

Can We Prevent Hoof Problems?

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■ Take Home Message

Lameness remains a major problem in dairy cattle worldwide. In the UK the average incidence is around 50 cases per 100 cows per year, with much higher incidences being seen in some free-stall housed cattle. Because of its effect on subsequent fertility and production, the cost of a single case of lameness is estimated to be around £200 (\$450 Canadian), although this will vary enormously from case to case depending on severity. The prevention of any disease must be based on a thorough understanding of its cause, and the following points are discussed in this paper:

- ▶ the structure of the foot, viz. hoof, corium and bone
- ▶ how dysfunction of the corium ('coriosis') leads to the production of defective hoof horn
- ▶ when the defective horn reaches the weight bearing surface of the foot, lesions causing lameness may develop
- ▶ calving is a major cause of coriosis, which is why peak incidence of lameness is seen 2 to 4 months into lactation. Hoof growth stops but wear increases. This leads to a thinning of the sole, which in itself predisposes to further bruising and coriosis
- ▶ many of the other on-farm factors that predispose to coriosis also occur at the time of calving, and include feeding, excess standing and changes in housing and management.

■ The Structure of the Foot

The foot consists of two digits, the outer or lateral claw and the inner or medial claw. In hind feet the outer claw is larger than the inner and is the major weight-bearing surface. In front feet this is reversed, with the inner claw being the larger and weight-bearing. This is thought to be an important reason why

the majority of lesions occur in the outer claw of hind feet and the inner claw of front feet.

Each claw consists of three tissue components, namely hoof, corium and the bone.

- The hoof: subdivided into the wall, the white line, the sole and the heel. It provides the hard outer casing of the foot.
- The corium: also known as the 'quick', the corium is a support tissue that provides nutrients for the hoof and bones of the foot. Papillary corium, which extrudes hoof horn, is present at the coronary band and over the sole, laminar corium covers the wall, and at the heel the corium is modified to form the digital cushion
- The bones: the pedal bone and the navicular bone.

The wall of the hoof is extruded from the papillary corium at the hoof/skin junction of the coronary band. It passes slowly down over the laminar corium at approximately 5mm per month. The sole is a separate structure, produced by the papillary corium of the sole. Where the wall and the sole join there is a cemented junction known as *the white line*. Whereas the hoof of the wall and sole consists of tubular horn viz. hoof containing reinforcing bars, the white line contains only cellular cement. Being a cemented junction the white line is a point of weakness and once damaged becomes a common place for penetration by stones and other foreign bodies.

■ Hoof Overgrowth

Although often omitted from texts on lameness, hoof overgrowth is probably one of the most common factors leading to discomfort when walking. Overgrowth occurs primarily at the toe. This leads to an elevation of the toe, rotating the claw backwards. The angle of the anterior (front) wall of the hoof then changes from a normal of 45° to a slope of 30° and this places additional weight on the heel. Cows with overgrown claws have a poorer gait and walk with significant discomfort compared to well-trimmed animals. In addition, extra weight bearing on the rear edge of the pedal bone may predispose to sole ulcers. It is vital that both veterinarians and herdsman have a good understanding of the anatomy of hoof overgrowth before embarking on hoof trimming. Hoof trimming procedures will be described in the seminar.

■ Sole Ulcers

The primary cause of a sole ulcer is a pinching of the corium between the pedal bone above and the hard horn of the sole beneath. Pinching of the corium

produces bleeding and blood mixed with the horn leads to a weakening of hoof strength. As the average sole is 10mm thick, and as hoof grows at 5mm per month, haemorrhage is not seen on the surface of the sole until some 2 –3 months later. This is often referred to as bruising. Sometimes the compression and damage to the corium can be so severe that horn formation is totally disrupted. This then leads to 'hole' through the hoof and onto the corium of the sole beneath, in other words a sole ulcer.

In the normal foot the pedal bone is suspended within the hoof by the laminar corium. If the laminar corium becomes inflamed, viz. if laminitis develops, then this suspension is lost and the pedal bone sinks onto the floor of the hoof. This further increases the pinching effect. It also explains why cows that have been affected by sole ulcers or white line disease in one lactation are at least *three times* more likely to develop lameness in subsequent lactations. It is therefore especially important to prevent the development of sole ulcers in heifers.

Treatment consists of removing all under run and damaged horn and preferably applying a block, for example a Cowslip (Giltspur Ltd.), to the sound claw to minimize weight bearing on the ulcer site. Recent data has shown that removing weight bearing from the affected claw by applying a block to the sound claw *doubles* the rate of healing of sole ulcers.

■ White Line Disorders

The white line is a cemented junction between the wall and the sole of the hoof. Both the wall and the sole consist of reinforced tubular horn, whereas the white line is 'cement' only and as such it is an inherent point of weakness. The white line can be further weakened by aspects of management, housing, feeding and particularly by calving. These points are referred to later. Once weakened, it is easy for a stone or dirt to become impacted into the white line cement. The foreign body may act as a 'wedge' and further increase the fissure within the white line until it penetrates the corium. Bacteria carried in will multiply in the corium producing an abscess. It is the pressure from the pus-filled abscess that produces lameness. Infection (pus) escapes from the corium by taking the line of least resistance. This usually leads to a discharge through the softer horn of the heel. Sometimes pus tracks along the laminae of the wall to discharge at the coronary band. If infection accumulates in the toe, then lameness can be particularly intense because there is no route for drainage. Treatment consists of draining the abscess, and removing all the under-run horn to expose the underlying corium. New horn will then grow from that corium. Ideally a block should be applied to the sound claw to remove weight bearing from the affected claw. This reduces weight bearing on the diseased claw, leading to improved cow comfort, increased production and better healing.

■ Prevention Of White Line Abscesses And Sole Ulcers

As both conditions are caused by similar factors, their causes and control are dealt with together. The corium is the horn-forming tissue. Any disruption of the corium can lead to defective horn production, and this is eventually seen clinically as lameness, viz. sole ulcers or white line disease. The corium may become damaged because it is in a fragile state or because of excess trauma or both. The major factors involved in damage to the corium in dairy cattle can be subdivided into the headings of calving, diet, excess standing and general management.

Calving

The corium is in its most fragile state and most susceptible to bruising at the time of calving. The rings on the horns of beef cows, one for each calving, show that there is always a disruption in horn formation at parturition. It also explains why peak incidence of lameness is seen some 8 -16 weeks after calving, this being the period of time it takes for the damaged horn to grow from the corium of the sole to the bearing surface of the foot. Not only does horn growth decrease at the time of calving, but in heifers especially there is increased wear. This will occur from excess standing while they are waiting to be milked, to feed, to find a place to lie down and while there are interactive movements during social recognition with other herd members. The overall effect is a *negative net growth*, viz. the rate of horn growth is less than the rate of wear. This increase in wear relative to growth may be so great that the sole becomes extremely soft and this further bruises the underlying corium and predisposes to sole ulcers and white line disease. . In addition the suspensory apparatus of the pedal bone becomes much more 'elastic' at the time of calving. This allows extra movement of the bone within the hoof, the additional movements leading to friction and formation of a 'blister'. This is seen later as a sole ulcer.

It is not known whether it is the process of calving itself which leads to increased fragility of the corium, or the initiation of milk production. Recent work has suggested it may be the sudden increase in milk production, leading to a massive demand for sulphur-containing amino acids, which could be a critical factor. Sulphur-containing amino acids are essential both in the construction of keratin, the main component of horn, and for milk production. Clearly at the time of calving there is a sudden repartition of nutrients towards milk production and perhaps this leads to the formation of poor quality horn. Recent studies in a UK intervention study involving over 1100 cow-years on trial showed that cows which develop lameness gave more milk (366 litres) than their non-lame counterparts. However, within this higher yielding 'lame cow' group, when cows went lame their yields reduced by an average of 396 litres per cow (Green et al 2002). The interesting feature is that, using

sophisticated data analysis, the reduction in yield was detectable for several months before the onset of clinical signs of lameness. This strongly suggests that there is a primary defect that later leads to the onset of hoof problems.

Not only is the corium inherently more susceptible to damage at calving, but there is often a change in diet, housing and social grouping superimposed immediately after calving which can lead to further damage to the corium and increase the incidence of lameness. The effects of this will be more severe in heifers.

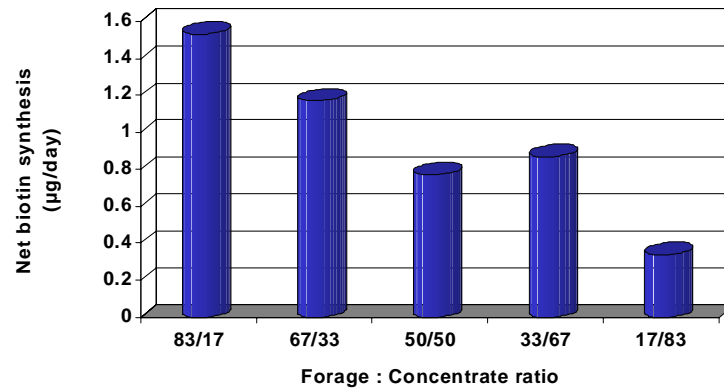
Feeding

A second feature of calving is the decrease in the rate of rumination shown by all cows.

This reduced rumination combined with the marked increase in concentrate feeding which occurs at the time of calving leads to an increased risk of rumen acidosis. Rumen acidosis has been linked to coriosis, leading to an even greater increased fragility of the corium. The problem is compounded by rations that are low in effective long fibre.

Rumen acidosis also leads to depressed biotin synthesis, and lack of biotin further exacerbates poor horn formation, especially of the white line cement. Figure one shows that changing from 83:17 forage : concentrate to a 50 – 50 ration *halves* the rate of ruminal biotin synthesis (Da Costa Gomas et al 1998). In one UK trial involving 1109 cow-years, supplementation with 20mg/day of biotin lead to a 50% reduction in lameness caused by white line disease. No effect was seen until after 120 days of supplementation, this being the period that it takes for the white line cement to reach the weight-bearing surface of the sole (Hedges et al 2001, Figure two). In addition, only 17% of biotin supplemented cows were re-examined for white line lameness compared to 30% of the non-supplemented control cows (Blowey et al 2000). Many dairy rations are now supplemented with biotin, although clearly the best preventive measure is to avoid the development of periparturient rumen acidosis.

Synthesis of Biotin - an *in vitro* study



(Da Costa Gomez et al., 1998)

Figure One. The influence of dietary acidosis on the rate of *in vitro* rumen biotin synthesis.

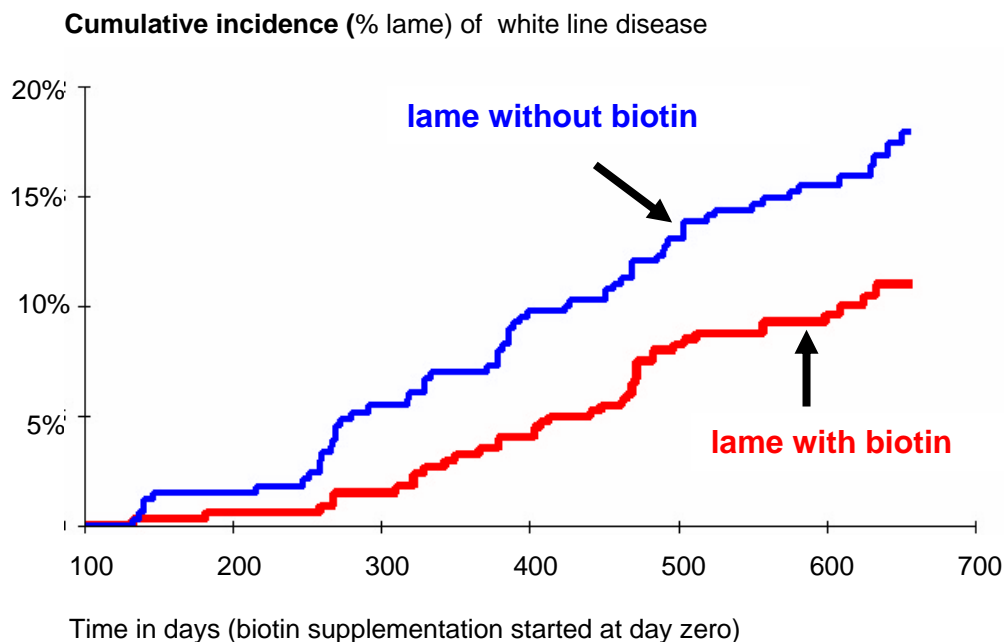


Figure Two. The influence of 20mg/day biotin supplementation on the cumulative incidence of clinical lameness caused by white line disease in dairy cattle (Hedges et al 2001)

The rate of resumption of rumen contractions immediately post-calving is considerably affected by the quantity of long fibre in the diet. Cows on low fibre/high concentrate diets have much poorer rumen movements and are much more susceptible to rumen acidosis than cows on high long fibre diets. Straw is an excellent material to ensure that rumen contractions – and therefore rates of salivation and sodium bicarbonate production - are maintained at an optimum level. Prepartum concentrate feeding in the transition ration is also important as this stimulates the development of the rumen papillae. Well-developed papillae are then present to absorb the acid produced by high concentrate post-partum rations and there is less risk of rumen acid overload.

Cows should therefore be fed a diet high in long fibre, to stimulate rumination. Concentrates should be built up slowly after calving, reaching a peak at no less than 2-4 weeks after calving. No more than 4.5-5.0kg of concentrate should be given at each feed and the final dietary concentrate:forage ratio should be such that it prevents acidosis.

Excessive standing.

Poor free stall comfort, due to suboptimal dimensions or lack of bedding, and heifers that have not been trained to use the free-stalls, will both lead to increased standing. Lack of loafing area and blind-ending passageways which heifers particularly feel reluctant to enter, further exacerbates the problem. The post-partum heifer is particularly susceptible to these changes as she is often brought into the milking herd with very little preparatory integration. She is often last to feed and to be milked, and she must establish herself in the social dominance order within the herd. All of these changes occur around the time of calving when the growth of sole horn is minimal.

Management.

Horn absorbs moisture from the environment and damp horn is softer than dry horn. Cows should therefore be managed and housed to keep their feet as dry as possible. This can be quite difficult in free-stall systems. Regular twice daily scraping of the passages, plus a sprinkling of lime added to the free-stall bed to aid mastitis control, helps to keep hooves dry. Cows should be handled gently. Rushing them along tracks means that they are unable to avoid standing on stones and this can lead to additional bruising of the corium, especially in the freshly calved cow where the corium is already more fragile and the sole is thin due to 'negative net growth'. The provision of special "cow tracks", with a soft tree-bark surface, will help. Feet should not be over-trimmed at drying off, and cows and heifers should be allowed access to hard surfaces during the dry period to promote an increased thickness of the sole.

■ Digital Dermatitis (Hairy Warts)

Low-grade lesions of digital dermatitis are often visible on the feet of dry cows, seen as chronic areas of thickened crusty skin around the interdigital cleft. Although not causing lameness in the dry period, these lesions develop into raw open sores in early lactation to produce typical clinical signs. The control of digital dermatitis must therefore be based on preventing the development of these 'subclinical' lesions, and this can be achieved by foot hygiene, including regular hoof disinfection.

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Enterprises, Box 537, Alexandra Bay, NY13607, and is reprinted with their permission.

■ References

- Blowey RW, Hedges VJ, Green LE and Packington AJ (2000), The Effect of Biotin Supplementation on the treatment of white line lesions in Dairy Cows Proc. Int. Rum. Digit Symp. Parma, Italy, September 2000, pp311 + 312
- Campbel J, Greenough PR & Petrie L (1996) The effects of biotin on sandcracks in beef cattle, Proc. 9th Int. Symp. Dis. Ruminant Digit., Jerusalem, page 29
- Dacosta Gomez C, al Masri M, Steinberg W & Abel HJ (1998).Effect of varying hay/barley proportions on microbial biotin metabolism in the rumen stimulating fermentor RUSITEC. Proc. Soc. Nutr. Physiol., no.7, pp14-28
- L.E. Green, V.J. Hedges, R.W. Blowey, A.J. Packington, and Schukken Y. H. (2002), The impact of clinical lameness on the milk yield of dairy cows Proc. 12th Int Symposium on Disorders of the Ruminant Digit, Florida (in press)
- Hedges VJ, Blowey RW, O'Callaghan C, Packington AJ, , and Green LE (2001), A Longitudinal Field Trial of the Effect of Biotin on Lameness in Dairy Cows, J Dairy Science 84 1969 - 1974

