THE ROLE OF BODY CONDITION IN LAMENESS CONTROL

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SUMMARY

The aetiopathogenesis of the claw horn lesions (sole haemorrhage, sole ulcer and white line disease) remains relatively poorly understood. Recent changes to our understanding of the structure and function of the claw suggest that the digital cushion, a pad of connective tissue and fat under the distal phalanx, may play a key role in protecting the foot against the development of claw horn lesion. Previous work has demonstrated that the thickness of the digital cushion is related to body condition i.e. it appears that fat is mobilized from the digital cushion during period of weight loss and visa versa. Three recently published studies from the UK all demonstrate that loss of body condition score precedes cows being identified or treated for lameness, suggesting that managing the rate and extent of body condition score loss may be a key lameness prevention strategy.

INTRODUCTION

The aetiopathogenesis of the claw horn lesions (principally sole haemorrhage, sole ulcer and white line disease) remains relatively poorly understood. Early descriptions of claw horn lesions in cattle suggested an aetiopathogenesis similar to that described for 'laminitis' in horses. In cattle, rumen acidosis (associated with the overfeeding of concentrates) was suggested to lead to inflammation and degradation of the laminae and eventually increased movement and sinkage of the distal phalanx within the hoof capsule. Movement of the distal phalanx led to compression of the dermis and the formation of the claw horn lesions. Whilst in cross sectional studies the claw horn lesions have been associated with high concentrate diets, causality cannot be attributed using this study design. At the same time, this theory has proved difficult to induce experimentally.

Whilst overall the 'laminitis' theory has yet to be completely disproven, other experimentally evidence has suggested an alternative aetiopathogenesis. Firstly Tarlton et al (2002) demonstrated a loss of supporting strength within the laminae around parturition i.e. the distal phalanx was more mobile during this period. Secondly the importance of the digital cushion, a support structure of connective tissue and fat under the distal phalanx, is increasingly being recognised. The digital cushion is a series of three parallel masses which run longitudinally beneath the distal phalanx and is thought to protect the more sensitive foot structures during foot strike and limb loading. The content of the cushion changes with age, starting with loose connective tissue in heifers before filling with fat in parities two and three (Räber et al 2004, Räber et al 2006). Finally a range of anatomical and biomechanical factors particularly related to the relative shape and size of the bony architecture in the distal limb, have been identified which contribute to / exacerbate other aspects of the aetipathogenesis (Nuss, 2014). Whatever the initial cause of overload in the hoof capsule, the claw horn lesions result from damage to the tissues responsible for horn production, succinctly described by Nuss (2014) for sole ulcers: 'Continuous displacement leads to compression of the sole corium, which in turn initiates the cascade of vascular compromise, ischemia caused by congestion, oedema and thrombosis, interrupted keratogenesis and finally sole ulcer'.

Interest in the digital cushion as a potentially important aspect of the aetiopathogenesis of claw horn lesions has increased since it was demonstrated that the thickness of the digital cushion was positively associated with body condition score at the time of
examination i.e. thinner animals had thinner cushion and visa versa (Bicalho 2009). In the same study the prevalence of claw horn lesions was associated with the thickness of the cushion i.e. animals with thin cushions had a greater number of lesions. The work implied that thin cows had mobilised fat from the digital cushion during weight loss, resulting in compromised claw function and the development of claw horn lesions. However, as this study was cross sectional in nature, no direction to the relationship could be attributed i.e. alternatively lame cows with claw horn lesions could have lost body condition, resulting in thin digital cushions.

This work and work that has followed has attempted to address the question, ‘Do lame cows become thin, or thin cows become lame’. Undoubtedly lame cows become thin, a number of previous studies have demonstrated that lameness has a range of negative effects on feeding leading to lose of body condition (reviewed by Huxley 2013), however if thin cows become lame, body condition score management could be an important lameness prevention strategy.

RECENT UK STUDIES

Three separate UK field studies conducted by the author and colleagues have recently investigated the temporal relationship between body condition score change and lameness.

**Study One (Green and others 2014)**

This longitudinal study was conducted on data collected from a single, 600 cow UK herd over a 44 month period. Lesions diagnosed when animals were identified and treated for lameness was recorded. All animals were assessed for BCS at approximately 60 days intervals throughout the study period. Mixed effect binomial logistic regression models were used to investigate the association between BCS and treatment for lameness.

A BCS <2.5 was associated with an increased risk of being treated for lameness caused by sole haemorrhage, sole ulcer and white line disease in the following 0-2 and for sole ulcer and white line disease in the following 2-4 months i.e. a low body condition score preceded lameness treatment by a number of months.

**Study Two (Lim and others 2015)**

The second paper describes a longitudinal study conducted across four UK herds over an 18 month period. Animals were condition and mobility scored every 13-15 days by a single observer. In total 6889 observation from 731 cows were analysed in a multilevel multistate discrete time event history model to investigate the transition of lameness (assessed by mobility score) over time.

Animals with a low BCS at calving ≤2.25 had a higher probability of becoming lame, and if they were already lame, they were less likely to recover. Similarly, when the BCS at the current visit was compared to the BCS at calving, cows which had lost condition had a higher probability of becoming lame, and if they were already lame, they were less likely to recover. Interestingly the converse effect was also identified, an increase in BCS from calving was associated with a lower probability of becoming lame, and if they were already lame, they were more likely to recover.

**Study Three (Randall and others 2015)**

The final study was conducted on a very rich dataset available from the SRUC Royal Crichton research dairy herd. Animals were condition and mobility scored every week.
Nearly 80,000 observations from 724 cows over an eight year period were available for analysis in mixed effect multinomial logistic regression models.

Low BCS three weeks prior to a repeat lameness event (i.e. not the animals first ever lameness event) was associated with a significantly increased risk of lameness. A BCS <2 was the greatest risk, BCS >2 led to a reducing risk. Animals with a low BCS 16 or 8 weeks prior to a first lifetime lameness event (i.e. the first time an animal was ever identified as lame) were at greater risk of lameness but only if their first lifetime lameness event occurred when they were in 2nd lactation or greater. Finally animals which lost body condition in the four weeks after calving were at greater risk of a future lameness event.

**DISCUSSION**

The studies described above have highlighted the importance of body condition score management as a tool for lameness control. Importantly, all three studies identified that change of body condition score preceded animals either being identified lame by mobility scoring or being treated for lameness, using robust multivariate statistical techniques. They suggest that managing body condition score at a herd level may lead to a reduction in the overall risk of lameness caused by the claw horn lesions. Further prospective studies are needed to test the impact of herd level BCS management on lameness to demonstrate that this is an effective and practical control strategy for use on farm.

**REFERENCES**


