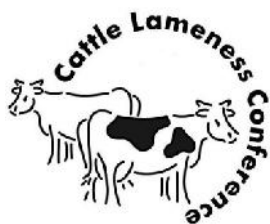


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




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# CATTLE LAMENESS CONFERENCE

Topics are:

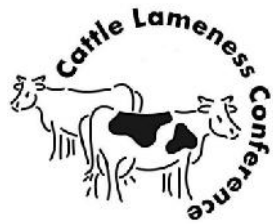
-  Practice based lameness projects
-  Pathogenesis of claw disease
-  Review of lameness research
-  Digital dermatitis monitoring
-  Digit amputation

Wednesday 2<sup>nd</sup> May 2012

Pitch View Suite  
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# GENERAL INFORMATION



## TIMETABLE of EVENTS

08:45	ARRIVE / REGISTRATION / COFFEE and POSTER DISPLAY	
09:25	CHAIRPERSON'S INTRODUCTION Nick Bell, Royal Veterinary College	
09:35	35 years of practice-based lameness projects; and what will the next 20 bring?	Roger Blowey Wood Veterinary Group
10.15	Questions and Discussion	
10:30	COFFEE and POSTERS	
Session 2 Chairperson: Jon Huxley, University of Nottingham		
INTRODUCTION TO SESSION 2		
11:00	Functional anatomy of the bovine foot – failure of key structures in pathogenesis of claw disease	Christoph Mülling University of Leipzig
11.40	Questions and Discussion	
11:50	Research review into the lame cow	Colin Mason Scottish Agricultural College
12:30	Questions and Discussion	
12:40	LUNCH and POSTERS	
Session 3 Chairperson: Brian Pocknee, The Dairy Group		
14.00	INTRODUCTION TO SESSION 3 and VOTING ON POSTERS	
14:10	Digital dermatitis: What is unsought will go undetected	Jessica Stokes Soil Association
14.50	Questions and Discussion	
15.00	Digit amputation – getting it right for a productive future	Sara Pedersen Nantwich Veterinary Group
15.40	Questions and Discussion	
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16.00	TEA and DEPART	

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<sup>1</sup>Agri-Food and Biosciences Institute, Hillsborough, Northern Ireland;

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<sup>1</sup>Agri-Food and Biosciences Institute (AFBI), Agriculture Branch, Hillsborough, Co Down, Northern Ireland; <sup>2</sup>Faculty of Veterinary Medicine, University of Glasgow, Glasgow, United Kingdom; <sup>3</sup>School of Biological Sciences, Medical Biology Centre, Queens University Belfast, Belfast, Northern Ireland

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Roger Blowey

Wood Veterinary Group, 125 Bristol Road, Quedgeley, Gloucester, GL2 4NB, UK

## CHAIRPERSON'S INTRODUCTION

Welcome to the 4<sup>th</sup> Cattle Lameness Conference at the new venue, Worcester Rugby Club. We are very grateful to Nottingham University for providing us with excellent facilities for the first three conferences but for logistical reasons we have needed to move for at least this year. An added benefit of this move is that it should make the conference more accessible to the West of the country where most cattle (and I suspect cattle advisors) are found.

This year has seen a number of important practical developments. DairyCo officially launched the Healthy Feet Programme which, so far, has been well supported and exceeded enrolment targets. Furthermore, the DairyCo lameness research programme was started and we look forward to seeing the results coming through in the next couple of years. One of the most exciting pieces of political news was from the National Association of Cattle Foot Trimmers who are adopting a mentorship scheme for newly qualified trimmers and simplifying their "category system". The category system has allowed farmers and advisors to identify the most qualified and up-to-date foot trimmers, although it may have caused confusion for some. As a result, at the recent AGM, the foot trimmers elected for a simpler "licensed" and "unlicensed" scheme which will be self-regulated.

Scientifically we have not witnessed any major breakthroughs but a steady building on current understanding. The pathogenesis of sole ulcers and "laminitis" still remains a topic of intense debate, but a number of researchers have now shown us various pieces of the jigsaw. Perhaps most enlightening has been the work on the anatomical changes associated with sole ulcers and so we are delighted to welcome Dr Christoph Mulling who is an outstanding speaker with international expertise in this area.

Also presenting at this conference is Mr Colin Mason, representing the Scottish Agricultural College, Dr Jess Stokes who has just completed her PhD on the dynamics of digital dermatitis infections under various control strategies, and Mrs Sara Pederson and Mr Roger Blowey, both vet practitioners with strong interests in lameness research.

As always we would like to thank all our sponsors. Without the support of sponsors this conference would not be possible.

Next year is the International ruminant lameness conference and symposium which will be in the UK hosted by the University of Bristol (dates to be announced). This biennial event should be of great interest to you all as it always attracts the best researchers in this field from around the world. We hope to see you there.



Nick J. Bell  
Chairperson of the Cattle Lameness Conference, Royal Veterinary College  
On behalf of the CLC Organising Committee



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# PAPERS



## 35 YEARS OF PRACTICE-BASED LAMENESS PROJECTS; AND WHAT WILL THE NEXT 20 BRING?

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### SUMMARY

The paper describes lameness projects that have stimulated my interest over the past 35 years. These include the reporting of digital dermatitis, describing a link between biotin and white line lesions, the EU LAMECOW project, rib fractures in adult cows and more recently the increasing incidence of non-healing hoof lesions. I am grateful for the generous help and support of many highly competent researchers. I speculate on the challenges and possible advances in our understanding of lameness in the future.

### INTRODUCTION

My first recollection of dealing with lame cows was on the family farm at home in Devon. My father milked 20 South Devon cows and with a few pigs and sheep this was enough to bring up a family of 6 children. How things have changed! As a child I remember one chronically lame cow which we poulticed regularly with a hot mix of bread and bran, placed in a hessian sack and tied on for 24 hours. At that stage one of the treatments for foul in the foot was to take a length of rope, cover it thickly with Stockholm tar and pull the rope between the claws so that the Stockholm tar remained within the interdigital space. I have no idea if either of these treatments worked, and I certainly did not commit them to paper at the time, but they do remain etched in my memory.

My next assessment of the importance of lameness would have been in the first edition of *A Veterinary Book for Dairy Farmers* (2). The commissioning of this book took place in December 1982, with the first edition being published three years later, in 1985. For that first edition especially, I put considerable effort into reading around a wide range of subjects; it was a challenging time, assessing whether what I had done for the previous 15 years in general practice was in fact acceptable. This certainly applied to lameness and looking back on the lameness section as I write this I am relieved to see that at least a good deal of what I believe today remains written there! Of course it was before the days of Digital Dermatitis and its associated problems.

### Digital Dermatitis

One of my first published papers on lameness related to digital dermatitis. The condition was seen in a dairy herd just north of Gloucester. At that stage I had not started attending meetings of the International Ruminant Digit Symposium. I had no idea what was causing these 'open sores' on the feet and as a consequence tried a range of treatments, most of them injectable antibiotics. David Weaver suggested that it might be Digital Dermatitis and this was then confirmed in association with Mike Sharpe at the VLA and published in the *Veterinary Record* in 1988 (3). That started my initial interest in Digital Dermatitis and it has certainly led to a challenging career generally in the field of lameness.

In 1989 I was asked to present papers on Infectious Skin Disorders at the International Ruminant Digit Symposium in Liverpool, often referred to as the "Footy Fetish Club". This was the start of my association with a group that has lasted for over 20 years and taken me down many interesting routes. The group has been a great inspiration to me and I would like to thank the members for their continued enthusiasm.

While scanning the literature for references about the use of antibiotics for the treatment of treponemes I read that the lesions of Lyme disease in man, caused by the organism *Borrelia burgdorferi* were very similar to those of Digital Dermatitis. Prof Stuart Carter at the University of Liverpool, was researching Lyme Disease in dogs so I wrote and asked if he would be prepared to do bovine serology for me. This was an exciting step forward, because we were able to show that animals with Digital Dermatitis were sero-positive to *Borrelia burgdorferi* and in animals/herds where the condition had not been reported the animals were sero-negative (16). Serology was then used as a marker of the onset of infection and work in associated with Richard Laven and Bridget Drew at Bridget's EHF showed that calves as young as three months old became sero-positive, although disease was not seen until a considerable period later.

At this stage DD was becoming an increasing problem in North America, and I was able to obtain a licence that allowed me to send biopsies of Digital Dermatitis lesions to Richard Walker and Derek Read at the University of Davis, California. They confirmed that the organisms causing DD in the UK were identical to those causing DD lesions in the USA. This was the beginning of our realisation that a worldwide syndrome was developing.

It later transpired that the *Borrelia burgdorferi* was not in fact the cause of Digital Dermatitis, but other related Treponemes, and these were identified by Nick Evans, Ibrahim Demarkan and others in Stuart Carter's group as *Treponema denticula*, *Treponema media* and *Treponema phagedenis* like organisms (15 +17)

### Deep Pedal Curettage

At the farm where DD was identified there was an increasing number of cows with infected sole ulcers that failed to heal. I can remember one morning being presented with five extremely lame cows, all sole ulcers with a deep pedal infection, plus at least another 20 to trim. Almost in a fit of exasperation, I realised that what we now refer to as retro-articular abscesses could not possibly heal unless they were drained adequately. It was on that morning that I attempted the technique of what is now known as deep pedal curettage or "coring". The objective was to allow drainage of the retro-articular space. Provided the sinus was flushed regularly and the cow kept on a parenteral antibiotic for a week to 10 days, it was surprising how many healed. I kept a record of all the cases I treated and found an approximate 80% success rate, success being defined as a return to reasonable locomotion and production (4). Of course the technique involved severing the deep digital flexor tendon and as a consequence many recovered cows walked with a dorsal rotation of the toe.

Effective treatment has always been important for vets in practice. A student project with Cath Thomas involving about 100 cows showed that the length of time that a Cowslip remained on the cow's foot was an average of 74 days (8). If hoof grows at 5mm per month and the average sole is 10mm thick, then it should take two months, or 60 days, for a sole ulcer to repair.

### Biotin and Lameness

While the DD work was proceeding I was approached by Adrian Packington of Roche Pharmaceuticals (now DSM Ltd.) and asked about the possibility of developing an intervention study to evaluate the effects of biotin on lameness in dairy cattle. We roughly outlined our project then looked for a suitable statistician to ensure our results would be valid, and a researcher to assist with the legwork. This was the beginnings of a very fruitful relationship with Laura Green at the University of Warwick and Ginny Hedges in whose PhD the effect of biotin was demonstrated. The study involved five herds in a split herd intervention trial (a total of 1,109 cow years on trial), where every

lame cow was examined, recorded and photographed by a vet. We were able to show that the addition of 20mgs of biotin per cow per day halved the incidence of white line disease overall and in older animals, where white line disease is more prevalent, it decreased the incidence by almost a factor of four (20). Cows being supplemented with biotin also had a much reduced recurrence rate, suggesting that biotin improved the rate of healing of white line lesions. This project especially showed me the advantages of professional help, ie from Laura Green and Ginny Hedges. We got so much more data from the project through their involvement.

From the biotin project we were able to demonstrate that the four main lesions causing lameness at that time were digital dermatitis, sole ulcers and white line disease, all occurring at an incidence of around 12 cases per 100 cows per year, and foul of the foot that occurred at an incidence of 8 cases per 100 cows/year. The project also demonstrated that there was a marked increase in lameness in early lactation and particularly the peak in sole ulcers and white line disease some 2-3 months after calving. I remember discussing the effects of calving with John Webster at a BBSRC meeting, and I would like to think that it partly led to his research project and the later identification of the mechanism of 'hoofase'. This is the mechanism whereby a relaxation of the pedal bone suspension leads to an increase in the animal's susceptibility to lameness from two weeks before to two weeks after calving.

Another interesting offshoot of the biotin project was the distinction made between a sole ulcer developing beneath the flexor tuberosity of the pedal bone and a heel ulcer which is thought to develop as a result of a cartilaginous change in the central fat pad of the digital suspensory ligament (7).

#### EU LamCow Project

The EU Lam Cow Project was a multi-national, multi-disciplinary research investigating a wide range of parameters associated with lameness, ranging from the micro-vasculature of the cow's foot to risk factors on farm. My involvement with the project was initially to train the 50 participating farms to identify lesions and thereby record accurately. The group of 50 farms was then split into a treatment and a control group, with each intervention farm being visited and advice given on factors that might reduce the risk of lameness (1). At this stage the major lesions causing lameness had been identified by two years of data recording. The objective was to compare the incidence of lameness on those farms given advice, compared with those farms that did not receive advice. Results showed a small effect only (in press). Part of the problem was that the remaining period of study following the intervention advice was relatively short, so that any farm given advice, for example to reduce the level of sole ulcers, would not see a major effect until all existing cows with sole ulcers had been removed from the herd, either as a result of culling or simply on an age basis.

#### Milk Yield and Lameness

In both the EU Lam Cow Project and the biotin project the influence of lameness on milk yield was studied and perhaps surprisingly, values were very similar. Cows that become lame appear to be higher yielding than cows that do not become lame, and the average lame cow lost approximately 400 litres of milk (19). For the EU Lam Cow Project it was shown that cows with a sole ulcer lost 570 litres and cows with white line disease 370 litres. The interesting fact is that the drop in yield occurs some while (up to four months prior to clinical signs of lameness being seen). This fits our known pathogenesis of hoof lesions.

## Rib Swellings

During the course of an insurance investigation, the complainant pointed out to me that lame cows had swellings on their ribs. The investigation of the incidence of palpable rib swellings in a range of other farms was the basis of a PhD by Emma Boyling under the guidance of Nick Bell. She was able to show that the average incidence of palpable rib swellings in the dairy herds studied was around 15% and that these palpable rib swellings were associated with age of cow and with level of lameness (11). It is my own opinion that rib swellings represent a significant welfare issue.

## Non Healing Hoof Lesions

Around 2005 a previously unreported lesion started to appear, a condition that we now know as 'toe necrosis', or 'apical necrosis of the pedal bone' (10). It was suggested that this was more prevalent in herds where DD was not well controlled. Soon after, further non healing hoof lesions were reported from the UK, the Netherlands and USA. Biopsies were taken from a range of these lesions, and in a high proportion of cases the 3 organisms involved with DD were identified using PCR (18). Similar changes have now been seen in other countries, including a huge outbreak in grazing herds in Chile (14)

## THE FUTURE

Those who predict the future must do so with some trepidation, because one thing is certain – not all predictions will be correct! I have chosen to take the easy option and at least start this section with investigational work that is on-going, part of it in my garage at home!

## Pedal Bone Changes

For many years I have been interested in the effects of hoof lesions on the pedal bone. It is well recognised that the corium produces the hoof, and any disruption of the corium leads to poor hoof formation, with subsequent sole ulcers and white line disease. The corium also feeds and remodels the bone and hence an inflamed and/or disrupted corium can lead to abnormalities of the pedal bone.

As semi-retirement loomed I decided to study the effects of these bone changes in more detail. Although I had prepared sporadic specimens in the past, from February 2011 I began to collect, boil, preserve and examine pedal bones from the digits that I had amputated during my work in general practice. The first thing to become apparent was that there had been a big change in the reasons for amputation. Whilst the textbooks recorded infected coronal pedal joint as a major indication, it became apparent that by 2010/2011 non-healing hoof lesions were the major reason for amputation (12). Previous work had shown that non-healing lesions were associated with digital dermatitis infection.

Further studies then demonstrated the importance of changes within the pedal bone on the development of hoof lesions (13). It is hypothesised that disruption of the corium leads to a proliferative osteitis. This results in the formation of small, sharp exostoses, like stalactites, that penetrate the corium from above, and that this could be a further reason for non-healing lesions. Further work is needed to determine the time of onset of these lesions, and whether we can find any means of preventing what are at present irreversible changes.



## Development of Sole Ulcers

As part of the pedal exostoses project described above, pedal bones were processed and examined from 20 random cows at slaughter. It was found that even in cattle with intact hooves, ie in the absence of non-healing lesions, exostoses develop beneath the flexor tuberosity of the pedal bone. This must significantly predispose to sole ulcers. It is hypothesised (but not yet proven) that in the early stages of the formation of sole ulcers, if the feet become bruised then the bruising disrupts horn formation in the sole, but the bruising also stimulates overgrowth of horn on the flexor tuberosity, further predisposing to sole ulcers. This further emphasises the work from Nick Bell that has demonstrated the importance of early treatment of lame cows, and the importance of early block application to the sound claw. In addition, it is the opinion of this author that a shoe that supports the weight of the cows on the wall of the hoof rather than the sole, will be beneficial in terms of the prevention of pedal bone lesions. This needs to be studied in more detail.

## Genomics of Digital Dermatitis and other causes of lameness

Farmers have long recognised that in any herd there are some cows that never get DD whilst other cows never get rid of the lesions, however frequently they are foot bathed. In a joint study with Rachel Scholey, three herds were scored for DD on a monthly basis for 6 months. Samples of DNA were analysed and it was found that much of the difference between 'DD' and 'non DD' cows could be attributed to SNP's (single nucleotide polymorphisms), especially on chromosome 26 (21). In practical terms, farms should identify such cows and AI them to beef. Further studies are required to determine the genomics of sole ulcers, white line defects and non-healing hoof lesions.

## Foot Bathing

There are already a plethora of studies on foot bathing, but it surprises me that we remain unsure as to whether two baths in series are required, whether this increases the level of soiling in the second bath, and what the optimal frequency of foot bathing is. As the incidence of non-healing lesions increases I would predict that daily or twice daily foot bathing, including transition cows and perhaps all dry cows, will become as common as post milking teat dipping.

## Housing, Handling and Environment

I predict that the trend in increasing herd size and rising yields will continue for the majority of herds, carrying with it an increased risk of lameness. Housing for the future must allow much more space, more cow comfort, improved handling facilities, better air flow and maintain a much drier environment.

In the large herds especially, there must also be a better understanding of human animal interactions. Calm quiet handling is essential to reduce disease, and I predict that our understanding of this will increase considerably in the future.

## New Diseases

It is likely that new diseases will contribute to lameness. My own brief experience of outbreaks of *Mycoplasma weyonii* and *M haemobos* shows that both can have a dramatic effect on herd performance and on animal welfare. Studies are currently on going.

## Treatment Options

I remain amazed that we are not sure of the best approach for even the most basic of lameness treatments! I have asked the question in relation to white line lesions at many

meetings. Perhaps there is no difference between the options that will be demonstrated, but when our colleagues in companion animal practice perform such skilled surgical techniques, I am embarrassed that we are not sure.

## CONCLUSIONS

Clinical observation remains an important part of advances in our knowledge. Looking back over this paper it was clinical observation that noted Digital Dermatitis, clinical observation to differentiate heel ulcers from sole ulcers, clinical observation that identified the rib swellings and clinical observation that emphasised the importance of non-healing hoof lesions.

It's my belief that the disease will continue to change and for those of you who intend to carry the torch forward in the future my advice would be simply to look, remain involved on a day to day basis and if you possibly can, work in conjunction with established research groups. For the research groups, don't overlook the part that enthusiastic practitioners are prepared to play in the development of larger scale projects.

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## NOTES



## FUNCTIONAL ANATOMY OF THE BOVINE FOOT – FAILURE OF KEY STRUCTURES IN PATHOGENESIS OF CLAW DISEASE

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### SUMMARY

This paper will present an overview on the functional anatomy of the bovine foot focusing on aspects which are important for our understanding of the pathogenesis of common claw diseases namely laminitis, sole ulcers and white line lesions. It will consider functional key structures within the foot, how they get altered and damaged and how their structural and functional failure leads to claw diseases with all their welfare and economic implications. In identifying these key structures this paper will also present and discuss some options for efficient preventive measures targeting maintenance of these structures.

### INTRODUCTION

The claw is a much more delicate and sensitive organ than it appears to be. Understanding how it functions is an excellent beginning to understand and successfully prevent lameness. Lameness is a clinical sign with a multifactorial etiology. It is a response to pain associated with foot lesions. In the dairy industries lameness is the most visible animal welfare concern and one of the largest economic threats (31). In order to prevent cows from becoming lame we need a better knowledge of the risk factors and we need to understand how risk factors contribute to the damage and failure of key structures in the claw.

The bovine claw is the interface between the dairy cow and the environment. The organ systems of the individual animal as well as the environment permanently interact with the tissues, cells and molecules of the claw. The result of the interaction very often is detrimental to the structure and subsequently the function of the claw and ultimately detrimental to the animal. Anatomically the claw is designed for stance and locomotion on pasture type ground and requires limited periods of standing. Standing and walking on hard floors and extended periods of standing inevitably lead to damage and failure of the key structures of the claw. Over the past years we have gained insight in the impact of the cow's environment on structures in the foot, either directly mechanical chemical or biological or indirectly by altering the cow's behavior which in turn may lead to damage of the claw tissues. Environmental factors cow comfort and cow behavior are understood as key factors in the multifactorial etiology of claw diseases and in lameness prevention (1, 2, 6, 7).

#### The bovine foot – key structures

The performance of the foot is genetically determined and limited. The interaction between the following listed key structures and the environment results in a cascade of physiopathological events leading to adaptation changes, alterations or damage and functional failure of the tissues.

The key structures of the claw are:

1. the dermal vascular system
2. the dermo-epidermal junction
3. the dermis (including the suspensory apparatus of the digit)
4. the sub cutis (digital cushion)
5. the horn producing living epidermis

## 1. DERMAL VASCULAR SYSTEM

### Functional Anatomy

The dermal vascular system of the claw is unique in its three-dimensional arrangement, complexity and density, which is the reason for its high susceptibility to structural damage and disturbances to the perfusion of blood (13, 14). Structural peculiarities in the vascular system and arterio-venous anastomoses (AVAs) in particular have been described as having a central role in development of laminitis. AVA's are shunts between the arterioles bringing the blood into the capillary bed and the venule draining the blood from the capillary bed. They are supplied with nerves which innervate the smooth muscle cells in vessel walls which can close blocking perfusion of the capillary bed of the dermis. Micro-corrosion cast studies by Hirschberg and others (13, 14) have demonstrated that there are almost no AVAs in the vascular system of healthy claws. There is an increase in the number of AVAs if a disease is present, e.g. during laminitis or in the periphery of sole ulcers. Consequently the hypotheses favoring closure of AVAs as a major initial event in the pathogenesis of bovine laminitis do not apply.

The overall microvasculature in the dermal papillae and lamellae is quite extensive, but not all branches of this microvascular bed are perfused at all times. A primary pathway supplies the dermal and the adjacent epidermal cells with their basic needs. The actual perfusion pattern is adapted permanently to the tissue needs by pre-capillary sphincters. The primary pathways ('thoroughfare channels') are the main routes within the capillary bed where the central arteriole and venule are connected inside a papilla. The functional pathway required for proper supply of the adjacent epidermal layers is provided by the extensive capillary network of the papilla. AVAs are located outside the microvascular bed and regulate extreme demands for perfusion. Therefore, they occur at the base of the lamellae and papillae and in the deeper layers of the connective tissue of the claw, not within the lamellae and papillae. AVAs are subject to remodeling and are formed on demand (14, 15).

### Physiopathology – Failure

The dermal microvascularisation and the perfusion patterns are highly adaptable to metabolic and functional requirements. Mechanical forces during weight-bearing deform the papillae and open or close pathways within the vascular system of the papillae. Pressure in the vessels and tissues increases during laminitis which could be explained by compromised function of AVA's but a much more likely explanation would be coagulopathy. The pressure increases in the capillaries together with transvascular movement of fluid in tissues may be caused by an increased post-capillary resistance. This resistance is believed to be the result of a reduction in the diameter of the venules draining blood from the capillary bed (5).

The initial local reaction to laminitis that takes place in claw tissues are alterations in the vascular endothelial lining (roughening) and in the microcirculation (changes in the rate of perfusion). These events are followed by the activation of a variety of interacting and cross linked inflammatory and regulatory cascades. MMP activation is only one part of the jigsaw.



Hirschberg and Plendl (15) investigated the formation of new blood vessels in diseased claws. Based on their studies of the morphology of microvascularisation and angioadaptation of the claw they postulate a central role of pododermal angiogenesis in the pathogenesis of laminitis. It well may be that further studies on the angiogenesis, i.e. the de novo formation of blood vessels will significantly contribute to the understanding of laminitis.

## 2. DERMO-EPI DERMAL JUNCTION

### Functional Anatomy

The dermo-epidermal interface is a highly developed and specialized region at the border between dermis (connective tissue) and epidermis (epithelium) (20). The living epidermal cells located on the interface proliferate and show high metabolic activity. All nutrients and substances required for the epidermal cell metabolism have to pass from the dermis into the epidermis. The living epidermal cells have to withstand a high mechanical load while transferring all the mechanical forces between the underlying bone and the outer horn capsule and the environment. With its complex functions the dermo-epidermal interface is a structure of crucial importance for the integrity and normal function of the claw. It establishes the attachment of the living epidermis to the underlying dermis. All molecular signals between dermal and epidermal cells run through this interface.

### Physiopathology – Failure

Epidermal-dermal interactions (also referred to as dermo-epidermal cross-talk) play an important role in regulating the proliferation and differentiation of keratinocytes which play an important role in repairing surgical and traumatic injuries of the claw capsule. Early in the pathogenesis of laminitis alterations in the dermo-epidermal region have been reported such as initial molecular and structural changes followed by functional disturbances. In addition, on the dermal side, activation of MMPs (27) leads to degradation of collagen as well as activation of growth and necrosis factors, molecular and structural alterations in the basement membrane (11) and alterations of capillary walls.

Changes on epidermal side have to be considered as secondary changes due to disturbed nutrient and oxygen supply. A double paracrine regulation of keratinocyte growth and differentiation has been postulated and described in vitro (22). Interleukin-1 (II-1) and keratinocyte growth factor (KGF/FGF-7) and their receptors are major mediators in this epidermal-dermal signaling. II-1 is synthesized in the Keratinocytes of claw epidermis. It is released upon physical or chemical injury of the cells but also by the effects of cytokines. II-1 binds to receptors on dermal fibrocytes where it causes release of Keratinocyte Growth factor (KGF). KGF is an important stimulator of keratinocyte proliferation. There is preliminary evidence for a reciprocal regulatory mechanism present in the bovine claw involving II- 1 produced in the epidermis and KGF originating from fibroblasts. It is reasonable to assume that mechanical load triggers II-1 release and thus activates the paracrine regulation. Increased rate of proliferation and horn formation would be the consequence. In addition MMPs would become activated by the released Interleukin 1. The collagen of the innermost layer of the basement membrane is a substrate for activated MMP's which degrade this collagen disrupting the integrity and regulatory/communicative functions of the basement membrane (11).

### 3. DERMIS INCLUDING THE SUSPENDING SYSTEM OF THE PEDAL BONE

#### Anatomy & Physiology

The pedal bone of the claw is suspended by a collagen fiber system inside the claw capsule the same time it is supported by a system of digital fat cushions acting as support and shock absorber during locomotion. The pedal bone is kept in a certain position preventing excessive pressure on the sensitive dermis and living horn producing epidermis.

All of the structures between the surface of the bone and the inner aspect of the cornified claw capsule contribute to suspensory functionality; in their functional entity they are called suspensory apparatus of the pedal bone. This apparatus consists of a dermal component with a deeper reticular layer and a superficial papillary layer (dermal papillary body) and an epidermal component including living epidermal layers and the inner parts of the stratum corneum. The dermal and epidermal components of the suspensory apparatus are arranged in interdigitating dermal and epidermal laminae. The collagen fibers run upwards from their insertion in the bone to the basement membrane of the dermal lamellae where they are anchored and thus connected to the lamellar epidermal of the claw capsule (35, 36). It is mainly the suspensory apparatus of the digit that determines the degree of tissue compression in the sole beneath the pedal bone.

The quality of the collagen fibers of the connective tissue is of critical importance for the function of the suspensory apparatus, i.e. to hold the pedal bone in a stable position inside the claw capsule (18, 27, 35). For whatever reason a loosening or increase in length of this connective tissue occurs it will lead to displacement (sinking, rotation, tilting) of the pedal bone within the horn capsule and subsequent increase in pressure onto the soft tissue between bone and horn.

#### Physiopathology - Failure

10 years ago the basic concept of the role of the suspensory apparatus of the pedal bone of the claw and the digital cushions was introduced by Lischer et al. (18). Subsequently the outstanding role of these structures during the pathogenesis of subclinical laminitis was elucidated. Several possible pathomechanisms involved were discovered and hypotheses formulated that explain the changes which occur in these structures and ultimately lead to sinking of the pedal bone and tissue destruction causing a broad spectrum of laminitis associated lesions such as hemorrhages, sole ulcers and white line disease. Recently the biomechanics and histology of the claw suspensory apparatus have been investigated in experimentally induced acute laminitis (8). Acute laminitis is not associated with a decrease of mechanical stability in the suspensory tissues of the claw. This work supports previous studies that demonstrated strength of the suspensory apparatus was affected by housing but not by nutrition (17, 27). The results also suggest that risk factor contributing to subclinical laminitis have a more long term degenerating influence on tissue causing the alterations and subsequent dislocation of the pedal bone. And finally they highlight that acute laminitis is a different disease than subclinical laminitis/claw horn disruption in cattle.

An elongation, loosening or an increasing elasticity in the collagen system suspending the pedal bone inside the claw capsule are central to hypotheses explaining the pathogenesis of subclinical laminitis (21, 22).

During the periparturition period and throughout the onset of lactation the properties of the connective tissue of the suspensory apparatus undergo changes leading to decreased stability of the collagen system of the dermis (16, 22). As a result, there is increased mobility of the pedal bone inside the claw capsule (18, 21). Changes in position of the pedal bone, i.e. sinking rotation tilting to axial or abaxial) have been demonstrated in pathomorphological studies (3, 18, 21). They will cause increased pressure onto the

dermis and living epidermis (germinative layer). The soft living tissues are caught between a rock and a hard place. Disruption of tissue elements including blood vessels is the consequence causing hemorrhages varying in size and severity depending on nature and severity of the positional changes of the pedal bone. More severe damage leads to penetration of the protective horn capsule, i.e. development of a sole ulcer.

The critical questions are: What are the hazards/risk factors causing degradation of the collagen? And what are the local mechanisms in the claw mediating alterations of tissue? Evidence is accumulating that housing and claw trimming are major hazards (32, 33, 34).

Experiments designed to explore the importance of housing feeding and parturition/lactation indicate that the structural integrity of connective tissue was most severely compromised by housing in cubicles. Parturition and lactation amplified this effect whereas feeding had no significant influence (32, 33, 34). Within this context it must be re-emphasized that the dermis is exposed to high local mechanical pressure (12, 28, 29, 30), particularly when cows stand for excessively long period throughout the day. Cubicle housing in comparison to straw yards leads to elevated level of pro MMP2 and active MMP 2 in the connective tissue of the claw (27, 34).

A group of proteolytic enzymes resident in connective tissue, the Matrix Metalloproteinases (MMPs) play a central role in the degradation of collagen. Under conditions of physiological homeostasis MMPs are controlled by their tissue inhibitors (TIMPs) which normally prevent excessive MMP activity. In disease, elevated levels of active MMP result from decreased TIMP activity and increased conversion of inactive pro MMP into active MMP. When MMP expression is elevated and TIMP activity decreased bioactive molecules activate MMP's. Pathological activity of MMPs leads to increased collagen degradation resulting in decreased stability and ultimately failure of the suspensory apparatus (27). The increased mobility of the pedal bone within the capsule causes displacement (sinking and/or rotation or tilting or a combination of these movements) of the pedal bone to a degree depending on the localization and severity of the collagen degradation. There is strong evidence that mechanical irritation and/or overload associated with housing and exposure to concrete are major risks for the structural and functional integrity of the suspensory apparatus.

## 5. SUBCUTIS WITH THE SUPPORTING DIGITAL CUSHIONS

### Anatomy & Physiology

The digital cushion extends forward beneath the pedal bone and is made up of three cylindrical parallel oriented bodies each with a capsule of connective tissue filled with soft fat (23, 24).

During normal gait the heel bulbs make the first contact with the ground and the weight will be distributed equally between the outer and inner claw. While the resilient bulbs reduce the initial shock on the posterior part of the claws the weight of the animal is smoothly transferred to the wall and adjacent sole by slight splaying of the claws. The dermis of the sole and heel and the underlying fat cushions in the subcutis function as 'shock absorbers' bearing a considerable proportion of the impact of the first phase of each step the animal makes when walking. The digital cushion acts in conjunction with the retinaculum of the digit and surrounding soft-elastic horn of the bulb. The elastic tissues of the retinaculum expand laterally when compressed during weight-bearing. The lateral pressures are transferred to the wall which normally has high tensile strength functioning as a spring to absorb some of the energy of locomotion. This tensile strength of the wall diminishes if the quality of the horn of the wall is compromised by disorders such as subclinical laminitis, horizontal or vertical fissures, or heel erosion.

It has been assumed that the weight distribution between the medial and lateral claws is equal. However, the medial claw is frequently smaller than its lateral counterpart. Prolonged exposure to concrete surfaces causes the solear surface of the lateral claw to flatten and increase in width. This process changes the dynamics inside the claw. Instead of weight-bearing being confined to the wall part of the load is transferred to the central part of the sole of the sole. This creates abnormal pressure on the dermis of the sole. This process accounts for the 'traumatic' component in the etiology of subclinical laminitis and the lesions associated with it.

#### Physiopathology - Alterations of the digital cushions

There is a change in composition of the digital cushions as the animal gets older. In heifers the fat cushions are not completely developed and functional. They develop to full shock absorbing capacity during the first 2 lactations. The fat content is significantly higher in cows (38%) than in heifers (27%) (23, 24). The cushions in the heifers are composed predominantly of loose connective tissue with abundant amorphous ground substance. In cows however, there was a marked increase of adipose tissue with progressing age (24). A comparison of the digital cushions between sound claws and claws with sole ulcers revealed that the phalanx of ulcerated claws had sunken and the solear dermis and subcutis were thinner than in the controls. The cushions contained significantly less adipose tissue than the controls but had been replaced by collagenous connective tissue (18). The fatty acid composition and the size of the fat cushions change under the influence of metabolic disorders, in particular in cows with lipid mobilization syndrome (LMS, ketosis). The fat in the digital cushion has a high content of monounsaturated fatty acids (MUFA). MUFA are mainly produced endogenously and the greater the quantity of these fatty acids in the fat tissue, the softer it is; the structural fat in the digital cushion possesses its own 'fat softener' (25). Heifers have significantly less fat in the cushions and slightly more saturated fatty acids (SFA) than the cows. This indicates that the change from SFA to MUFA and the proliferation of fat occurs at first parturition and during the following lactation. It is possible that these changes in the heifer's digital cushions make them less resistant to pressure load. Epidemiological studies have shown that there is a higher tendency for sole lesions to occur at the beginning of the first lactation (9, 10, 26).

Changes in cushions seem to be correlated with metabolic changes around parturition and associated changes in body condition. Bicalho et al. measured the thickness of the cushions in heifers using ultrasound. They found that body condition scores were positively associated with digital cushion thickness (4).

#### CONCLUDING REMARKS

Weakening of the structural integrity of the key structures of the foot directly leads to functional failure and disease. The bovine foot is not constructed for permanent use on hard flooring. Continually walking and in particular excessive standing on concrete or asphalt causes reactions and adaptation changes to the point of damage of the tissue. Cow comfort must be interpreted as part of the interaction between the foot of a dairy cow and its internal and external challenges; the most critical period in this aspect is the time around calving. There are anatomical differences in the digital cushion between heifers and cows. It is therefore important to give the heifers enough time to adapt to the new housing conditions of dairy cows.

Damage and failure of the key structures and resulting claw diseases have a multi-factorial etiology. Multi-factorial means that practically every aspect of dairy cow management has to be scrutinized for its potential impact on the key structures of the foot. Preventive measures must be geared to managing the stress level that precipitates the complex physiopathology intrinsic to the foot.

Looking at the biomechanical aspects of claw diseases it is clear that professional functional hoof trimming helps to prevent or reduce the damage to the functional important key structures and prevent or at least limit the severity of functional failure. Routine claw trimming helps to balance the load carried by the claw. Sole ulcers develop as a consequence of a partial failure of the suspensory apparatus. Whenever possible the ulcerated claw should be unloaded with a claw block on the sound claw.

The better we understand how the key structures of the bovine foot function the better we will be able to identify key risks in dairy management and the better we will be able to prevent failure of key structures of the foot with all its economic and welfare implications.

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## NOTES



## RESEARCH REVIEW INTO THE LAME COW

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### INTRODUCTION

The welfare, production efficiency, economic and environmental impacts of lameness in dairy herds are unfortunately accepted facts in dairy farming systems throughout the world. In short there is no other disease syndrome affecting dairy cows that can have such a significant negative effect on a cow's life and potentially lead to her premature death (1).

Without doubt if the industry can apply the current knowledge base on lameness control more fully, significant reductions in on farm lameness prevalence could be achieved. Central to this are industry lead initiatives such as the DairyCo Healthy Feet programme, milk buyer initiatives and veterinary practice based health planning, mobility scoring and farmer training. These are therefore exciting times and current research needs to consider how best to get the message across to the farmer to be implemented for the benefit of the cow (2). This paper will discuss some of the findings from different areas of SAC's lameness research over the last 10 years and their practical application on farms.

### LAMENESS INCIDENCE AND PREVALENCE

Knowing lameness incidence or prevalence is important as a benchmark for future control at a farm level, to highlight success in control and to motivate farmers to make changes where necessary. Lameness incidence is hard to define absolutely with large variations between farms, recording methods and criteria. Lameness prevalence, that is the number of cows that are lame at any given time point, is perhaps a more accurate measure. Studies have shown average prevalence ranging from 20% in the 1990's (3) to 36% in the recent Healthy Feet Project (4). This does not necessarily indicate an increased prevalence over time, especially given that the range of prevalence in the healthy Feet Project study of 0% - 79%.

Studies carried out by SAC have provided similar prevalence estimates and highlighted some of the differences resulting from different management systems. One study compared 40 conventional farming systems with 40 organic farming systems in the UK and showed an overall lameness prevalence of 19% (5). It was considered that this figure was lower because of a higher proportion of straw yard systems on study farms. The study reported half as many lame cows on organic farms using straw yards compared to conventional farms using cubicles. Another study on 37 dairy farms across the UK highlighted the difference in lameness prevalence between management systems. This study showed a lameness prevalence of 15% for grazing herds and a lameness prevalence of 39% for zero grazed herds (6).

It would seem that overall there has not been significant progress in reducing lameness prevalence in the national herd. Given the painful implications for each cow making up this prevalence data (7) national lameness prevalence is too high. However this masks significant achievements on individual farms and reduced levels of lameness are achievable on all farms irrespective of system (8).

One final consideration with any lameness prevalence estimate is the ability of farmers to detect and appreciate when a cow is actually lame. At the start of a lameness

technology transfer project run on 12 farms in South West Scotland (MDC Link Farms project) study farmers were only identifying 35% of the lame cows identified by mobility score. However by the end of the project this figure had increased to 95% of lame cows identified by mobility score. This increase was achieved by quarterly whole herd mobility scoring with feedback and discussion of the results with the farmers.

## MANAGING FIRST LACTATION HEIFERS

Hirst (9) showed that if a heifer is lame in its first lactation it is approximately three times more likely to be lame in the second lactation. Similar research at SAC (10) examined the feet of 31 heifers over four lactations and showed that lesion and locomotion scores were significantly higher by the 4<sup>th</sup> lactation. Therefore looking after the heifer's feet should have an overall long-term beneficial effect reducing lameness incidence at a herd level. For the dairy farmer with a large herd and pressure on staff time this is a really important message for lameness control in the long term and anecdotal experience controlling lameness on farm supports this. Given this message when should heifer's feet be trimmed in first lactation and how can risk factors be managed to reduce the risk of heifers going lame?

## WHEN TO FOOT TRIM FIRST LACTATION HEIFERS?

Work by Manske (11) has shown the beneficial effect of trimming cows twice a year (once in the autumn and once in the spring); although the authors do state that the specific requirement to do this would depend on individual herd need. SAC research has shown in repeated studies over the last 15 years that there is a peak in sole lesion score 14 weeks after calving and white line lesion score 9 weeks after calving (12). Putting both research findings together a general recommendation to farmers to trim first lactation heifers feet routinely 2 -3 months after calving has shown in practice to bring about real benefits in foot health both in first lactation and building in foot health for future lactations.

## CUBICLE TRAINING HEIFERS

Putting untrained heifers into cubicles after calving occurs on some farms. Field experience suggests that untrained heifers take some time to become accustomed to using cubicles and so tend to lie down less when they first enter the main milking herd which may contribute to subsequent lameness. An SAC study (13) investigated the effect of training heifers to use cubicles during the winter before first calving on claw horn lesion development in the year after calving. Heifers were either housed in a cubicle shed (trained, n=31) or in a straw yard (untrained, n=33) during the winter prior to calving. Heifers remained on the straw yards until batches of four or more animals had calved and were ready to enter the main milking herd. Claw horn lesions were monitored at regular intervals throughout first lactation. For sole lesions there was a significant beneficial effect of training to use cubicles. The take home message for farmers is that for herds using cubicle housing all heifers are trained to use cubicles prior to calving. The 'training period' need be no longer than one month and for herds without cubicle accommodation for young stock, housing the heifer group in the main cubicle shed for a short period after the cows have been turned out for the summer is a compromise that many farmers find practical.

## DIETS FOR REARING HEIFERS

The diet fed to rearing heifers is also a significant pre-disposing factor to the development of lameness in first lactation. In one experiment conducted at SAC's Crichton Royal Farm the foot health of a group of heifers was observed from birth to 6 months after first calving. The animals were paired and were fed to one month prior to calving either a low dry matter wet fermented diet based on grass silage or a high dry matter unfermented diet based on straw and a concentrate mix. Subsequently in their first lactation all cows were fed the same grass silage based diet. There was a significantly greater incidence of clinical lameness in the heifers reared on the wet fermented ration with the greatest difference seen in the sole lesions (14). In addition from cow observations not surprisingly heifers fed a dry diet spent less time eating, more time lying down and their slurry produced was less more viscous. These behavioural and walking environmental effects may have been the most significant factors in reducing the levels of lameness. This again is an important take home message for farmers and if consistent straw supplies can be obtained there are significant benefits to be had from rearing heifers on a drier diet.

## INFLUENCE OF GENETICS AND ENVIRONMENT ON LAMENESS

The inter-relationship between lameness, yield and genotype was studied using the Langhill herd at Crichton Royal Farm. Two different genetics lines have been selected one bred from the top 5% genetic merit bulls for fat and protein yield (select) and the other, to match the UK average genetic merit (control). The two genetic lines were managed in two contrasting approaches to dairy herd management, a high forage system (HF) and a low forage system (LF) with an equal split of genetic lines in each system group. In the HF system, the cows grazed when sufficient herbage was available and were fed a complete diet containing between 70% and 75% forage in the dry matter when grass heights fell below set values and housed in the winter months. In the LF system, the cows were housed throughout the year and were fed a complete diet containing between 45% and 50% forage in the dry matter. The low forage diet contained approximately 1000 kg concentrate while high forage diet contained approximately 3000 kg concentrate in a year. The forage component of the complete diet consisted of grass silage, maize silage and whole crop (15).

Hoof health was assessed in a number of different ways: Weekly locomotion scoring (1 - 5 score) was carried out to identify the weekly prevalence of lameness in the herd and any individual clinically lame cow needing to be examined. Clinically lame animals (score 4 – 5) were examined and treated on a weekly basis by a veterinarian or by the herdsman where necessary and records of the lesions were recorded. Any chronically lame animal was reviewed monthly with detailed records of the lesions recorded.

Hoof examinations of all feet of all first lactation heifers were carried out every two months. Heifers were first examined up to 2 months before first calving and every 2 months thereafter giving 6 examinations of each heifer through the first lactation. Information from this was used to derive white line and sole lesion scores which give a 'sub-clinical' picture of hoof health. Further information on hoof health was gained by recording in detail any lesions seen at the routine foot trimming sessions carried out by contract foot trimmers who lifted the feet (usually just the hind feet) of all animals twice a year.

Considering the locomotion score data no significant effect of genetic line was shown to affect lameness. However the management system effects were more marked with LF cows having higher locomotion scores compared to HF. When the findings from the clinical examinations of lame animals were investigated, the higher levels of lameness in

LF was seen to be due more to infectious causes of lameness such as digital dermatitis and inter-digital growths.

Lesion development data was studied separately for sole and white line lesions from the routine observations made every 2 months on first lactation heifers. Sole lesions were found to be worse at 110 days post calving (on average). At peak sole lesion score LF heifers had marginally significantly more lesions than HF heifers. Within HF select cows had more lesions than control.

Peak lesion score for line lesions occurred, on average, at 60 days post calving in first lactation animals. Scores for LF were higher than for HF, and were higher in select compared to control cows.

Looking in more detail at the systems, three production systems were defined based on feed type and housing. The 3 systems were, low forage-continuously housed system, high forage-housed (over winter), and high forage-at grass (grazing in summer). In the study 1102 weekly prevalence records from the period 2004 and 2009 were used. Weekly lameness prevalence was calculated as a proportion of the herd that had cows with a locomotion score of 3 and above. Again in this analysis the low forage-continuously housed system had higher weekly lameness prevalence than those in the high forage system. Cows that alternated between indoor and outdoor life (high forage) had higher weekly lameness prevalence when they were outdoors than when they were back indoors suggesting that whatever benefits on feet health they got whilst outdoors were exhibited when the cows returned indoors (16).

## CONCLUSION

Production and health data from the herd continues to be generated on a daily basis and it is hoped that the large data set will allow us to answer further specific questions relating to lameness issues. Work so far suggests that the genetic effects on lameness are small compared to the environmental ones. That is not to say that there are no genetic effects, but that we need to develop more sensitive measures to assess and control them further.

It has been shown that cows with a history of lameness are higher yielding throughout lactation compared to cows that were never lame, that is lameness is a production disease associated with higher yield (17). Clearly both genetics and management system can have an effect on this and further studies on the links between body condition score, milk yield and lameness are planned to look further at the susceptibility of high yielding cows to becoming lame.

## ACKNOWLEDGEMENTS

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## NOTES



## DIGITAL DERMATITIS: WHAT IS UNSOUGHT WILL GO UNDETECTED

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### INTRODUCTION

Digital dermatitis (DD) is a dynamic infectious skin disease of dairy cattle. Reducing the number of individual cases of DD by early detection and treatment, and preventing new cases from occurring are necessary disease management strategies. Traditional inspection of the lifted foot using a crush is logistically challenging as a routine method of detection, particularly as herd sizes increase. Locomotion scoring can be used to identify foot lesions but DD can be present in the absence of lameness (11).

Rodriguez-Lainz et al. 1998 (9) observed individual cows in a milking parlour for approximately two minutes each and found an apparent DD prevalence of 20.5% (24/117) compared with an actual prevalence determined by inspection in the crush of 27% (32/117). The milking parlour sensitivity was 0.72, with a specificity of 0.99. However, the sensitivity and specificity may not be a genuine reflection of agreement as the crush examination took place up to a month after inspection in the parlour in which time lesion status could have changed.

More recently, Thomsen et al. 2008 (12) investigated a rapid screening method taking approximately 15 seconds to observe each cow in the parlour. This study found a sensitivity of 0.65 and specificity of 0.84 in the parlour compared to inspection in the crush. Relun et al. 2011 (8) reported more promising data with a sensitivity of 0.90 and specificity of 0.80 for detecting the presence and absence of DD lesions in the parlour taking between 30 – 60 seconds per cow. This variation can be attributed to differences in experimental method, including the time taken to observe each cow as well as differences in observer training, experience and accuracy. Taken together these studies suggest that by increasing the time taken to observe each cow, the reliability of detecting DD in the parlour can be increased.

Laven, 1999 (6) validated the Borescope as a tool for identifying DD in standing cows and reported individual lesion specificity of 0.84, and sensitivity of 0.82. The technique was subsequently adopted in several research contexts (13). However, the reliability of a straight forward visual inspection in the milking parlour has not been compared with using the Borescope. In practice, the expensive and cumbersome nature of the Borescope prohibits wide scale application on farm as a routine detection aid.

In order to identify a measure that can be used to classify lesions by stage of infection over time, the reliability of different scoring systems has been assessed. A range of characteristics have been used to monitor lesion progression previously and there is currently no industry gold standard (1, 2, 13, and 4). Before a scoring system that described lesions by stage of infection was developed, researchers used size, depth and colour (9, 6). These characteristics have the advantage of being simple to measure and easy to apply in a farm context; however they do not on their own depict the stage of infection. Scoring systems which describe the various stages of infection were developed by Dopfer et al. 1994 (3) and Vink (13). Although there is much agreement between these systems, the latter was developed in the UK and was therefore used in the development of the described approach.

## An Approach to In Parlour Observation of Digital Dermatitis

The approach to in parlour detection of DD lesions in dairy cows described here was developed and validated as part of a larger longitudinal study investigating the prevalence and severity of DD associated with two common management strategies. Following the development of this approach it was used during afternoon milking to assess the hind feet of all cows on 15 UK dairy farms at monthly intervals over the course of a year. The herds sizes ranged from 80 to 301 cows. All farms had herringbone parlours to enable inspection from the pit that allowed for easy viewing of the heel. After the herdsman had attached the milking cluster, each cow's hind foot was cleaned off with a high pressure hose if the foot was dirty enough to obscure lesions. The nozzle was angled downwards across the hoof with water hitting the heel at a 45° angle to ensure minimal splash up. The foot was carefully examined for DD lesions using a head torch. Digital dermatitis was scored for presence/absence and the stage of lesion was assessed according to Vink (2006).

## DISCUSSION

Research suggests that DD is not always accompanied by obvious lameness (7). The tool used to identify and monitor foot lesions needs to be valid and appropriate for the specific disease under investigation and sensitive enough to identify early cases. Monitoring of DD lesions in the milking parlour has been shown to be a reliable method to assess the effectiveness of on farm prevention and treatment strategies (8). Regularly screening cows for DD in the milking parlour has a number of benefits. Thorough cleaning of the feet can act as a prevention measure against infectious disease. When feet are clean, lesions are easier to identify and the procedure should take less time. Early identification and treatment of cases can reduce the infection reservoir within a herd which is currently understood to be the lesion itself (1). Early treatment intervention can also reduce the likelihood of developing chronic cases (10).

The uptake of this approach as a routine management tool will be largely dependent on the feasibility on farm, such as the parlour set up and provision of labour. During the longitudinal study across 15 farms the duration of milking was not affected by the presence of a scorer carrying out this assessment. However, the current methodology requires another person to carry out the assessment, or the herdsman to spend time integrating this approach into their milking routine.

During a recent phone survey, eighty two percent of farmers (n = 90) reported using the milking parlour to normally detect DD (11). This suggests this approach is being used empirically on farm. Future work should therefore focus on the formalisation and uptake of this method across the dairy industry, including the development of a standardised scoring system. Research that indicates a relationship between stage and colour of DD lesion may provide a basis for developing an easy to describe and simple to measure scoring system that can be practical applied on farm.

## CONCLUSION

In the case of routine assessments, protocol is largely shaped by the feasibility of regular whole herd lesion monitoring. Detecting lesions in the early stages of infection is important from both welfare and disease management perspectives. Regular examination in the parlour following the method described here can be used to identify the presence and absence of DD lesions. This method should be considered for routine on farm detection and monitoring of DD.

## ACKNOWLEDGEMENTS

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## NOTES





## DIGIT AMPUTATION – GETTING IT RIGHT FOR A PRODUCTIVE FUTURE

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### SUMMARY

Whilst surgical techniques involving the bovine digit have been documented as far back as the 19<sup>th</sup> century, there have been relatively few published studies comparing and contrasting surgical options and evaluating survival post-surgery, particularly in field conditions. Since the termination of the over thirty month scheme (otms) in 2006, the introduction of disposal costs for cows culled on farm and the increasing value of cattle, there has been a greater demand for digit amputations to be performed in the field, even if viewed only as a salvage procedure. The objective of this study was to evaluate the effect of digit amputation on survival in the herd and hence whether it is a viable treatment option in the uk. A retrospective case-control study of 182 amputations was conducted. Amputation of hind digits was associated with reduced survival in the herd, with the first 100 days post-amputation being the greatest risk period for culling. In the long term cows that had undergone a hind amputation could expect to remain in the herd for an average of 22 months, in comparison to 39 months for controls. In contrast amputation of a front digit did not significantly influence survival in the herd. Further follow up studies are required to determine how indication for amputation influences outcome to further aid in decision making regarding potential candidates.

### INTRODUCTION

Claw disorders account for 99% of lameness cases in dairy cattle, irrespective of breed or management, the majority of which are infectious in origin (1). Lameness is often a chronic, painful condition and thus a serious welfare concern (2). Treatment is most effective when administered at an early stage, however, it is often delayed or inappropriate due to problems with recognition of the onset of lameness or time constraints as a result of increasing herd sizes (3).

Unless new cases of lameness are dealt with promptly and effectively it creates an opportunity for infection to enter the claw. This may be via the sole (e.g. sole ulcer), wall (e.g. white line disease) or interdigital space (e.g. necrobacillosis). Once infection enters the claw it invariably spreads to the surrounding structures. The capsule of the distal interphalangeal (coffin) joint, navicular bursa and deep digital flexor tendon sheath are grouped very closely together in the middle third of the foot and are particularly vulnerable. As a result this often results in one or more of these structures becoming infected following the introduction of infection into the foot.

Once infection has entered the foot and sepsis occurs, conservative treatment is rarely effective and in many cases prolongs the animal's discomfort (3). In the UK veterinary involvement is often sought once initial treatment by the farmer has failed. This treatment may involve a combination of curative foot trimming, administration of antimicrobials and/or anti-inflammatory drugs. At this point there are three treatment options available: (1) immediate slaughter, (2) amputation of the affected digit or (3) one of the several digit salvage techniques e.g. joint lavage and arthrodesis. The aim of treatment is to resolve the lameness and associated pain as quickly as possible, whilst minimising the impact on productivity. Digit amputation is often the treatment of choice in the field as it is relatively simple and quick to perform.

## DIGIT AMPUTATION

### CHANGING INDICATIONS?

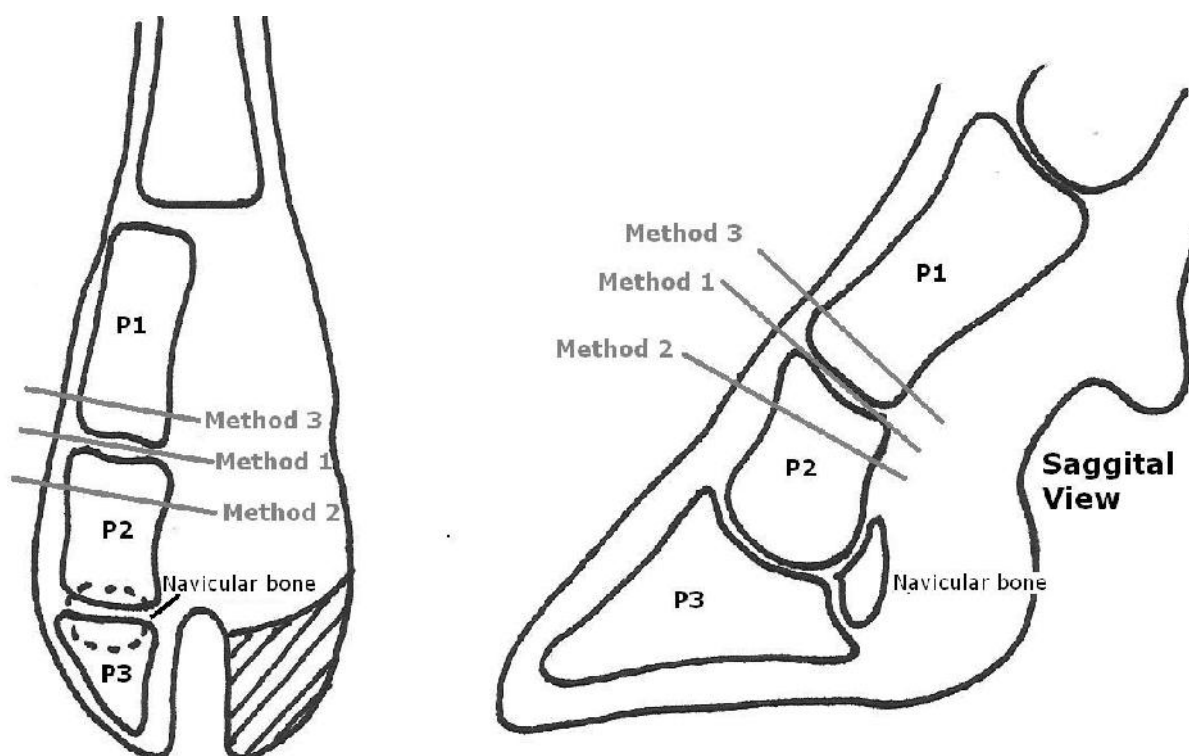
It is apparent from recent discussions in the literature that the indications for digit amputation have altered in the last decade. Historically sepsis of the soft tissue structures and/or joints of the foot have been the main indication, however, more recently this has not been the case. Blowey (41) first reported an apparent increase in the number of amputations being performed due to non-healing white line disease (often referred to as 'wall ulcers') and toe necrosis. Both of these lesions have been found, in many cases, to be infected with the same treponemes found in digital dermatitis cases (5). The chronic inflammation within the corium may also provoke a proliferative osteitis of the pedal bone (6) and production of soft horn which has a 'honeycomb' like appearance (7), both of which exacerbate the non-healing aspect of the condition. As a result, non-healing white line disease, non-healing sole ulcers and toe necrosis are now reported to be the most frequent reason for digit amputation (4, 8).

### Method

As discussed above there are various levels at which amputation can be performed, the most common of which are (Figure 1):

- Method 1: Disarticulation of the distal interphalangeal (DIP) joint
- Method 2: Amputation through proximal Phalanx 2 (P2)
- Method 3: Amputation through distal Phalanx 1 (P1)

Figure 1: Amputation Sites



Methods 1 and 2 are the most commonly performed techniques in UK practice, with Method 1 being slightly more complicated and time consuming compared to the relatively simple Method 2.

Post-amputation recovery time is dependent on the rate at which granulation occurs. For granulation to occur quickly and effectively the tissue requires a surface to which it can strongly adhere. Following Method 1, unless all of the articular cartilage has been scraped away, granulation may be delayed since tissue adheres poorly to the remaining cartilage.

Method 2 results in an area of exposed bone which provides a more adhesive surface, however, this proximal part of P2 often becomes necrotic due to disruption to its blood supply by the diaphyseal artery, as described by Osman (9). In the majority of cases this P2 fragment forms a sequestrum and sloughs off exposing the articular cartilage of the DIP joint. Unless this is subsequently curetted off to expose the underlying bone, granulation is very prolonged and in some cases does not occur. Therefore, following Method 2 recovery can often be protracted and result in poor outcomes.

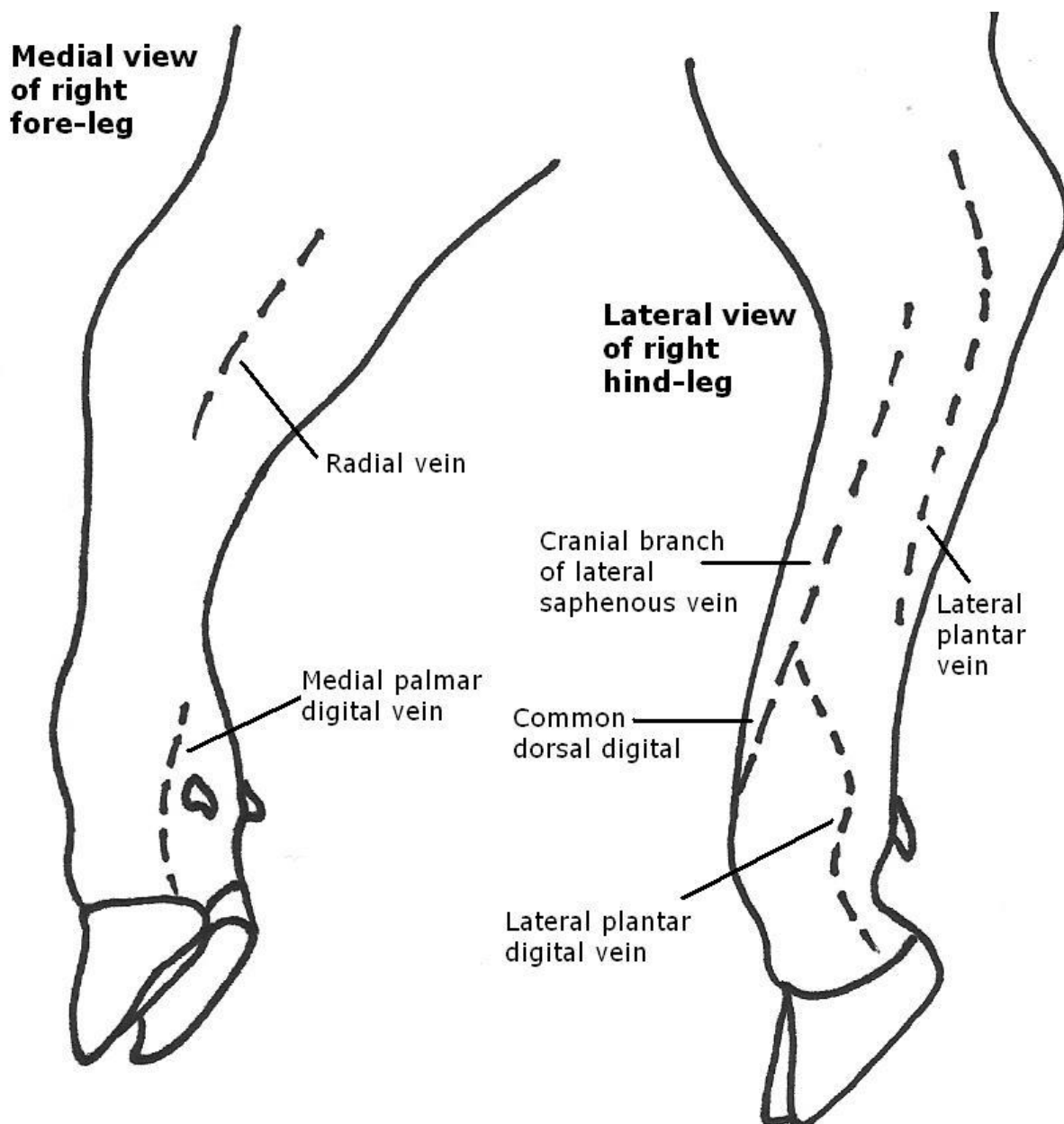
The technique of choice is Method 3 since it results in exposed bone on which granulation tissue can adhere, the amputation is performed at a level where the blood supply to the remaining proportion of P1 is not disrupted and the risk of necrosis is low and, in many cases, the tissue surrounding the amputation site is healthy rather than infected. The higher amputation site also avoids contact of the stump with the ground, particularly if the heel of the partner digit is low.

### Surgical Technique for Amputation through Distal P1 (Method 3)

The surgery is usually easiest to perform with the cow standing and restrained in a foot crush which allows good access to the limb that is to be operated on. Occasionally, if a front digit is to be amputated and the foot is not easily accessible it may be preferable to sedate the cow, cast her and perform the procedure with her in lateral recumbency with the digit to be amputated uppermost. Pre-operative antimicrobials and anti-inflammatories should be administered as soon as the cow is restrained so that they have time to take effect.

Anaesthesia of the digit is most effectively achieved through intra-venous regional anaesthesia (IVRA). A tourniquet is applied to the mid-carpal region if a front digit is being amputated or above the hock for a hind digit. In some cases the leg strap that is used to elevate the hind leg can be used as a tourniquet if it is made into a self-tightening loop. 20-30mls of Procaine without adrenaline should be infused into a superficial vein - the use of a 19 gauge butterfly catheter is recommended. In the hind limb the cranial branch of the lateral saphenous vein is easily accessible and is located over the metatarsus approximately 20cm distal to the hock. If visible or palpable the distal branches of the saphenous vein can also be used; the lateral plantar digital vein and common dorsal digital vein. In the fore-limb the radial vein and medial palmar digital vein are the most accessible (Figure 2). However, if access is restricted and a superficial vein cannot be found or IVRA is unsuccessful due to extensive oedematous swelling, a three-point nerve block should be carried out, which provides good anaesthesia of a single digit. 5-10ml of Procaine should be administered subcutaneously at three sites; below the accessory digit 1cm abaxial to the midline on the palmar/plantar surface, lateral (or medial) at the level of the accessory digit and midline on the dorsal surface at the level of the accessory digit.

Figure 2: Suggested superficial veins for IVRA (adapted from Edwards (10))



Whilst waiting for the anaesthetic to take effect, the foot should be prepared for surgery. Shaving of the skin is unnecessary, however, the interdigital space, digits and skin from the coronary band to just above the accessory digits should be cleaned. In most cases dry wiping of the area is sufficient. If the area is particularly soiled and requires scrubbing then a surgical scrub should be used and the whole foot thoroughly dried prior to amputation to reduce the risk of contaminated water gravitating down onto the amputation site.

To ensure that the digit is amputated at the correct level an initial incision must be made between the claws to a depth of approximately 2.5cm (1"). This can either be achieved using a scalpel blade or embryotomy wire, however, it is important to ensure that whichever method is used that the incision is exactly in the middle of the digits so that no damage occurs to the supporting structures of the remaining healthy digit. Clean embryotomy wire is then introduced into the incision and the digit is then amputated by

cutting obliquely towards the accessory digit. The distal part of P1 should be easily identifiable in the amputated digit if the procedure has been carried out correctly.

The exposed stump should be examined for the presence of residual necrotic or infected tissue and this should be excised if present. Any protruding interdigital fat should also be trimmed away as it may interfere with homostasis. The stump should be treated with an antibiotic powder before being covered with a non-adhesive dressing. To aid in haemostasis a large ball of cotton wool should be placed in the dead space created by the amputated digit and the whole foot then bandaged using a reasonable amount of pressure. This dressing should be changed after four days and then re-dressed for a further ten days. Parenteral antibiotics should be given for a minimum of 4 days and extended if there is excessive swelling or pain. Non-steroidal anti-inflammatories should also be repeated as necessary.

As discussed previously, recovery time is dependent on the speed at which granulation tissue forms, however, in most cases the stump completely heals over with skin in approximately 6 weeks. Recurrent infection or formation of excessive granulation tissue can prolong healing. During the recovery period it is important that cows are monitored closely and also housed appropriately. Whilst straw yards are appropriate in the short term, once the bandages have been removed the straw can irritate the exposed wound and cause unnecessary pain.

## MATERIALS & METHODS

A retrospective case-control study was performed. The clinical records of two large predominantly dairy practices based in Cheshire and Shropshire, UK were used to identify Holstein cows that had undergone a digit amputation (AMP) between 1<sup>st</sup> April, 2000 and 31<sup>st</sup> January, 2011. All amputations were performed by vets who routinely amputated the digit at the level of distal P1. Only farms that regularly milk recorded and were registered on a computerised dairy herd management software programme (Interherd™) were included to ensure sufficient data was obtained. In total 182 cases of digit amputation were identified and enrolled in the study.

Data was collected using a combination of practice records, farm records, Interherd™ data and a questionnaire that was sent out to clients. Herd, lactation number in which AMP occurred, calving date prior to AMP, days in milk at time of AMP, digit amputated and 305-milk yield (kg; MY) for the lactation in which AMP took place and the previous lactation were collected. All cases were followed for at least 14 months post-amputation and if culled during the follow-up period the cull date was recorded.

Of the 182 cows that had undergone AMP, 169 could accurately be matched with one herdmate that served as a Control. No Control had ever undergone AMP or appeared in the study more than once. Control cows were matched as closely as possible based on DIM at the time of digit amputation, previous 305-day lactation yield, lactation number and age group. First lactation heifers were matched with other first lactation heifers based on days in milk and age. Herd, lactation number, calving date, days in milk, 305-day milk yield (MY), and cull date if prior to the end of the follow-up period were recorded for the Controls.

Survival time for both AMPs and Controls was calculated from the date of the amputation until removal from the herd and was censored if the cow still remained in the herd at the end of the follow-up period.

## Data Analysis

Statistical analysis was performed using the computer statistics programme MINITAB

(Version 16). To assess if the digit amputated (i.e. fore-, hind, lateral or medial digit) was a confounding effect on survival, Kaplan-Meier survival analysis was used with log-rank comparison of survival curves.

Kaplan-Meier survival analysis was then used to compare survival between AMPs and Controls at 100 and 365 days post-surgery and also long-term (>365 days; at least 14 months). The Kaplan-Meier estimate of mean time to failure was used to estimate average survival time post-amputation, as this takes into account those animals that have not been culled at the end of the study and therefore have censored survival times.

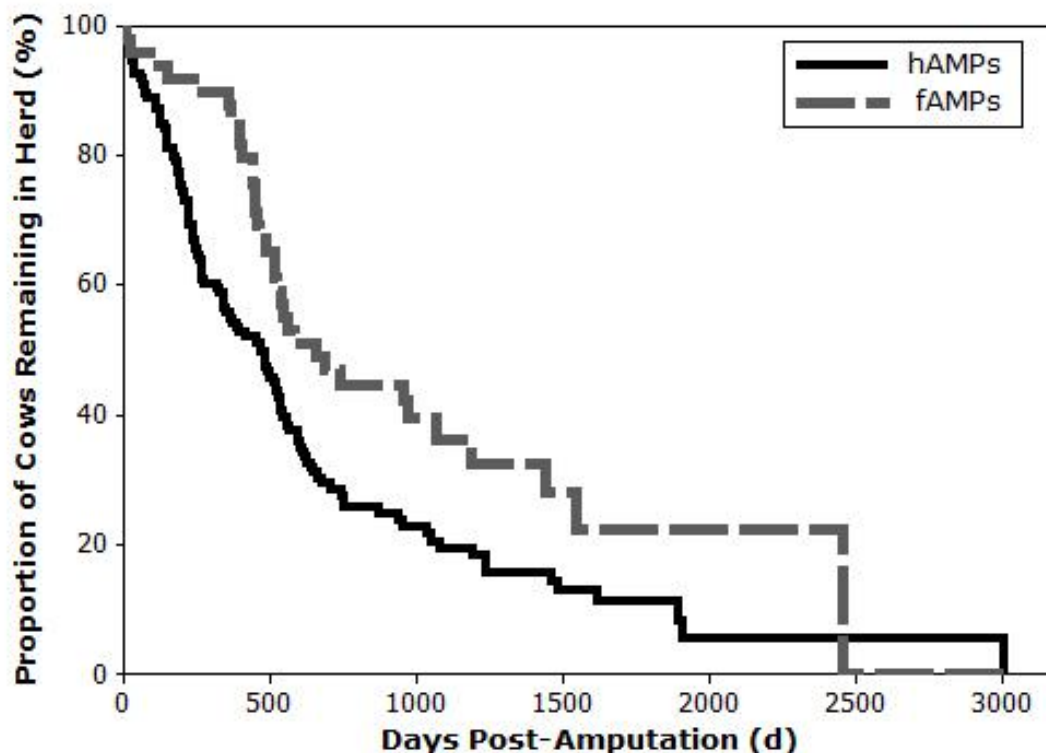
## RESULTS

Data was collected on 182 cases of digit amputation on Holstein dairy cows from 57 farms (n=1-11). AMP was performed during the 1<sup>st</sup> to 11<sup>th</sup> lactations (Mean = 3.67; SD = 2.04) and from the day prior to calving until and 842 days post-calving (at dry off; Mean = 216.50; SD=171.10). Four AMPs were performed on in-calf heifers. 108 AMPs involved a hind lateral digit, 23 a hind medial, 10 a front lateral and 39 a front medial digit.

### Effect of Digit Removed on Cow Survival

Survival analysis was used to determine if the digit removed significantly influenced survival in the herd. There was a significant difference between survival of hind and front digit amputees ( $p=0.008$ ; Figure 3), however, there was no significant difference when medial and lateral claws were considered separately ( $p>0.05$ ). For the purposes of further analysis hind and front digit amputations were considered separately.

Figure 3: Kaplan-Meier Survival Plot showing survival in the herd following a hind or front digit amputation

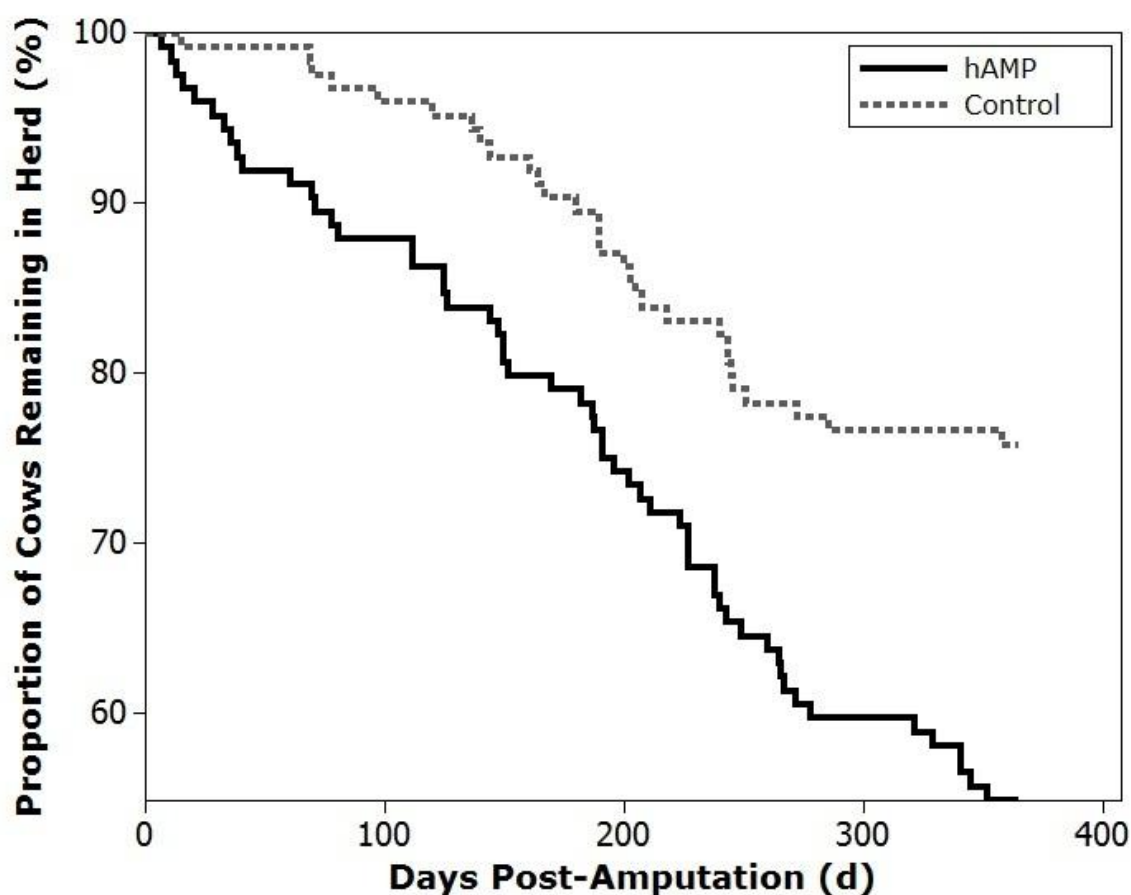


### Hind digit amputation (hAMP)

Survival analysis was conducted on 124 hAMP-Control pairs at 100 days, 365 days post-amputation and >365 post-amputation (at least 14 months).

Survival at 100 and 365 days was significantly different between hAMPs and Controls ( $p=0.018$  and  $0.000$  respectively). The proportion of hAMPs in the herd at 100 and 365 days post-amputation was 87.9% and 55%, compared to 87.9% and 76% for Controls. Figure 4 shows the survival curve for hAMPs and Controls in the 365 days post-amputation. Examining the gradients of the survival plots it is apparent that the first 100 days post-amputation is the critical time during which culling rates for hAMPs is much greater than for Controls. The main reasons for hAMPs being culled during this period were lameness in the remaining digit, unresolving infection in the stump and farm policy to cull the cow following recovery whilst she still had a market value.

Figure 4: Kaplan-Meier Survival Plot for hAMPs and Controls for 365 days post-amputation



As expected, long term survival in the herd (>365 days) was also significantly lower in hAMPs ( $p=0.000$ ). The Kaplan-Meier estimate for mean time to failure (i.e. survival time) was 667.00 days (22.2 months; 95% confidence interval; lower =522.52, upper =811.48) for hAMPs and 1187.33 days (39.6 months; 95% confidence interval; lower =949.91, upper =1424.75) for Controls.

## Front digit amputation (fAMP)

Survival analysis was conducted on 45 fAMP-Control pairs at 100 days and 365 days post-amputation and >365 days post-amputation.

Survival was NOT significantly different between fAMPs and Controls at 100 days ( $p=0.982$ ), 365 days ( $p=0.988$ ) and long-term (>365 days;  $p=0.374$ ) post-amputation. At 365 days post-amputation 84% of both fAMPs and Controls were still in the herd.

The Kaplan-Meier estimate for mean time to failure (i.e. survival time) was 1069.84 days (35.6 months; 95% confidence interval; lower =784.75, upper =1354.94) for fAMPs and 1157.33 days (38.6 months; 95% confidence interval; lower =949.91, upper =1424.75) for Controls.

## DISCUSSION

The distribution of digits requiring amputation in this study is consistent with previous studies that have identified the most common sites of lameness. Nearly 75% of the digits amputated were in the hind limbs, with the lateral digit amputated in 59% of cases. Findings of other studies have indicated that up to 90% of lameness develops in the hind limbs with 87-90% of cases being localised to a lesion in one or more digits, most commonly the lateral digit (11, 12).

Whilst the forelimbs are responsible for supporting approximately 60% of a cow's body weight, it is the hind limbs that provide most of the propulsive force (8). Therefore, due to this, and the fact that hind limbs are more at risk of lameness lesions, it would be expected that the prognosis for hAMPs would be less favourable than for fAMPs. The findings of this study support this theory but contrast the findings of Pesja and others (13) who found differing success rates depending on the hind digit that was amputated with front digit amputations performing better than hind lateral amputations but worse than hind medial amputations. However, this study only involved 85 cases in total and included cattle of varying ages, breeds and sex, making direct comparisons difficult.

Previous studies have consistently shown that long-term survival time in the herd is reduced following digit amputation and the results of this study support this in the case of hAMPs. Average survival post-amputation has been the most commonly used measure of survival post-surgery. However, this does not allow the inclusion of data from cows that are still in the herd at the end of the study. Survival analysis was used in this study as it shows the rate of culling by time and allows animals that have not been culled to be included in the analysis. Although this is accepted as the most appropriate method of analysing survival (14), it can only give an estimated average survival time based on the data available.

Comparison of survival at 100 days post-amputation was not significantly different between Controls and fAMPs but was for hAMPs. This was a surprising find since the initial period after surgery is often expected to be the greatest period of risk for removal from the herd irrespective of the digit amputated. However, the relatively small sample size for fAMPs may have masked any apparent differences between fAMPs and Controls.

Another possible explanation for the differing survival rates between hAMPs and fAMPs is the indication for amputation. Due to the retrospective nature of the study this information was not available, however it is hypothesised that more fAMPs are performed due to toe necrosis in comparison to hAMPs and thus a potential explanation is that because the site of infection is further from the amputation site it results in improved healing.



In this study 55% of hAMPs and 84% of fAMPs survived at least one year post-amputation. The figure for hAMPs is consistent with the findings of Starke and others (15) who reported 54% of cases remained in the study a year after amputation by disarticulation of the DIP. However, this was a small study (n=26) and since it did not involve controls it is not known what effect amputation had on normal culling rate.

Comparison of survival between hAMPs and Controls over the course of the study showed that Controls survive significantly longer in the herd, which is consistent with the findings of Bicalho and others (16), although the difference in survival times between the two groups was greater. Mean survival time post-amputation for both fAMPs and hAMPs was higher in the current study than the previously reported 13.5 month by Starke and others (15) and 20 months by Pesja and others (13). A possible explanation for the higher survival rate in this study is that surgical intervention was carried out sooner, that cases were selected more carefully due to veterinarian experience or improved recovery rates due to the site of amputation.

Survival following surgery, along with cost and welfare considerations, is one of the main factors involved when deciding whether to perform a digit amputation. Due to the multifactorial losses associated with lameness, not only should the cost of surgery be considered, but also the potential losses associated with reduced milk yield and, most importantly, replacement cost if the cow has to be culled following surgery. However, in most situations the cow would require culling if she did not undergo digit amputation. This study shows that survival following surgery is higher than most previous studies, with 55% of hAMPs and 84% of fAMPs surviving one year post-surgery with mean survival times of 22 and 36 months respectively. In some cases cows remained in the herd up to 6 years after having a digit amputation. This provides a strong argument for digit amputation as a treatment for end-stage conditions of the digit.

## CONCLUSIONS

Lameness is not considered a life-threatening condition in cattle and therefore many lame cows are only examined by veterinarians once initial treatment administered by the farmer has failed. At this stage disease has commonly progressed to such an extent that slaughter or salvage procedures are the only options available. This study shows that digit amputation can provide a successful alternative to slaughter and that cows can be expected to remain in the herd for a considerable period of time.

## ACKNOWLEDGEMENTS

The author would like to thank the vets and clients of both Nantwich Farm Vets and Lambert, Leonard & May for all their help with data collection and also for their enthusiasm for the study without which it would not have been possible.

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## NOTES



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# POSTERS



## HOW TO HELP FARMERS MAKE CHANGES ON FARM

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### INTRODUCTION

Traditionally veterinary medicine has focused on the 'technical' aspects of animal health and welfare, and especially to promoting our knowledge and understanding of disease aetiology, pathogenesis, treatments, and preventive measures. In addition, more recently, there has been increased recognition of the importance of 'knowledge transfer'. However real improvements in cattle lameness will only occur if farmers take action and implement management changes on their farms; this requires them to change their behaviour and working routines. Unfortunately, there is substantial evidence to suggest that possession of knowledge per se is insufficient to generate human behavioural change; thus whilst it is vital to continue in our pursuit of further knowledge regarding lameness control, and to become more proficient at transferring that knowledge to farmers, this alone is far from sufficient. It is crucial that veterinary practitioners understand human behaviour and learn how to influence it, so that they can be as effective as possible at helping farmers to take action and reduce lameness.

In the absence of any formal appreciation of the subject of human behavioural science, as Homo sapiens ourselves, we often have a natural tendency to semi or sub-consciously make assumptions about the behaviour of our fellow colleagues, based upon our own perceptions, thought processes, values and understanding of the world around us; but these can often be wrong, and by making false assumptions (and by becoming frustrated when action isn't taken) we can in fact, unintentionally, serve to hamper the uptake of management changes on farm.

The aim of this study was to help practitioners understand how to encourage and assist farmers to make changes on farm, by applying concepts sourced from the human behavioural sciences, in order to promote understanding and provide inspiration.

### METHODS

A literature search of the human behavioural health sciences (see for example, Conner and Norman, 2005) and related disciplines such as social marketing (McKenzie-Mohr and Smith, 2000) was conducted. Successful application of these methods in a herd health context by animal welfare scientists (see for example, Whay and Main, 2009) was also reviewed. This information was then combined with a critical appraisal of (and reflection upon) years of experience in farm practice, in order to propose a clinically useful (and simple) framework for understanding farmer behaviour in a herd health context. Thereafter, a clinical assessment of the key factors involved at different stages of the framework was made, in order to help practitioners objectively identify, understand, and hence target, their efforts to helping farmers make changes as effectively as possible.

### RESULTS

Numerous models exist that attempt to explain human health behaviour, but none are perfect. Our poster summarises the framework that we consider the simplest, yet most clinically useful, when human behaviour health science is applied to a dairy herd health setting. It is a four point 'stage model' combined with the 'theory of planned behaviour' Ajzen (1985); the latter is not shown separately for brevity. In addition, we have also summarised some key aspects of our clinical assessment. Detailed explanation of the behavioural model and clinical assessment presented in this poster can be found in Higgins et al (in press).

## FUTURE WORK

There is considerable scope to use the framework and the clinical assessment presented in this study as a basis to gather empirical data and explore the issues we discuss, along with the optimal behavioural interventions that can help facilitate farmers to make changes in the future. For example, clinical trials aimed at reducing lameness (or any disease) could simultaneously capture social science data regarding the stage of change of the farmer at the outset, objectively quantify the key issues required to make progress, and the outcome of any measures taken to initiate and propagate change.

In addition it is clear that the success of veterinary education hinges entirely on whether or not we can help farmers take action, when required. Yet, as the most highly evolved creatures on the planet, the behaviour of *Homo sapiens* is incredibly complex to both understand and influence. Thus whilst it is acknowledged that veterinary curriculums are burgeoning, there is still a very strong case, and urgent need, to teach applied human behavioural science to veterinary students; we need to truly embrace the fact that we are the 'people's profession' and go beyond just teaching communication/personal skills.

## CONCLUSION

It is unfortunate, but unlike most clinical skills, our abilities to help farmers make changes on farm do not necessarily improve with clinical experience; in fact, our abilities can all too easily deteriorate over time. Farmers who are slowest to embrace change are the most likely to go out of business, and it is these farmers who are in greatest need of an advisor who understands human behaviour and who is therefore best placed to help them to take action.

## ACKNOWLEDGMENTS

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## THE RELATIONSHIP BETWEEN DAIRY COW BEHAVIOUR IN A CUBICLE HOUSE AND SUSCEPTIBILITY TO DIGITAL DERMATITIS

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### INTRODUCTION

Digital dermatitis (DD) is widespread on UK dairy farms and is a major cause of lameness (1). Prevalence of the disease appears to be influenced by housing and management factors, with groups that have more contact with slurry experiencing a higher prevalence of DD (2). However, it has been found that some animals within a group do not develop the disease whilst others may be infected repeatedly (2). The impact of an animal's behaviour on their susceptibility to digital dermatitis does not appear to have been examined in detail previously. The aim of this study was to determine if there is a relationship between the behaviour of individual dairy cows and their subsequent susceptibility to DD during that lactation.

### MATERIAL AND METHODS

Fifty Holstein-Friesian dairy cows were selected from a group of 90 cows housed in an automatically-scraped, concrete-floored cubicle house. Animals were selected on the basis of similar calving date (mean  $\pm$ SE dpp at the start of the study =  $45 \pm 2.4$ ) and no history of DD or heel horn erosion during the current lactation. Animals were fed a total mixed ration diet with additional concentrates fed through out-of-parlour feeders (OPFs). All animals were on a concurrent feeding study consisting of three different concentrate levels. The cubicle:cow ratio was maintained at a minimum of 1.1:1 during the study.

Behavioural observations were conducting at the start of the study and two months later (periods 1 and 2). In each observation period animals were observed for 3.5 hours each day for eight consecutive days. These observations took place within the following time slots: Days 1,5 = 09:30 – 13:00 hours; Days 2,6 = 17:30 - 21:00 hours; Day 3,7 = 21:30 - 01:00 hours and Days 4,8 = 01:30 - 05:00 hours. The behaviour (Table 1) of each animal was recorded by scan sampling every ten minutes during observations. The number of scans when the animal performed each behaviour was calculated for each period.

Table 1 Behavioural categories used in observations

Category	Definition
Lying in cubicle	Animal lying in cubicle
Standing in cubicle	Standing with all four feet in the cubicle
Standing half in cubicle	Standing with front feet in cubicle and rear feet in passageway
Feeding	Feeding from either OPFs or feed face
Passageway	Standing or walking in a passageway
Crosswalk	Standing or walking in crosswalk
At feed face	Standing or walking at the feed face
Hind feet in slurry	Sum of all behaviours except lying and standing in cubicle

Animals were monitored weekly from two weeks post calving for the presence of DD (on back legs only) while milked in a rotary milking parlour. Animals showing DD, lameness, mastitis or other health problems during the first behavioural observation period were eliminated from the analysis, leaving a total of 35 animals (of which 25 developed DD).

Data were analysed using Genstat v14 (VSN International, 2011) Each behavioural parameter was analysed separately using a multivariate logistic regression model where future occurrence of DD (0/1) was the dependent variable and parity, feeding study

treatment and number of scans when an animal performed the behaviour of interest were independent variables. Consistency of behaviour between periods 1 and 2 was examined using Spearman's rank correlation coefficient.

## RESULTS

Behaviour in all but one category showed a significant positive correlation between the two observation periods (data not shown; for category 'Crosswalk' a non-significant positive trend was shown ( $p=0.095$ ), for all other behaviours  $p<0.05$ ). The results of the logistic regression analysis (Table 2) showed that animals that spent more time 'standing half in cubicle' and 'in passageways' were more likely to develop DD. Occurrence of the other behaviours did not affect the occurrence of DD.

Table 2 Relationship between behaviours performed in period 1 and development of DD

Behaviour category	Sessions when behaviour was observed (%)			Results of logistic regression
	No DD	Developed DD	SE	
Standing half in cubicle	5.19	14.23	2.02	$p<0.001$
Standing in cubicle	4.06	4.90	1.62	$p = 0.557$
Lying in cubicle	55.81	53.48	2.50	$p = 0.497$
Feeding	19.42	14.94	1.20	$p = 0.353$
Standing/walking at feed face	7.90	4.25	1.69	$p = 0.128$
Time in crosswalks	2.23	2.30	0.70	$p = 0.605$
Time in passageways	5.35	5.88	0.65	$p = 0.015$
Hind feet in slurry total	40.13	41.60	2.44	$p = 0.273$

## DISCUSSION AND CONCLUSIONS

In this study, animals that spent more time standing half-way into a cubicle and those that spent more time in passageways were more likely to develop DD during that lactation. Standing half in a cubicle and in passageways may increase the amount of time when the heels are in contact with slurry, which has previously been linked with DD prevalence (2). The results of this study suggest that individual differences in the behaviour of dairy cows affect the likelihood of developing digital dermatitis.

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## ASSUREWEL: EMBEDDING MOBILITY SCORING INTO FARM ASSURANCE SCHEMES

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Farm assurance schemes in the UK aim to provide assurance to consumers and retailers of compliance with welfare standards. Standards are primarily set on the provision of input resources deemed necessary to satisfy welfare. There is a growing body of evidence that supports the use of welfare outcome assessment to objectively assess welfare using a set of animal based measures.

AssureWel is a five year collaborative project involving the University of Bristol, RSPCA and Soil Association that aims to deliver optimal welfare assurance within Freedom Food and Soil Association schemes using outcome assessment, as well as promoting the uptake of this approach within other assurance schemes. In order to select the most appropriate measures and define sampling strategies that are feasible and robust within a certification context, a consultation exercise and pilot study has been carried out.

The set of inspectable measures includes, individual measures (mobility, body condition, hair loss and lesions, and dirtiness), herd measures (lameness management, broken tails, lying comfort, response to stockman, cows requiring further care), and record measures (mobility – verification of self-assessment, mastitis, heifer survivability and cull and casualty cows). Training and standardisation for assessors will ensure the delivery of a consistent evaluation. This approach is being introduced into Freedom Food and Soil Association schemes in 2012. AssureWel is working with Red Tractor to support the implementation of the majority of these measures in 2013.

An advisory service is available to promote positive husbandry changes which are tailored to farm specific requirements. This approach provides a mechanism for welfare improvement by highlighting and focusing on farm specific areas for improvement, as well as promoting the uptake of outcome monitoring by farmers as a routine management tool.

This poster will describe with, specific reference to lameness management, how, an assessor mobility scores cattle with the farmer at inspection; including a discussion of on farm monitoring and treatment of cows, which will become an integrated part of the inspection process to reduce lameness across the UK dairy herd.



## FOOTBATHING STRATEGIES FOR DIGITAL DERMATITIS CONTROL

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### INTRODUCTION

Digital dermatitis (DD) remains the main cause of infectious lameness. The exact aetiology of DD is still unknown, but several bacteria, spirochaetes in particular, have been implicated (1). Therefore, regular footbathing of the cows with an antimicrobial solution is often viewed as an essential part of the prevention and control of DD. Using a pre-rinse bath before a treatment bath in order to reduce contamination of the solution and maintain its effectiveness has been recommended as good practice (2). Furthermore, footbath length is important; the longer the footbath, the more contact time between cows' feet and the antimicrobial footbath solution. A footbath length of 3-4 metres has been advised to ensure that cows take at least 3-4 steps in the treatment footbath (2). However, it is not always possible to follow both recommendations. In addition, commonly-used footbath solutions such as copper sulphate are potentially damaging to the environment, and efforts should be made to ensure that they are used in the most efficient manner possible. Therefore the aim of this study was to compare the effectiveness of two footbathing regimes in the control of digital dermatitis; 1) a pre-wash footbath containing water followed by 5% copper sulphate treatment bath, 2) two treatment baths of 2.5% copper sulphate.

### MATERIALS AND METHODS

This study was conducted during the winter housing period (2010-2011) with 73 lactating Holstein-Friesian dairy cows. All cows were footbathed after (twice-daily) milking on 4 consecutive occasions each week using 2 commercial split footbaths over a 13 week period. Each compartment of the footbaths was 233 cm long and filled to a depth of 15 cm, giving a volume of 60 L. There was a distance of approximately 125 cm between the 2 footbaths. Each side of the footbaths constituted a treatment as follows: 1) Left side: pre-rinse bath (i.e. water) followed by a 5% copper sulphate treatment bath, and 2) Right side: both 2.5% copper sulphate treatment baths. Digital dermatitis was scored on the hind claws of all animals during milking on a weekly basis using a 5-point nominal scale developed by Döpfer et al. (4): M0 = no DD lesions; M1 = early stage DD lesion, 0.5-2.0 cm; M2 = classical ulcerative stage DD lesion, >2 cm; M3 = healing stage DD lesion, covered by a scab; M4 = chronic stage DD lesion, characterised by dyskeratosis or proliferation of the surface. The effect of treatment on the proportion of hind feet in different DD lesion stages was analysed in Genstat 12 using a Generalised Linear Mixed Model (GLMM) analysis. To determine the degree of healing, the transitions of DD lesions were defined as binomial data and scored as 'healed' when lesions were improving on both hind feet (i.e. M1, M2, and M4 lesions followed by M3 or M0 lesions), and as 'not healed' when 1 or both hind feet had lesions that were either getting worse or not improving from week to week.

### RESULTS

Both treatments had high occurrences of M0 and M3 lesions, and very low occurrences of M1, M2, and M4 DD lesions during the study period (Figures 1 and 2). There were no significant treatment differences found during the study in the proportion of hind feet with lesions of different stages ( $P>0.05$ ). In addition, the proportion of hind feet that showed evidence of healing lesions, did not differ between treatments ( $P>0.05$ ). In total, there were 650 and 654 transitions between the different DD lesion stages recorded during the study on the left and right hind feet, respectively.

Figure 1: Proportions of different digital dermatitis lesion stages (M0 to M4) observed over the study period when a pre-rinse (water) bath was followed by a 5% copper sulphate footbath

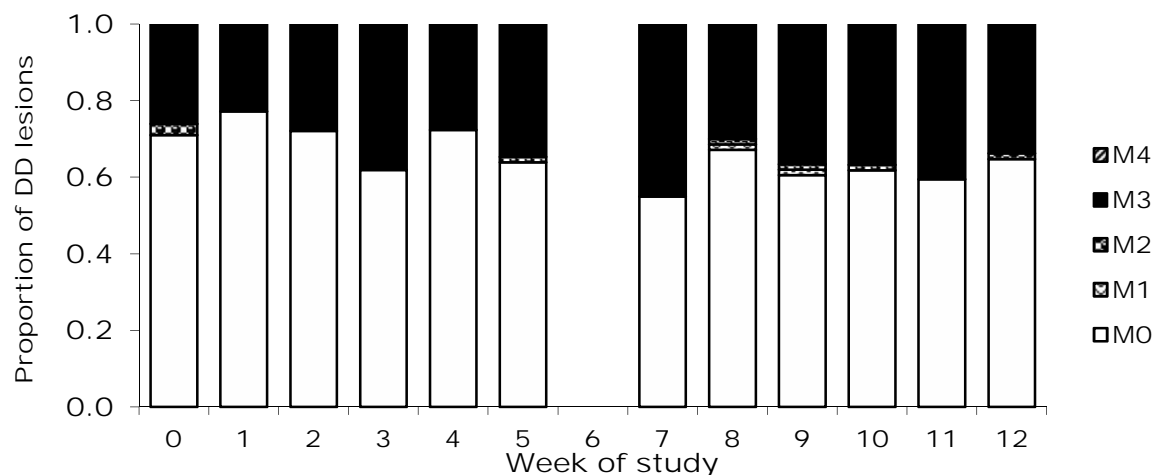
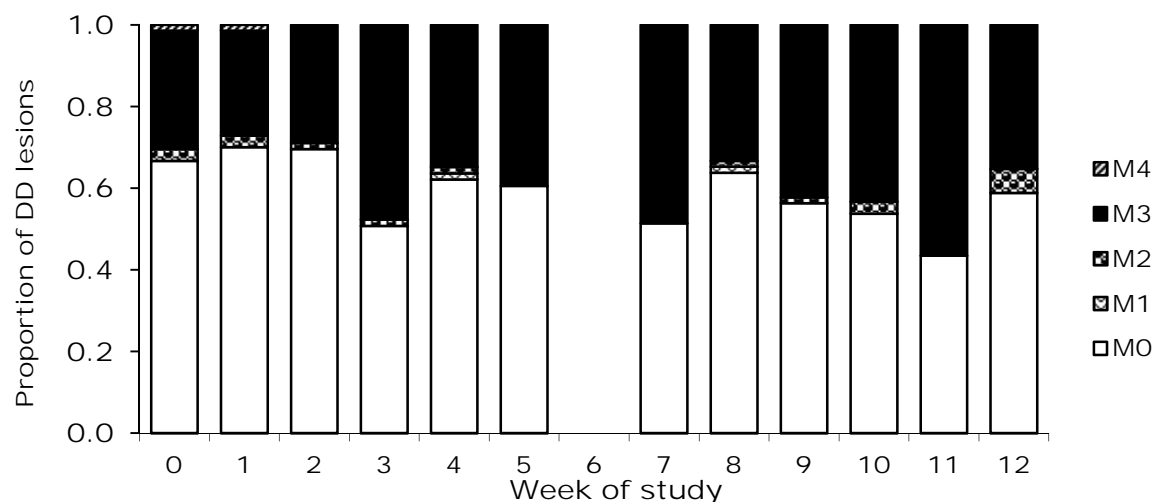


Figure 2: Proportions of different digital dermatitis lesion stages (M0 to M4) observed over the study period for when cows walk through two sequentially placed 2.5% copper sulphate footbaths



## CONCLUSIONS

There does not appear to be any benefit in the control of DD by splitting a copper sulphate solution between two successive footbaths (each with 2.5%) rather than having a pre-rinse bath (with water) followed by a treatment bath (with 5% copper sulphate).

## ACKNOWLEDGEMENTS

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## A PRELIMINARY STUDY OF PEDAL BONE CHANGES IN AMPUTATED DIGITS

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This preliminary study examines changes in pedal bones taken from 33 amputated claws in 2011 and 36 claws (18 feet) chosen at random from a cull cow abattoir. The major changes seen are listed below:

1. Pedal bones from abattoir claws showed a variation of:
  - i) length from 62 to 115 mm;
  - ii) width from 25 to 33 mm and in height (extensor tuberosity to solear surface) from 33 to 55mm.

This needs to be repeated with a standardised approach to measurements. It is hypothesised that such a large variation in pedal bone size should be considered when establishing standards for hoof trimming.

2. The major indication for amputation of digits was non-healing hoof lesions, most probably associated with digital dermatitis and other organisms. This indication is not mentioned in standard texts dealing with digit amputation, where joint infection is given as the most common cause.
3. Pedal bones from digits amputated as a result of non-healing lesions commonly exhibited a proliferative osteitis, especially over the solear surface, and it is hypothesised that this could lead to further non-healing.
4. Other proliferative osteitis lesions commonly included exostoses on the extensor process and fusion of the navicular bone with the pedal bone. It is hypothesised that both changes will lead to some gait impairment
5. Pedal bones from older cows, and from cows with sole ulcers, developed an overgrowth of the flexor tuberosity. It is hypothesised that this will predispose to further ulcers, and hence the use of blocks, and especially shoes that take weight on the hoof wall, should be used more frequently. This finding needs further examination.

This poster will be presented using actual bone and hoof specimens.





## NOTES

