Laminitis: Causes, Risk Factors, and Prevention

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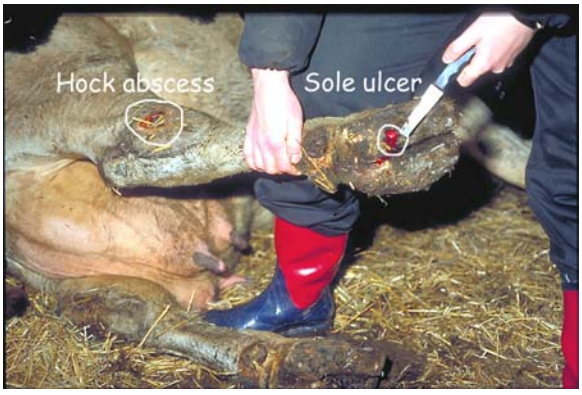
Definition of the Problem

Laminitis has been described in many species, but is most common in the equine and bovine. Although bovine laminitis is most common in lactating dairy cows, it has been reported in all ages and sexes. Laminitis is an inflammation of the laminar corium of the hoof wall, and also includes the sole corium in the bovine definition. 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 that the inflammation is primarily associated with a dysfunction of the digital vasculatory 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 related to nutrition, which will be discussed later. Due to mechanical stretching of the attachment between the inner and outer laminar structures of the hoof wall, which has been affected by inflammation (i.e. laminitis), the claw bone can rotate and or sink inside the hoof. Depending on the severity of the initial lesion, the mobility of the claw bone inside the capsule and the counter pressure on the sole from hard floors, the sole corium can suffer contusions and secondary lesions of the sole area can develop.

The pathological alterations inside the rigid hoof capsule cause considerable pain and lameness, which is an animal welfare issue. A United Kingdom survey (Clarkson et al., 1996) revealed a yearly lameness incidence of 55%. 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 importance than infectious diseases (Kossaibati and Esslemont, 1993), due to severe influence on reproduction and consequently putting the cow at a higher risk for culling (Sprecher et al., 1997). Other common lesions associated with laminitis are: sole hemorrhages and double soles, hemorrhages, fissures and abscesses of the white line, toe ulcers and toe necrosis, and under-running of the heels. Thus, laminitis has a major economic impact on dairy operations, particularly with the worldwide movement towards housing cows in intensive, confined conditions that triggers laminitis problems. As we understand the causes and better identify the risk factors, it will be possible to prevent and reduce the effects of the laminitis syndrome.

History

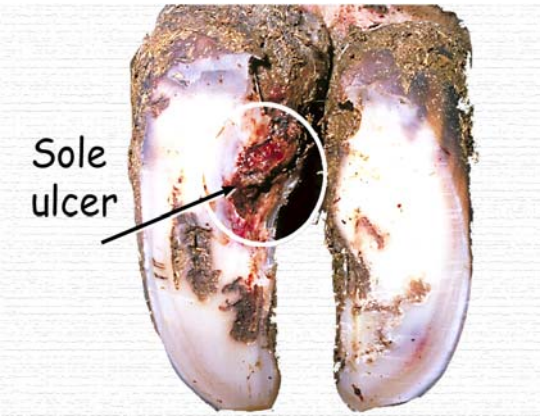
The Greeks (Aristotle) associated equine laminitis with indigestion. The Greek name of the disease, Kritiasis, relates to overfeeding of barley. Xenofon noticed the characteristic symptoms, the hooves started to bleed and the horse became recumbent, which were due to pain in the hooves. These are the same symptoms observed in cattle today (Picture 1). During the last centuries, science has also considered that trauma significantly contributes to the disease development. Later, toxic or chemical agents through the feedstuff or metastatic spread from infections have been proposed as etiology (cause of disease).



Picture 1

Bovine laminitis has a shorter history in the literature than equine laminitis. The Swedish Veterinary Journal in 1896 reported, “Inflammation of the claw corium is the most common cause of lameness and the often deformed claw can be secondarily affected by a purulent process and/or the claw capsule could fall off. The affected animal is useless for work due to life-long lameness”. Rusterholz disease, i.e. sole ulceration (Picture 2), was described in the 1920s (Rusterholz, 1920), but was not associated with laminitis at that time. The first Ph.D. thesis on bovine laminitis was published

by Nilsson (1963). It included a thorough description of the symptoms and the pathology, which is still relevant today. Since the same clinical symptoms are observed in equine laminitis, it is natural that research on the etiology of equine laminitis is used to explain bovine laminitis also.



Picture 2

Nutritional Influence on Laminitis

In equine experimental models, laminitis can easily be provoked by feeding excess quantities of carbohydrates, voluntarily consumed or infused. The same methods have not been successful in bovine experiments, but challenging diets have provoked laminitis symptoms and claw horn lesions. Researchers have used claw horn lesions as retrospective tools to estimate the influence of various laminitis risk factors. Most strikingly Livesey and Fleming (1984) showed that restricting forage in the diet resulted in 68% of the cows with clinical laminitis symptoms at calving, and 64% of them got sole ulcers two to three months later. The control group fed the same amount of concentrates, but provided free access to forage had 8% clinical laminitis and 8% sole ulcers. The study also showed the association between clinical laminitis and sole ulcers. In a series of metabolic studies, a higher concentrate-forage ratio (Manson and Leaver, 1987), a higher concentrate amount (Manson and Leaver, 1988a), and a higher dietary protein intake (Manson and Leaver, 1988b), all resulted in higher lameness scores than in controls 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). In a two-year cross over trial Peterse et al. (1984) showed that separately fed diets with higher concentrate ratios caused significantly more sole ulcers. In a parallel study with total mixed rations, a high-concentrate diet also resulted in more sole ulcers, although 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 the diets fed and sole hemorrhage scores. On the other hand, high sole hemorrhage scores were significantly correlated with the feeding of concentrates less than four times daily, less time to eat at the manger and the feeding of concentrates before roughage (Bergsten, 1994). These results indicate an interference with the rumen metabolism. Recent U.K. studies (Logue et al., 2000) compared wet and dry forages fed to heifers before calving under the same housing conditions and with the same energy and protein in the diets. High moisture diet (grass silage, DM 19%) resulted in significantly more lameness and sole lesions before and after calving than dry non-fermented diet (straw and concentrates DM, 86%).

A concentrate diet fed without enough functional fiber from the forage results in less chewing, and, consequently less buffering saliva. It may result in a drop of the rumen pH and a more rapid passage of the ingesta through the rumen. Based on 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. No associations were, however, found between rumen acidosis (pH < 5.8) and sole lesions on an individual cow basis, or between different diets and acidosis when sampling at 8 and 22 days in milk.

It is not clear whether the acidosis *per se* or the process causing the acidosis is most significant for the development of laminitis. Andersson (1981) infused large amounts of lactic acid directly into 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. They could not succeed in inducing laminitis although some symptoms of discomfort were seen. Prentice (2000) noticed in an experimental study, where steers were overfed carbohydrates to reduce rumen pH, that a steer developed laminitis and sole lesions although rumen pH was not below 5.9. Other calves did not develop severe laminitis although they

had a lower rumen pH and rumenitis. Thus, the high-starch diet could possibly provoke laminitis and sole lesions without necessarily causing rumenitis or reducing rumen pH.

Collard et al. (2000) observed the energy balance and health in a dairy herd and found that animals with laminitis had a significantly longer and more negative energy balance at 50 and 100 days after calving, and a more severe energy deficit than non-laminitis cows. However, 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, and feedstuffs contribute, but it is possible that the quantities are insufficient in high yielding dairy cows and/or that an acid environment negatively affects biotin-producing microorganisms. Biotin supplementation has been shown to decrease the incidence of white-line lesions (Midla et al., 1998; Green et al., 2000).

Pathophysiology: Missing Link to the Nutritional Influence

There are several theories behind 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 (Nilsson, 1963), the release of histamine from protein sources in the diet was supposed to be an explanation for laminitis. This was a theory of the cause of equine laminitis already in 1934. Åkerblom (1977) in the first Ph.D. thesis on equine laminitis, demonstrated experimentally that E. coli bacteria enzymes could decarboxylate histidine to histamine in protein-rich grain. It has not, however, been possible to provoke laminitis symptoms by a separate histamine injection intravenously either in equines or bovines. But, when steers were first overfed with grain and then given a histamine injection, they showed severe laminitis symptoms that lasted for one to four days (Takahashi and Young, 1981).

Another often-adopted explanation to the etiology of laminitis is that large quantities of endotoxins (toxin released from G-negative bacteria such as E. coli) are produced and absorbed, such as when the gastro-intestinal metabolism is disturbed in an acid environment. The mucosa of the bovine fore stomachs are particularly resistant, while mucosa of the intestines may be more susceptible, which may explain the higher susceptibility for laminitis in horses than in cattle. In the circulatory system endotoxins are extremely potent and trigger a prostaglandin cascade (chain reaction). An imbalance of the prostaglandins tromboxane (increasing) and prostacycline (decreasing) will follow. Trombes will be produced, which obstruct the capillaries of the laminar corium in the hoof. 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. Elevated levels of endotoxins have been observed in equine laminitis and trombosis has been found in the bovine corium (Andersson and Bergman, 1980). Trials to induce laminitis by injecting endotoxins into the circulatory system have been done in calves, but the results have been difficult to interpret (Mortensen et al., 1986).

A more recent hypothesis of equine laminitis is that enzymes, possibly metalloproteinases, have altered the basal membrane of the laminar corium. If their junction is affected, the corium and horn could be stretched apart, in proportion 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). Theoretically this might be a background also for bovine laminitis, since lactic-acid producing bacteria are associated with the disease. However, this hypothesis has not been verified 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 and Lisher, 1998; Figures 1,2). The degree of the contusion or

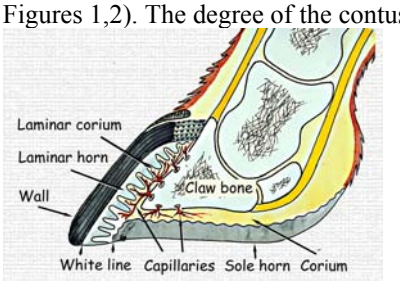


Figure 1

pinching of the corium depends on the counter pressure and causes a secondary inflammation of the sole corium with edema and hemorrhaging. The

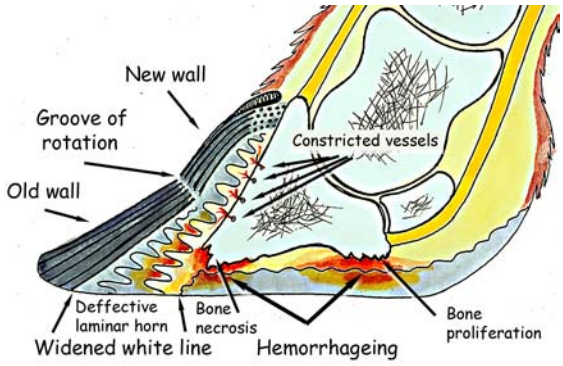
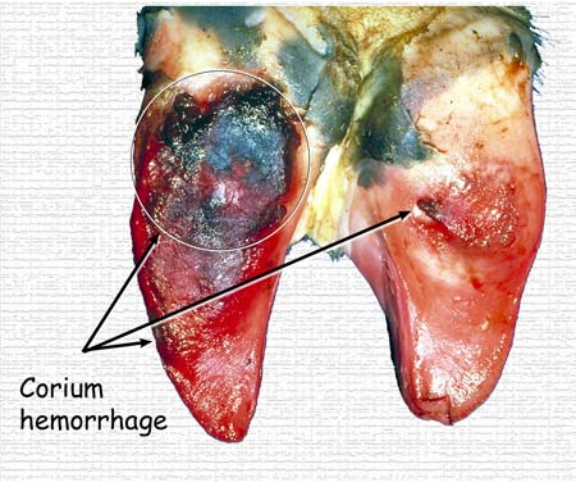


Figure 2

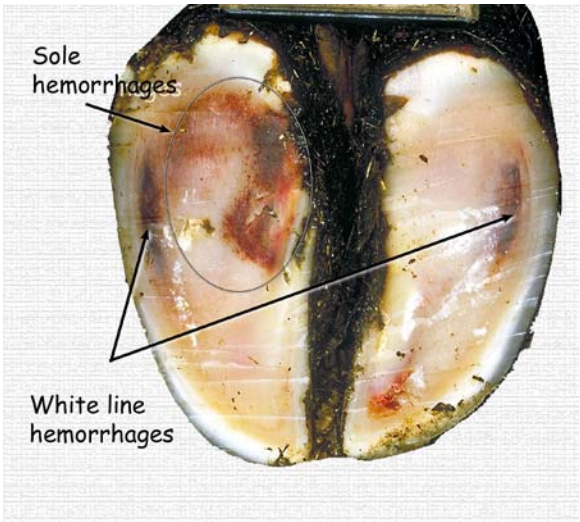
hemorrhages from the corium (Picture 3) will be incorporated into the growing horn and will later be visible at trimming (Picture 4). The lesions on the sole can be looked upon as a print of the bone where it hits the sole. The most commonly affected regions are the rear part of the claw bone, which also is the *typical* sole-ulcer site (Picture 5), the junction of the sole and the white line of the posterior outer wall,



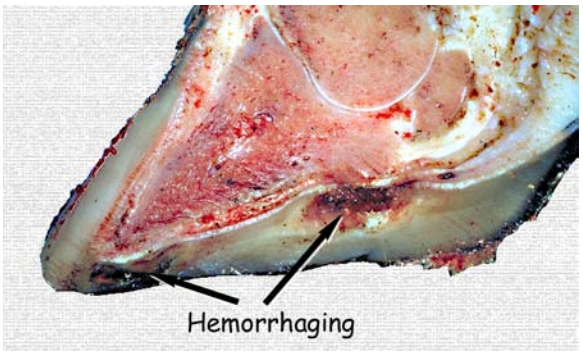
Picture 3

and the toe area (Picture 6). With normal growth conditions, it takes two to three months until the sole lesions are visible, thus the close relationship between laminitis and claw lesions has not always been well understood. Sometimes sole hemorrhages are misinterpreted as stone bruises.

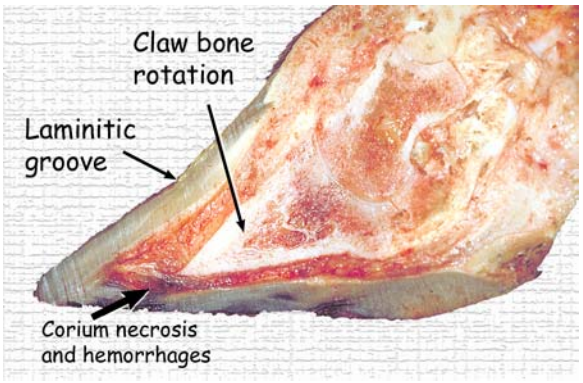
Changes of the bone position inside the claw capsule also affect the grooving of the wall. A horizontal break point will appear on the wall after each disruption of horn growth, also known as a hardship groove or laminitic ring (Greenough, 1985). The newly produced upper part of the hoof wall follows the new position of the bone while the lower part of the wall, beneath the groove, reflects the position of the bone before the laminitic period (Figure 2). 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 rotation of the claw bone and protrusion of the corium (Kehler and Sohrt, 2000).



Picture 4



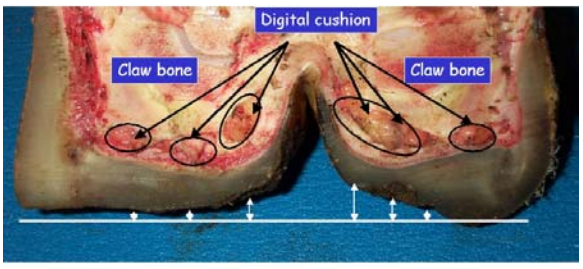
Picture 5



Picture 6

Claw conformation and weight distribution

The claw-floor interactions affecting the corium are dependent on the weight distribution between the inner and outer claws, loading on the sole and wall area within each claw, the suspensory apparatus made of collagen tissue, and the digital cushions made of fat pads (Picture 7).



Picture 7

Sole lesions are rarely seen in free-ranged cattle on soft surfaces. Their soles are concave from the outer wall to the central part. Tranter and 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 growth and wear in the overloaded sole 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 predisposing factor for the development of sole lesions. Because, 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 cushions. Lischer et al. (2000) compared normal feet and feet affected with sole ulcers from slaughtered cows and found that claws with sole ulcers had a more sunken claw bone, more compressed soft tissue and less fat in the cushions.

Risk Factors for Laminitis other than Diet

Changes around calving

Around calving, dairy cows are introduced to a lactation diet and most often also to new housing facilities. In primiparous cows these changes are more provocal and the less experienced cows are thus presumed to be more susceptible to laminitis than pluriparous cows.

Vermunt and Greenough (1996) observed sole hemorrhages in Holstein heifers several months before calving, and the lesions were more severe in housed animals than in those in a dry lot (straw yard). Some of the animals developed sole ulcers shortly after calving and all the lesions were reduced at two months postpartum. Logue et al. (2000) scored sole lesions in heifers from several months before calving. White-line lesions peaked two months after calving and sole hemorrhages four months after calving. In my study (Bergsten and Frank, 1996a), 60 heifers in early pregnancy were tied on either concrete floors or on rubber mats and were challenged with high- or no-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

Table 1: Median sole hemorrhage scores based on sum of scores from all digits in tied heifers before and after their first calving on concrete stalls or rubber mats. The scoring system used a scale from 1 to 5 where 1 means a slight hemorrhage of a small area and 5 means exposed corium i.e. sole ulcer, or severe hemorrhages over a large area.

	2 weeks pre partum	13 weeks post partum	Net change in scores
Concrete	1	9	8
Rubber mats	1	4	3
Significance	n.s.	**	*

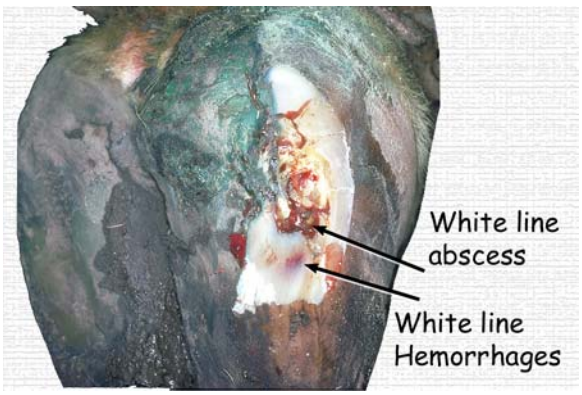
Modified from Bergsten and Frank (1996b). n.s. = not significant * = P < 0.05, ** = P < 0.01

a high- or a low-concentrate diet (Bergsten and 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 (Table 1). Furthermore, 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; 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 experiments, another group of heifers was tied up on concrete floors during the winter before calving in the spring. The presence of sole hemorrhages was compared to the sole-hemorrhage scores of heifers calving on concrete floors the previous autumn on the same diets. The spring-calvers had higher scores than the autumn-calvers before calving, but significantly lower scores after calving. The interpretation was that spring-calvers had a longer period to adapt to concrete floors before calving than the autumn calvers who came from pasture just a few weeks before calving. It could be concluded from the two experiments (Bergsten and Frank, 1996a,b) 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 presented above, and also in the field study (Bergsten, 1994), hard floors increase the risk of subclinical laminitis. When cows tied on rubber mats were compared to cows in freestalls with rubber mats and concrete slatted floors, significantly more white-line hemorrhages were found in the latter group (Bergsten and Herlin, 1996). Other studies also revealed that animals tied on rubber mats had less claw lesions than those in systems where the animals are to move on concrete floors (Thysen, 1987, Rowlands et al., 1983). Moreover, a tearing of the weakened hemorrhagic wall, as when the animal turns around, can cause a fissure between wall and sole in the white line and a white-line abscess (Picture 8).



Picture 8

Even beef cattle experience laminitis and are susceptible to different housing conditions and exercise. Naert et al. (1999) compared diets with different fiber levels and types of housing for beef bulls. There was no difference in subclinical laminitis between the fiber levels, but a group that was able to exercise had less laminitis compared to a group that was not exercised.

Table 2: Medium lying score and medium sole score of heifers in comfortable and uncomfortable stalls,

1 month and 1-3 days before, and 1 month after calving. The sole scores is based on sole hemorrhage scores .

	1 month pre partum		1-3 days pre partum		1 month post partum	
	Lying score	Sole score	Lying score	Sole score	Lying score	Sole score
Comfortable cubicles	0.27	0	0.23[a]	10	0.38	12[a]
Uncomfortable cubicles	0.31	0	0.03[b]	10	0.40	16[b]

Modified from Leonard et al. (1994), values in the same column with different superscripts are significantly different, $P < 0.001$.

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 freestalls with their rear feet on the alley. Longer standing times increase the loading and exposure to unhygienic conditions, 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 freestalls 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 freestalls 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 two months earlier (Table 2).

The animals' behavior when being fed can influence the development of laminitis. Leonard et al. (1996) scored the interaction behavior in animals when feed space at the manger was reduced. The sole-hemorrhage scores were significantly higher in animals with high interaction scores than in those with low interaction score. It was concluded that competing for food at the manger could provoke interactions between animals resulting in sole hemorrhages. Moreover, increased walking distances, prolonged feeding time, and increased time in holding pens waiting for milking reduce the time available for lying down, and thus increase the load on the feet and the risk of lesions.

Also the behavior of a lame cow will be affected. Manson (1989) found that lame cows lost rank in the herd, ate during 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 (Bergsten et al., 1998) showed almost twice as many sole ulcers occurred 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 function is to prevent lesions by correcting the loading. Distl and Mair (1990) showed that when the distribution of weight between the claws is unequal and the sole bears too much weight, there is a 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. in cows in freestalls on abrasive concrete floors.

Preventing Laminitis, Claw Lesions and Lameness

Laminitis with secondary claw lesions is often a herd problem. As with other production diseases, laminitis is multifactorial and management decisions are critical to laminitis risk factors. Thus, the potential to prevent laminitis increases if farmers or managers understand and are aware of the problem and its consequences. In the future there will be larger herds and breeding for higher milk yield will

continue. Laminitis-related lesions have a high heritability (Manske 2001, personal communication). Therefore, it would be beneficial to include lameness and claw lesion records in breeding programs. Such records can help dairy producers to monitor herd problems and make decisions to improve hoof health, and to evaluate the effects of a preventive protocol (Bergsten, 2000).

The two most important preventive measures to reduce laminitis-related lesions are closely related to feeding and housing. Even if the etiology is not clarified, reliable recommendations can be made.

Diet and feeding management

Experimental and epidemiological studies of laminitis have shown that diet and feeding management are of great importance. All measures to increase an optimal rumination and reduce the risk for acidosis should be applied (Nordlund, 1995). Precautions concerning feeding include: a gradual adjustment to a lactation diet, use feeding routines that stimulate a natural digestion, and feeding well-balanced diets with enough functional fiber to promote rumination.

Even when a farmer follows up-to-date recommendations, lameness problems can occur in his 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 (Nocek, 2000). The recommendation for daily biotin supplementation is 20 mg per animal (Seymour, 1998). However, due to the slow horn growth, results from supplementation may not be evident until several months afterwards.

Housing and cow comfort

The awareness of the concrete floors' negative influence on claw health has increased during recent years. Primary advice is to provide an adjustment period of at least two months before calving, especially when moving animals from pasture or deep bedding to harder floors. Rubber mats reduce the risk of laminitis, and rubber mats on concrete areas are used more and more 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 good prevention, but could, depending on the cleaning system, interfere with hygiene.

To get a softer and more hygienic environment for the claws, a specific type of feed stall has been constructed in Swedish freestall barns (Picture 9). The individual feed stalls are ~1.60 m long, on a 0.2 m elevated platform with a divider between cows (0.80 m distance). The manger is elevated 20-40 cm above the foot level to make eating more comfortable. The cow can not lay down but can stand with all four feet on the rubber mat and still deposit manure on the alley behind the platform. With such feed stalls, the alleys can be scraped or flushed more frequently without disturbing the animals when they are eating than in traditional systems. Moreover, feed stalls reduce stress and social interactions in the herd by improving cow traffic and giving continuous access to feed, which has been shown to reduce laminitis.

Picture 9

Freestalls with enough lunge space and soft bedding to improve lying comfort are recommended. Optimal stall comfort will reduce involuntary standing and exposure to concrete.

Conclusions

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

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