

Preventing and Treating Lameness

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■ Take Home Message

- ▶ The hoof is a dynamic structure. Produced by the corium, it is growing at around 5mm per month, and under normal circumstances the rate of wear is equal.
- ▶ Damage to the corium leads to the production of poor quality horn. When this comes into wear in the sole 2 – 3 months later defects such as white line disease and sole ulcers may appear.
- ▶ Causes of damage to the corium include calving, excess standing, trauma to the hoof and dietary upsets.
- ▶ Digital dermatitis (hairy warts) is an infectious disorder, and like any other infection control is based on environmental hygiene and identifying and removing the clinical case that acts as a reservoir of infection. In confined housing systems regular (i.e. daily) foot disinfection must become a part of our control program.

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 (cubicle) housed cattle. Because of its effect on subsequent fertility and production, the cost of a single case of lameness is estimated to be around \$450, 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 aetiology. This paper discusses the structure of the foot, the pathogenesis of hoof lesions, in other words how dysfunction of the corium ('coriosis') leads to the production of defective hoof horn and finally the aetiology of lameness, viz. the on-farm factors that lead to defective hoof formation.

■ 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 lateral claw is larger than the medial 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 produces hoof horn, is present at the coronary band and over the sole; laminar corium covers the lower half of 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 from the hoof/skin junction of the coronary band down to the first half of the wall. It passes slowly 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 wall 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.

■ Sole Ulcers

The primary cause of a sole ulcer is a pinching of the corium between the flexor tuberosity of 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 laminae. If the 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 they are at least *three times* more likely to develop lameness in subsequent lactations. It is therefore very important to look after heifers as they enter the dairy herd.
- 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 and it is pressure from the pus that produces lameness. Infection 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.

■ 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.

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 liters) 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 liters per cow (Green et al., 2002). The interesting feature is that, using

sophisticated data analysis, the reducing 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

Increased Movement of the Pedal Bone within the Hoof

The pedal bone is known to be suspended within the hoof by attachments to the laminar corium on the axial and especially the abaxial wall, and by a pedal suspensory apparatus within the hoof that supports the rear of the bone. Increased levels of an enzyme 'hoofase' can be measured within the hoof for the two weeks before and two weeks after calving, and this leads to increased flexibility of the suspensory system, increased movements of the pedal bone, and hence an increased risk of bruising the corium.

Increased Hoof Wear

Many of the changes that occur in the periparturient cow lead to an increase in standing and subsequently an increase in hoof wear. The thickness of the sole at any one time is determined by the rate of growth minus the rate of wear, so an increased wear combined with the decreased growth that commonly occurs at calving will lead to a thinning of the sole. Thin soles further increase the probability of bruising of the corium and subsequent formation of poor quality hoof. Indeed, in some animals, and especially those moved into a new facility in total confinement systems, the sole becomes so thin that this in itself is the cause of lameness. If all four feet are affected then it often results in culling.

Even if they calve outside in a field, for a few days after calving cows, and especially heifers, will spend far more time standing and their lying times will be decreased. It is not known whether the decreased lying times are due to inherent nursing behaviour (attending to the calf), discomfort from the perineum, an enlarged udder, or to some other factor.

A range of factors lead to increased standing (and therefore increased hoof wear) after calving. Milk production starts, and the animal must wait in the collecting yard to be milked. The freshly calved heifer is often the last to enter the parlour, and hence her standing times are even more prolonged. If they produce milk then they must stand eat, and once again it will be the freshly calved heifer that may be forced to wait for the others to finish before consuming her ration. This is particularly the case if there is insufficient feeding space or blind ending narrow passage ways that the heifer is frightened from entering because of the risk of being bullied.

Immune Suppression and Increased Risk of Periparturient Diseases

Diseases such as peracute toxic mastitis are more common immediately after calving, despite the fact that the udder contains high levels of colostrum antibody. This occurs because of the immune suppression that is a feature of every periparturient animal. Two possible reasons for the immune suppression are thought to be 1) because the foetus is antigenically different to the dam, and hence if any foetal fluids leaked into the maternal circulation during the trauma of parturition there would be the risk of an anaphylactic reaction. In addition, 2) the dam might over react to the degeneration of her own tissue that may result from trauma in the birth canal.

Many diseases are therefore more common at the time of calving, and disease in itself also increases the fragility of the corium. In extreme cases, disease such as toxic mastitis will lead to a total cessation of horn production, and this leads to hooves with hardship lines and horizontal fissures. Heifers are likely to be worst affected, partly because they have often had no prior experience of the housing and milking system and partly because in many large dairies the heifers have been reared totally separate from the main herd, and may therefore lack immunity to the infectious agents within that herd. They are then exposed to a whole range of new infections immediately post-partum, when immune suppression is at its maximum and this further depresses horn formation. This leads to long periods of standing, producing increased hoof wear at a time when hoof growth is minimal, often referred to as “negative net growth”. Immune suppression is one possible cause for the marked postpartum increase in DD and foul referred to later.

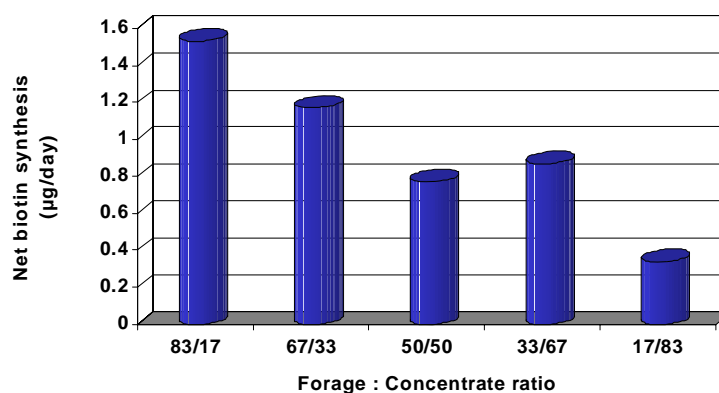
Feeding

A second feature of parturition 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 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 1 shows that changing to a 50 – 50 concentrate to forage ration *halves* the rate of ruminal biotin synthesis (Da Costa Gomas et al 1998). In one UK trial, supplementation with 20mg/day of biotin leads 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 2). The risk of white line disease increases with increasing parity, and for cows in their third lactation and above, biotin decreased the incidence of white line

lameness by a factor of three. In addition, only 17% of supplemented cows required a second treatment compared to 30% of the non-supplemented control cows (Blowey et al., 2000). Many 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 1. The influence of dietary acidosis on the rate of *in vitro* rumen biotin synthesis.

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 a much longer period of ruminal atony 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.

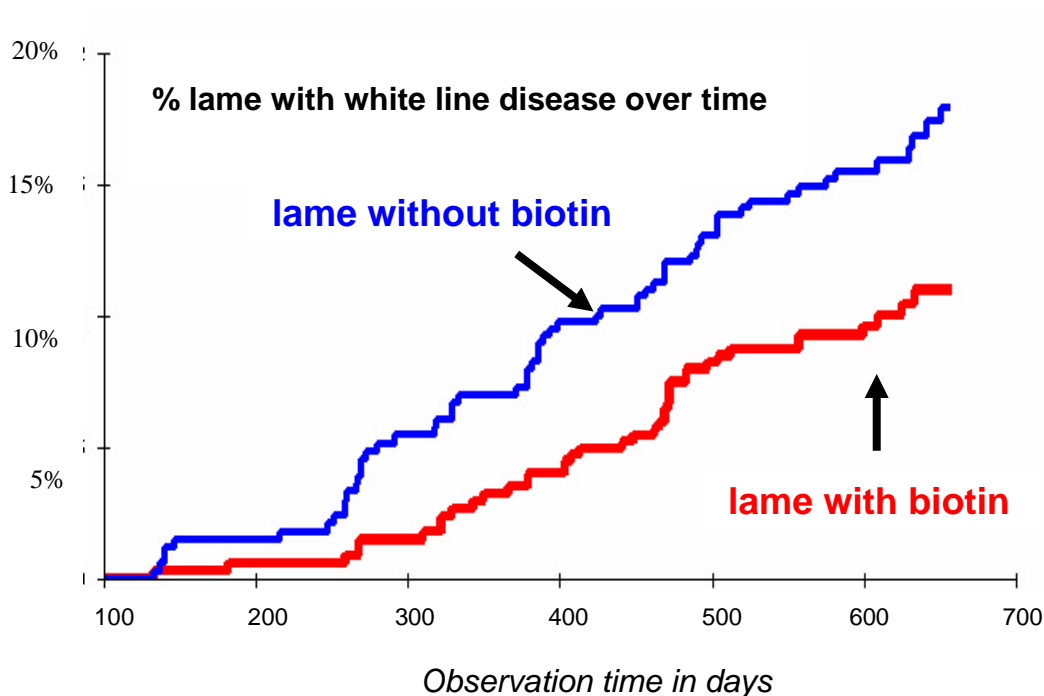


Figure 2. The influence of 20mg/day biotin supplementation on the incidence of clinical lameness caused by white line disease in dairy cattle (Hedges et al., 2001)

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 cubicle (free stall) comfort, due to suboptimal dimensions or lack of bedding, and heifers that have not been trained to use cubicles, will both lead to increased standing. Lack of loafing area and blind-ending passageways which heifers particularly feel reluctant to enter, further exacerbate 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 DD are often visible on the feet of dry cows, seen as chronic areas of hyperkeratotic 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 DD must therefore be based on preventing the development of these 'subclinical' lesions, and this can be achieved by foot hygiene, including regular hoof disinfection. Both DD and foul are known to increase dramatically after calving. This is likely to be due to a combination of periparturient immune suppression and increased standing, the latter leading to wet and dirty hooves. Many farms are simply not prepared to practice the regular hoof disinfection that is needed to control digital dermatitis, and yet the disease can be almost fully suppressed by regular foot bathing. When dealing with DD think mastitis. Regular post dipping (i.e. teat disinfection) is one of the most common mastitis preventive measures. In addition, a clinical case of mastitis is considered to be a risk to other cows and is treated and isolated accordingly. The same approach should be taken with any infectious disease, and especially DD. Clinical cases should be treated using a topical disinfectant or antibiotic, e.g. an intramammary tube, and covered with a dressing until healed. Unless we take this approach, cows with hairy warts will remain a constant reservoir of infection for other cows in the herd, and the problem will never resolve.

The required frequency of bathing will depend on the level of environmental challenge, but routines such as four days per week, or 9 days on and 5 days off are becoming increasingly common. Many farms foot bath cows on a daily basis and are pleased with the results. The foot bath should be sited in the usual cow flow walkway after the exit to the parlour, but not so close to the parlour that it obstructs the exit or slows milking. Anything that slows milking is unlikely to be done on a regular basis!. Ideally there should be enough room for

at least one whole side of the parlour to exit before the foot bath is reached, thus minimising any disruption to milking. Two foot baths placed in series, the first to wash the feet, the second with the active chemical, are ideal, and cows should of course exit into a clean and scraped yard. Make sure that the floor of the bath is comfortable walking surface. If high ridges are present the cows will not like walking through the bath, and this will lead to reluctance to enter, to increased faecal contamination, and a risk that the teats will become splashed with disinfectant. Foot baths with a foam rubber base or similar are sometimes used. When the cow stands on the rubber mat the liquid chemical from within the mat starts to form a pool around the foot and this acts as a bath. The system is easy to use, but it cannot be as effective as a standard foot bath, simply because as the depression produced by the cows foot begins to fill with chemical the cow starts to move onto the next stride and hence to foot does not get as effective a soaking as with the plain water bath.

A disinfectant foam system is also available ('Kovex'), set up such that cows walk through a layer of foam at the entrance to the parlour. The Kovex™ foam consists of a peracetic acid disinfectant, plus peroctranioque acid, a patented booster for the peracetic acid. The foam has adhesive properties to improve the adhesion to the hoof and a green dye is added to reduce the glare of the foam, making it less intimidating for the cows to walk through. A skin conditioner is added and a detergent, to assist the foam to penetrate the foot. The foam is deposited at the entrance to the milking parlour, to a depth of 12-14cms. Cows are therefore standing in the foam whilst waiting to enter the parlour, foam is carried into the parlour on their feet and remains on their feet during the milking process. The majority of cows therefore have their feet bathed in foam for some 5-10 minutes.

Trials have suggested that the foam is effective as a preventive, but the disinfectant seems to have a limited effect as a treatment of existing lesions. The system is easy to use and the chemicals are pleasant to handle, with no adverse environmental effects, but by its very definition foam is a liquid with holes in it, hence its ability to penetrate the interdigital space must be less than when using a liquid bath.

A variety of chemicals are used for foot bathing, each with their own advantages and disadvantages. Formalin is cheap and rapidly degraded in the environment, but is unpleasant to handle. Copper sulphate is more pleasant to handle but is not degraded in the environment, and copper poisoning is becoming an increasing problem in dairy cows. Ideally it therefore should not be simply discarded into the farm drainage system. Zinc sulphate, organic acids and disinfectants such as gluteraldehyde and peracetic acid are also effective. It is the method of administration, the frequency of foot bathing and the cleanliness of the environment that is more important than the chemicals used. Antibiotic foot baths are permissible in some countries, but as with mastitis, these should be considered as a treatment rather than a control measure. It is

this author's belief that within a few years disinfectant foot bathing will be almost as common as post milking teat disinfection.

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