) resulted in significantly more lameness and sole lesions before and after calving than dry diets (straw and concentrates DM 86%), although other ingredients were comparable.

A concentrate diet fed without enough func-

tional fibers from the forage results in less chewing, so the cow produces less buffering saliva. It may result in a drop of the rumen pH and more rapid passage of the ingesta through the alimentary tract. Based on the clinical observations Nordlund (1995) suggested an association between rumen acidosis and laminitis. De Chant *et al.* (1998) investigated different diets around calving, rumen pH, lameness and sole lesions. More sole lesions were seen two to three months after calving when changing from a high- to a low-fiber diet at calving compared to feeding the same diet before and after calving, or changing from a low- to high-fiber diet. No associations were, however, found between rumen acidosis (pH < 5.8) and sole lesions on an individual basis, or between different diets and acidosis when sampling at 8 and 22 DIM.

It is not clear whether to blame the acidosis itself or the process possibly causing the acidosis. Andersson (1981) infused large amounts of lactic acid directly in the rumen. Despite rumen pH of 5.0, he observed no symptoms of laminitis or sole lesions. Momcilovic *et al.* (2000) reduced rumen pH and increased blood D-lactate by giving high, readily fermentable diets to steers. However, the author did not succeed to provoke laminitis despite some symptoms of discomfort among the calves. Prentice (2000) noticed in an experimental study where steers were overfed starch to reduce rumen pH that a steer in the study group fed excessive amounts of starch did develop laminitis and sole lesions despite the rumen pH never going below 6. Other calves in the experiment group that received less starch, but had lower rumen pH, did not, however, develop laminitis. Thus, the high-starch diet could possibly provoke laminitis and sole lesions without necessarily reducing rumen pH.

Collard *et al.* (2000) observed the energy balance and health in a dairy herd and found an association between a high-metabolic load and

laminitis. Animals with laminitis had a significantly longer and more negative energy balance at 50 and 100 days after calving, and a more severe minimum and total energy deficit than non-laminitis cows. However, in a present trial it was not possible to evaluate cause and effect of the results.

There is evidence that biotin has an important role in the integrity of the hoof wall and thus, most likely in the development of laminitis. The rumen normally produces biotin, but it is probable that an acid environment could alter biotin-producing microorganisms. Biotin supplementation has shown to decrease the incidence of white-line lesions (Midla *et al.* 1998, Green *et al.* 2000).

Pathophysiology; missing link behind the nutritional influence

There are several theories that try to explain the alimentary background to the vasculatory dysfunction that affects the attachment of the hoof bone inside the hoof capsule.

When bovine laminitis was first described in the 1960s (Nilsson 1963), the release of histamine from protein sources in the diet was found to be a reasonable explanation to laminitis. This was also an equine theory of the cause of laminitis from the first Ph.D. thesis on equine laminitis some decades earlier. Åkerblom (1977) demonstrated experimentally that *E. coli* bacteria enzyme could decarboxylate histidine to histamine in protein-rich grain. It has not, however, been possible to provoke laminitis symptoms by injecting histamine alone in the blood in equines or bovines. However, when steers were first overfed grain followed by a histamine injection, they showed severe laminitis symptoms that lasted for one to 4 days. (Takahashi & Young 1981)

Another often-adopted explanation of the etiology is that large quantities of endotoxins (toxin released by gram negative bacteria such as *E.*

coli) are produced when the gastro-intestinal metabolism is disturbed in an acid environment. The mucous membranes of the gastro-intestinal system normally protect it from absorbing toxins into the blood stream. The mucosa of the bovine fore stomachs are especially fairly resistant while the intestines may be more susceptible. That may explain the higher susceptibility for laminitis in horses than in cattle. But, if the natural barrier is weakened or damaged and if absorbed to the circulatory system, endotoxins are extremely potent and trigger a prostaglandin cascade (chain reaction). An imbalance of the prostaglandins thromboxane and prostacycline is evident, and trombes are produced, which obstruct the small blood vessels (capillaries) of the laminar corium. The blood circulation is locally deteriorated and the result can be compared to a "heart attack" of the feet. The decreased oxygen and nutrient supply damages the corium's horn-producing cells, as has been explained earlier. Elevated levels of endotoxins have been observed in laminitis and trombosis was evident in the corium (*Anderson & Bergman* 1980). Researchers have also tried to provoke laminitis by injecting endotoxins into the circulatory system in cattle with results hard to interpret (*Mortensen et al.* 1986). A more recent hypothesis of equine laminitis has found that laminar enzymes, possibly metalloproteinases, have altered the basal membrane of the laminar corium. Once the junction is affected the corium and horn could be stretched apart from each other, relative to the severity of the lesion (*Pollitt* 1996). *Streptococcus bovis* in the equine gut has been suggested as a potential metalloproteinase activator in acute laminitis (*Pollitt* 1999). This is interesting as background for bovine laminitis, as lactic acid producing bacteria are associated with the disease. So far, this hypothesis has not been reproduced in cattle.

Traumatic effects of laminitis

Once the claw bone attachment in the horn capsule has been disrupted the claw is at risk for further damage. Due to the severity of the disruption, loading and biomechanics, the claw bone sinks and/or rotates more or less permanently inside the horn capsule and the prominent parts of the bone contuse the adjacent sole corium (*Ossent & Lisher* 1998). The contusion or pinching of the corium depends on the counter pressure and causes a secondary inflammation of the sole corium with edema and hemorrhaging. The hemorrhages from the corium will be incorporated into the growing horn and will be visible at trimming. The lesions on the sole can be compared to a print of the bone where it hits the sole. The most commonly affected regions are the rear part of the claw bone, recognized as the "typical" sole-ulcer site, the junction of the sole and white line of the posterior outer wall, and the toe area. The time lapse from the initial insult until the hemorrhage can be detected depends on the growth rate of the sole and the sole thickness.

As it takes two to three months until the hoof lesions are visible, the close relationship between laminitis and hoof lesions has not always been well understood. Sometimes sole hemorrhages are misinterpreted as stone bruises. Changes of the bone position inside the claw capsule also affect the grooving wall. You can see a horizontal break point of the wall, also known as a hardship groove or laminitic ring, after each disruption of horn growth (*Greenough* 1985). The newly produced wall horn of the upper part follows the new position of the bone while the lower part of the wall, beneath the groove, reflects the position of the bone before the laminitis period. Thus, the rotated bone inside the capsule is reflected as a concavity of the wall outside. In claws from slaughtered cows with chronic recurrent laminitis, a concavity of the dorsal wall was associated with a permanent ro-

tation of the claw bone and protrusion of the corium (Kehler & Sohr 2000).

Claw conformation and weight distribution

The weight distribution between the inner and outer claws, of the sole and wall area within each claw, and the shock absorbing mechanisms in the foot are related to the trauma of the sole corium towards the floor. At normal gait the heel bulbs and the outer wall will make the first contact with the ground and the weight will be distributed equally between the outer and inner claw. While the soft bulbs reduce the shock of the rear part of the claws the weight on the wall will successfully be transferred on the sole due to slight splaying of the claws. Inside the capsule, the suspensory apparatus made of collagen tissue and the digital cushions made of fat pads protect the sole corium.

Sole lesions are rarely seen in free-ranged cattle on a soft foundation and their soles are concave from the outer wall to the central part. Tranter & Morris (1992) found that when animals were moved from pasture to hard floors during lactation, the rear outer claw's natural sole concavity disappeared while the inner claw's concavity remained. This is a common observation from claws that have been on concrete for a while. A vicious cycle starts, which increases sole growth and wear in the overloaded area and causes asymmetric claws. Asymmetric rear claws, where the outer claw is larger than the inner claw and the sole is flat, is the most common site for sole lesions. Consequently, if the natural concave shape of the claws has disappeared, and the sole is flat or convex, the sole will take more weight than the wall initially, and shock absorption will rely more on the suspensory apparatus and the digital cushion. Lischer *et al.* (2000) compared normal feet and feet affected with sole ulcers from slaughtered cows. Claws with sole ulcers had a more sunken claw bone, and more

compressed soft tissues and less fat in the cushions.

Risk factors for laminitis other than diet

Changes around calving

Often, dairy cows experience many changes around calving, such as being introduced to lactation diet and new housing facilities. First-time heifers also experience these changes and they are thus presumed to be more susceptible than pluriparous animals. Vermunt & Greenough (1996) observed sole hemorrhages in Holstein heifers several months before calving and the lesions were more severe in housed animals than those in a dry lot. Some of them developed sole ulcers shortly after calving and the lesions of the all the animals were reduced at two months after calving. Also, Logue *et al.* (2000) scored sole lesions in heifers several months before calving, and white-line lesions peaked two months after calving and sole hemorrhages four months after calving. In my study (Bergsten & Frank 1996a), 60 heifers in early pregnancy were tied on either concrete floors or on rubber mats and were challenged with high- or low-concentrate diets. Sole-hemorrhage scores were generally low and no differences were found between groups. The same animals were grazed during the summer, regrouped, and housed on concrete floors or rubber mats in the fall. They were allocated to either a high- or a low-concentrate diet (Bergsten & Frank 1996b). All claws were scored for sole hemorrhages at trimming two weeks before and again 14 weeks after calving. The animals on the concrete floors had significantly higher scores than those on rubber mats. The combination of high-concentrate feeding and concrete floors resulted in significantly more sole hemorrhages than the low-concentrate diet and rubber mats. Webster (2000) found similar results where animals housed in cubicles with concrete floors had significantly more sole lesions than those

housed on straw yards, independent of diets. The lesions reached their highest values about eight weeks after calving.

In the same series of my experiments, another group of heifers was tied up on concrete floors during the winter before calving in spring. The presence of sole hemorrhages was compared to the sole-hemorrhage scores of heifers calving on concrete floors the previous autumn with the same diets. The spring-calvers had higher scores than the autumn-calvers before calving, but significantly lower scores after calving (Bergsten & Frank 1996b). The results were interpreted as spring-calving heifers had a longer period to adapt to concrete floors before calving, compared to the autumn-calving animals who came from pasture just a few weeks before calving. It could be concluded from the experiments above that calving per se, and environmental and management changes before calving were the main contributing factors for subclinical laminitis.

Floors and exercise

As revealed in the experiments above, hard floors increase the risk of subclinical laminitis. Also in a field study, animals on concrete floors had higher sole-hemorrhage scores than those tied on rubber mats (Bergsten 1994). When cows tied on rubber mats were compared to cows in cubicles with rubber mats and concrete slatted floors, significantly more white-line hemorrhages were found in the latter group (Bergsten and Herlin 1996). Danish studies came to the same conclusion that tied animals on rubber mats had less hoof lesions than those in loose housing (Thyssen 1987). These findings are in accordance with the observations that white-line lesions and trauma of the wall are seen less seldom in tied than in free moving cattle on hard floors (Rowlands *et al.* 1983). A tearing of the hemorrhagic weakened wall, as when the animal turns around, can cause a fis-

sure between wall and sole in the white line and a white-line abscess.

More recent studies (Jungbluth *et al.* 2003) found clairy cows in a free stall barn on soft slatted rubber mats to have significantly less laminitis related hoff lesions than those walking on traditional concrete slatted floors.

Cow comfort and behavior

With a comfortable environment, dairy cows will lie down 12 to 15 hours a day and most often when ruminating. Comfortable stalls mean soft bedding and enough space for rising and lying down. In uncomfortable stalls, cows often stand half in the cubicles with their rear feet on the alley. A longer standing time increases the loading and exposure to unhygienic materials, particularly for the rear feet. Leonard *et al.* (1994) illustrated the effect of prolonged standing due to uncomfortable stalls. Heifers were introduced either to comfortable cubicles equipped with rubber mats and open dividers, allowing better space for rising and lying down; or to concrete-based cubicles without bedding, with dividers more closed on the sides, and without lunge space. Researchers assessed lying time and sole-hemorrhage scores before and after the cows had been introduced to the cubicles at calving. At calving, heifers were lying significantly longer in the more comfortable stalls. Two months after calving, the animals in the uncomfortable stalls had significantly more sole hemorrhages. The sole hemorrhages thus reflected the longer standing time due to poor comfort that took place 2 months earlier.

The animals' behavior when being fed can also influence the development of laminitis. Leonard *et al.* (1996) scored the interaction behavior in animals where the feed space at the manger was reduced. The sole-hemorrhage scores were significantly higher in the animals with a high interaction score than in those with low interactions. The study concluded that

competing for food at the manger could provoke interactions between animals resulting in sole hemorrhages. On the other hand, the behavior of a lame cow will be affected. *Manson* (1989) found that lame cows lost rank in the herd, ate for a shorter time, consumed less dry matter, and had a higher eating rate than healthy herd mates.

Claw trimming

Claw trimming has proved to reduce lameness associated with laminitis when correctly performed. *Manson & Leaver* (1988b) showed that trimming the feet before calving resulted in fewer lame cows than when the feet were not trimmed. Preliminary results from a Swedish study (*Manske et al.* 2001) showed almost twice as many sole ulcers in animals trimmed only once each year compared to those trimmed twice. One function of claw trimming is to detect lesions at an early stage before clinical symptoms or severe lesions develop. The other part is to prevent lesions by correcting the loading. *Distl & Mair* (1990) showed that when the distribution of weight between the claws is unequal and the sole bears too much weight, there is greater risk for sole lesions. When the feet are trimmed, the weight is more equally distributed between and within the claws. However, it is more difficult to equalize the weight distribution in cows with excessive wear, i.e. cows in cubicles on abrasive concrete floors.

Preventing laminitis, claw lesions and lameness

Laminitis with secondary claw lesions is frequently a herd problem. As with other production diseases, laminitis is multifactorial and management decisions are critical to reduce most laminitis risk factors. Thus, the possibility to prevent laminitis increases if farmers or managers understand and are aware of the problem and its consequences. There will be larger

herds, and breeding for higher milk yield will continue. Laminitis-related lesions have shown to have a high heritability (*Manske* 2001, personal communication). It would be beneficial to include lameness and claw lesion records in breeding programs in the future. Such records can also help dairy producers monitor herd problems and make decisions to improve hoof health, and to evaluate the effects of a preventative protocol (*Bergsten* 2000)

Diet and feeding management

Based on experimental and epidemiological studies it is evident that diets and feeding management are of great importance for laminitis. All measures to increase optimal rumination and reduce the risk for acidosis should be made (*Nordlund* 1995). Precautions concerning feeding management include: a gradual adjustment to a lactation diet, use feeding routines that stimulate natural digestion and feed well-balanced diets with enough functional fiber to promote rumination.

Even when following up-to-date recommendations, lameness problems can occur in a herd. Troubleshooting should include not only the diets that are offered, but also how the diet is prepared and what actually is consumed (*Shaver*, 2000). Over mixing total mixed rations can reduce the mechanical efficiency of fiber in the rumen. The same phenomena can occur when cows sort feed at the manger. If there are reasons to believe that trace elements, minerals and biotin levels are insufficient, one should supplement these items. Biotin can be supplemented at 20 mg per animal per day. However, due to the rate of horn growth, do not expect results from the supplementation until months afterwards (*Seymour* 1998).

Housing and cow comfort

The dairy industry's awareness of concrete floors' negative influence on claw health has in-

creased during recent years. Primary advice is to provide a long enough adjustment period, especially if moving animals from pasture or deep bedding to harder floors. Changing ground surface just before calving should be avoided, and this adaptation should be made at least two months before calving.

Rubber mats have proved to be a durable material that reduces the risk of laminitis. Using rubber mats on concrete areas is seen more and more as a measure to reduce claw-floor challenges. Many farms put rubber on the floors along feed bunks, in holding pens and on the alleyways to the milking parlor. This is a good prevention, but could, depending on cleaning system, interfere with the hygiene. To get a softer and more hygienic environment for the claws, and thus prevent problems, a specific type of feed stalls have been constructed in Swedish cubicles barns. The individual feed stalls are 1.60 m long, on a 0.2 m elevated platform and use dividers between each cow (0.80 m distance). The manger is elevated 20-40 cm above the foot level to make eating more comfortable. The cow can stand with all four feet on the rubber mat and still deposit manure on the alley behind the platform. With feed stalls the alleys can be scraped or flushed more frequently without disturbing the animals when they are eating, unlike in traditional systems. Moreover, feed stalls reduce stress and social interactions in the herd by improving cow traffic and giving continuous access to food. Using cubicles with enough lunge space and soft bedding to improve lying comfort is noticed more and more. Optimal stall comfort will reduce involuntary standing and exposure to concrete. Still, increased walking distances, prolonged feeding time, and increased time in holding pens waiting for milking reduces the time available for lying down, and thus increases the load on the feet and risk for lesions.

Conclusions

Lameness is an increasing problem associated with higher production, more intensive feeding and confined conditions. Laminitis is a complicated disease of which the origin is not clearly understood. It results in claw horn lesions that highly affect the cow's well being, production and longevity. Laminitis can be avoided with physiological digestion, and secondary claw lesions can be avoided by accurate and physiological loading.

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