



## **Development of claw horn lesions- How do they start and where do they end up?**

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Lameness is the most important cause of poor welfare in dairy cattle; in addition, along with mastitis and poor fertility, it is one of the three most economically important diseases in dairy cattle.

In dairy cattle ~90% of lameness arises in the hoof. Lameness in the hoof can be divided into two categories – i) infectious lameness (principally foot rot (interdigital necrobacillosis) and digital dermatitis and ii) non-infectious lameness; i.e. claw-horn diseases such as white line disease and sole ulcer. Treatment of infectious diseases is simple and generally effective, although control, particularly of digital dermatitis, can be difficult to achieve. In contrast treatment of claw horn disease is palliative at best, focussed as it is on removing the damaged horn and reducing weight bearing on the affected site. Treatment does not restore normal hoof anatomy or repair the damage done to the horn-producing tissue. Therefore cows which have been treated for claw-horn disease remain at significantly increased risk of recurrence of such disease in both the affected foot and in the contralateral limb. A further difference between infectious lameness and claw-horn disease is the level of pain and discomfort; this is much greater and more prolonged in animals with claw-horn disease than animals with foot rot or digital dermatitis. Additionally, the pain and discomfort associated with

claw-horn disease begins significantly before lameness is detected, in contrast for cattle affected by infectious disease there is a very short interval between the onset of significant pain and the onset of clinical disease.

To understand why this is the case we need to know how claw horn lesions develop and the subsequent effects of those lesions on the hoof and the subsequent risk of claw-horn disease.

### **Starting at the beginning**

The most important risk factor for claw-horn disease is calving. In a landmark series of papers the University of Bristol group showed that the changes in ligaments which were an essential part of the preparation also had significant effects on the connective tissue of the hoof suspensory apparatus. In heifers, Tarlton et al (2002) demonstrated that biochemical and histological changes occurred within the suspensory apparatus of the hooves around the time of first calving, and it was likely that these changes were mediated by metalloproteinases (MMP), whose function is to degrade collagen. The Bristol group linked their findings with those of Lischer et al (2002) who showed that increased laxity of the connective tissue of the suspensory apparatus of the distal phalanx was a consistent finding prior to the development of sole ulcers. They hypothesised that the changes they



found in association with parturition were therefore the start or trigger for the development of claw-horn disease.

In a subsequent study by the Bristol group (Knott et al 2007); they confirmed the biochemical and histological changes they had seen in the earlier study and also showed that housing heifers in cubicles rather than straw yards also impaired the biomechanical resilience of the hoof. The effects of housing and parturition were additive, so the changes seen in parturient heifers kept in cubicles were greater than the changes seen in non-pregnant heifers kept in cubicles. The

hypothesis, therefore, is that the initial changes seen in the development of claw horn disease are due to a combination of the effects of parturition with other stresses, especially housing, and that it requires both stressors to be present for the initial corium damage to occur. This was supported by the sole haemorrhage data reported by Knott et al (2007), which showed that significant sole haemorrhages were only seen in heifers that had calved, but also that haemorrhages in cubicle housed lactating heifers were significantly worse than those on lactating heifers housed in straw yards (see Table 1).

Table 5  
Effects of housing, diet and parturition on histology of the laminae

House	Group	Width	Angle	Irregularity
Yard	YLP	18.11	1.44	3.08
	YMP	15.73	1.08	2.69
	YMF	14.89	1.22	2.55
Cubicles	CLP	14.75	2.55	1.91
	CMP	14.65	2.06	2.00
	CMF	15.23	1.68	2.08
Significance	House	0.027	<0.0001	<0.0001
	Diet	<i>0.79</i>	<i>0.74</i>	<i>0.96</i>
	Lactation	<i>0.17</i>	0.006	0.37

Non-significant *P* values are indicated in italics; for explanation of units see text.

Table 1: From Knott et al (2007)

This hypothesis is further supported by data from a longitudinal study where heifers were reared on cubicles from mating until calving and then transferred to another cubicle yard (Laven and Livesey 2002). Significant hoof horn haemorrhages were not seen in those heifers during the rearing period but after calving there was a significant increase in haemorrhages even though the heifers had spent more

than 9 months in the cubicle yard before calving.

**What is the initiating factor?**

It is the laxity of the supporting tissues which produces the damage to the corium which then is seen as haemorrhages, and, if the damage, is severe enough white line disease, sole ulcer and other claw-horn diseases.



This hypothesis was described by Christoph Lischer as the ‘tourist in a hammock’. As illustrated in Fig. 1, movement of the distal phalanx can

lead to the damage of the corium (the horn-producing tissue), either the corium of the white line or the corium of the sole (or both).

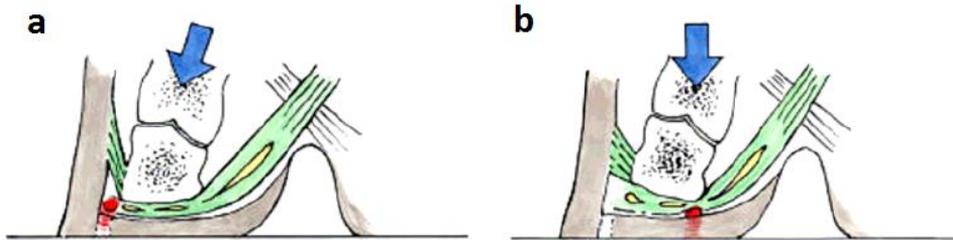


Fig 1: Diagrammatic illustration of how laxity in the supporting structures of the distal phalanx can produce damage to the corium and subsequently haemorrhages which become apparent on the palmar/plantar surface of the hoof (a – white line; b- sole) (from Lischer and Ossent 2002).

The analogy with a hammock illustrates clearly how calving and other stressors, such as housing interact to produce significant corium damage (see Fig. 2), with the wind

blowing through the palm trees being analogous to the concussive forces which occur when cows are housed on concrete.

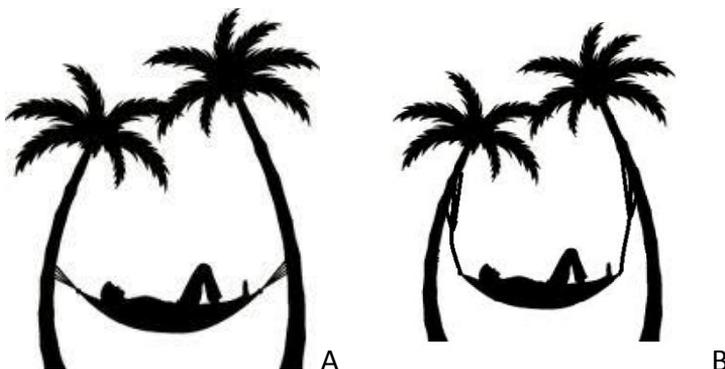


Fig. 2. In a) the hammock is tightly attached to the palm trees, while in b) it is loosely attached. Provided there is no external force, the main effect will be some amplification of the movement of the tourist’s small movements. However if there is an external force, such as a moderate wind, then the tourist in a) will experience some limited movement, whereas the tourist in b) will experience more movement which may be amplified by the movement of the loose attachments.

So the data on the impact of parturition strongly suggest that, particularly in heifers, the initial claw-horn disease lesions develop in the immediate post-partum period and that management of

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heifers during this period is crucial (e.g. Webster (2001) showed that housing heifers in straw yards for 8 weeks after calving and then moving them to a cubicle yard prevented most



of the post-parturient rise in hoof horn haemorrhages seen in heifers housed in cubicle yards after calving.

### **Does body condition score loss lead to lameness or does lameness lead to body condition score loss?**

It has long been known that lame cows lose bodyweight and body condition, but it is now clear that body condition score loss in non-lame cows is a significant risk factor for lameness. Both Randall et al (2015) and Lim et al (2015) showed that cows with a BCS <2.5 (5-point scale) had a significantly increased risk of becoming lame compared to cows with a BCS  $\geq 2.5$ . Lim et al (2015) were also able to show that loss of BCS also increased of developing lameness, and in addition that change in BCS was also associated with the chances of

recovery from lameness with BCS loss having a detrimental effect and BCS gain (in thin cows) having a beneficial one. There is thus a complex bidirectional relationship between BCS, BCS change and lameness.

These findings bring in to prominence the role of the digital cushion. The digital cushion consists of three parallel fat cylinders; axial, abaxial and central (Fig. 3). The axial and abaxial cushions are connected (in front of the flexor tuberosity of the distal phalanx) by multiple, transverse finger-shaped cushions. The flexor tuberosity itself is covered by the central fat pad. The main role of the digital cushion is to act as a dispersant across the pedal bone of the shock from placing the hoof onto the ground, but it also allows there to be significant movement of the pedal bone relative to the horn capsule.

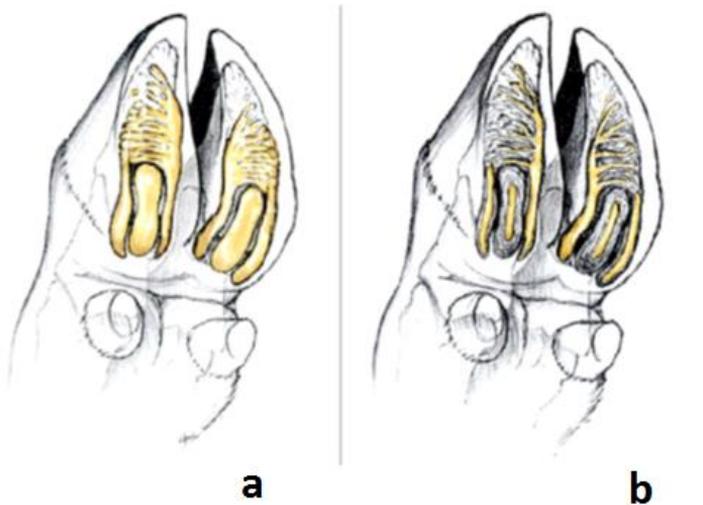


Fig. 3: Diagrammatic representation of the digital cushion. In a) the three parallel cushions can be clearly seen (normal situation); in b) the amount of fat in the digital cushions has been markedly reduced and it has been replaced by connective tissue (thin cow or previously lame cow). (From Lischer and Ossent 2002).

AS is illustrated in Fig.3, loss in body condition (either before or after

lameness) results in a reduction in the amount of fat in the digital cushion



(Bicalho et al 2009) and a consequent reduction in its shock dispersant qualities. It thus seems that a well-developed digital cushion plays a major role in reducing the risk of lameness and in increasing the rate and chances of recovery in lame cows.

Lameness may have impact on the digital cushion in at least two ways; firstly, there is the simple link between lameness and reduced feed intake. If this leads to increased fat mobilisation, then it is likely that some of that fat will be fat in the digital cushion. In addition, the digital cushions may act as a local reservoir for arachidonic acid (the precursor to inflammatory prostaglandins), and this arachidonic acid may be released when there is tissue damage and compression which is sufficient to cause local inflammation. This may either reduce the amount of fat in the cushions or alter the cushions shock dispersing qualities.

It is thus clear that monitoring and management of BCS is crucial for both lame cows and non-lame ones if the prevalence of lameness is to be reduced. We need to minimise BCS loss in lame cows (which will speed recovery and, possibly, reduce recurrence) and need to optimise BCS (and minimise BCS loss) in non-lame cows (which will reduce the risk of lameness developing in those cows).

It also clear that the bidirectional association between BCS and lameness leads to a self-perpetuating negative cycle where an animal which is lame loses BCS and therefore is at increased risk of subsequent lameness.

### **Is lameness treatment successful?**

We can clearly treat lame cows if the criteria for success are reduction in pain and apparent return to normal (or near-normal) locomotion. However as the studies on BCS show, lame cows, even when effectively treated by a veterinarian, still have a markedly increased risk of becoming lame again. So in terms of returning a cow to normality, lameness treatment is not successful.

It is likely that much of this is due to the delay in treatment, with cows having significant claw-horn disease for prolonged periods (>1 month) prior to being recognised as lame and treated. There is an increasing body evidence that early recognition and treatment of lameness (even if it is only moderate) can improve treatment success rates and reduce lameness prevalence (by reducing lameness recurrence) (Leach et al 2012; Thomas et al 2015).

It is likely that much of the benefit of early treatment is mediated by reducing the damage to the lame foot; however this may not be the only benefit. Thomas et al (2016) showed that treatment success (defined as a cow being non-lame 6 weeks after treatment) was markedly reduced when the delay in treating moderate lameness was only two weeks. They found that the main difference was that in the study where treatment was delayed a much higher proportion of cows were lame on the opposite limb 42 days after treatment than had been the case where treatment was instigated as soon as an elevated locomotion score had been observed. Interestingly, whereas adding a block



and an NSAID to trimming in the prompt study had increased treatment success rate, this was not the case in the delayed treatment study

This suggest that even in mild-moderate cases a short delay can have a significant impact in both the affected foot and in the opposite one. The delays built in to the study reported by Thomas et al (2016) were far shorter than is normally seen on farm, and the cattle were therefore still treated earlier than would have been the case if treatment had been farmer rather than research led.

We need more data on what is actually happening in the feet of cows during the early stage of claw-horn disease , but it is clear from recent research that we need to be focussing on treating cows earlier than is currently the case; i.e. when they are locomotion score 2 (on a 0-3 scale) rather than a score 3 cow.

### **What about the pedal bone.**

Claw-horn disease is an inflammatory process once the damage has become severe enough. There has been very little investigation of the impact of that inflammatory process on the structures of the hoof, particularly in cows with repeated bouts of moderate lameness. However, recent research looking at the pedal bone of cull cows strongly suggests that lameness is linked to the development of bony exostoses on the pedal bone, particularly in the region of the flexor tuberosity.

Newsome et al (2016) showed that lameness history and age were both associated with these exostoses. Such exostoses are also common in cows which have undergone digit

amputation, although there is a huge variation between cows with some claws having almost no exostoses and others having almost complete destruction of the joint surface (Laven unpublished observations). The study by Newsome et al (2016) was a post hoc analysis of bony change; as such it was never going to be a definitive study of the link between bony change and claw-horn disease. The main issue with the data is that effect of age on the development of exostoses is greater than the effect of lameness; however, it is quite feasible that the apparent age effect is mediated through multiple bouts of mild/moderate lameness which may have gone untreated. In New Zealand, lameness is significantly less common than in the UK (20 cases / 100 cows per year vs over 50 cases/ 100 cows per year), and preliminary studies suggest that bony exostoses are not common in cull cows (5 years +). Further research is needed.

If this hypothesis is correct then, bony change may be a key part of the self-perpetuating cycle of continuing lameness and could be a significant reason why lame cows go lame again. In addition, if as seems likely bony change is slow and therefore is more likely when lameness is prolonged and more severe than early treatment of lameness is likely to have long-term impacts as it reduces the risk of bony change. So the findings by Newsome et al (2016), if correct, add significantly to the push towards early diagnosis and treatment. If bony change is an important part of the pathogenesis of claw-horn disease then the findings of Newsome et al (2016) would have a major impact on our understanding of lameness prevention and treatment, which may be as



important as the impact of the research on parturition and lameness and the impact of the research on how poor body condition score produces lameness.

## Conclusions

Our understanding of the development of lameness has increased significantly in recent years and it has become clear how much of a self-perpetuating process lameness is.

The key driver in dairy cattle is the direct effects of parturition which when combined with other lameness risk factors lead to the development of claw horn lesions. The key impact of parturition is to increase the likelihood of inappropriate forces on the corium and it acts together with other factors that can produce the same effect (such as poor environment and overgrown hooves) to turn minor damage into damage that is detectable at the claw surface (as haemorrhages). If the damage to the corium is severe enough then these haemorrhages develop into clinical claw-horn disease. BCS is important as it determines the ability of the digital cushion to absorb and disperse the shocks associated with standing and movement, particularly

on hard surfaces. Low and reducing BCS thus increase the risk of factors such as poor environment producing corium damage.

The link to BCS is the first self-perpetuating negative cycle as lame cows lose BCS which makes them more prone to become lame and then more prone to lose BCS. The second self-perpetuating negative cycle is the development of bony exostoses as the result of chronic inflammation associated with lameness. These exostoses, even when small, significantly increase the risk of inappropriate forces on the epithelium of the corium, increasing the risk of more lameness and thus increased development of bony exostoses.

These negative cycles mean that we have to be much more proactive not only in preventing lameness but also treating it. Farmers across the world need to get the message that if they are only treating the lame cows that they see then they are treating cows too late. Cows should be treated as soon as they are detectably lame; this will only happen if farmers are actively looking (at least once a week for lame cows) and then treating those cows within 24 hours.



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