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11th - 15th FEBRUARY 2004
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MARIBOR, SLOVENIJA

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FOREWORD

At the 9th International Symposium on Lameness in Ruminants in Jerusalem we made a bid for the organisation of the next symposium. It was a close vote, which we lost to Luzern by only 3 votes. I was not too disappointed because I knew that it would be a miracle at the time, had colleagues decided for Slovenia, which was a new country just a few miles away from the Balkan tragedy. Then 4 years later, while in Parma, my colleagues started to persuade me that Slovenia should be the venue in 2004. Four years ago I was much more self-conscious, but accepted the challenge fdor organisation of the 13th International Symposium and 5 Conference on Lameness in Ruminants. The next challenge was the time and place for the event. Christoph Lischer said at the business meeting in Parma"we have never had a meeting in the wintertime !" Marvellous ! In Maribor, my home town, we have the longest illuminated ski slope in Middle Europe and in 2003, just 100 metres away will be the newly built Congress and Convention Centre. What an opportunity for us ! The decision was made. We would organise our Lameness Symposium in Maribor in the winter time !
The decision about the motto for the Symposium and Conference was probably the easiest one. Everybody knows that "PREVENTION IS BETTER THAN CURE" and so this phrase was adopted as the theme of the event.
The next step was the membership of the Scientific Committee. The decision was easy, and the only problem seemed to me to be whether the nominated persons would accept. And again I was lucky ! All agreed, and all did their job superbly. I made it even harder, as I sent them all the material without disclosing the authors of the papers, and I sent each paper to at least two members of the Committee. In that way they made really the best decisions about the submitted material.
We received 112 abstracts for scrutiny. The Committee accepted about 70 oral and 28 poster presentations. Then it was my turn to to arrange groups and to fix the time scale. I was happy to work with colleagues all willing to put their knowledge and expertise at my disposal. And a great friend said that when he realised what kind of event we try to organise, anybody who will miss the event could "kick himself in the knee". That was my greatest compliment. I hope that we succeeded in organising a good scientific event, including the new "meet the professor" sesssions, and that everyone will have great memories of Maribor and our small but beautiful country, where you can experience such a wide variety of scenery.
But we would never have been able to organise this scientific event and meeting of friends without the great support of three principal sponsors, ZINPRO Company, VETTEC and ECOLAB, and all the other gold, silver and bronze sponsors, who participate with their commitment and resources. But even my practice, though not making the biggest donation, probably played the most important part in this story. They helped me when I most needed it, and they gave my continual support in the preparations for 2004. Finally, it is necessary to admit that without my great family in its widest sense, I would not have been capable of organising this event, and as always behind every man is the woman who always supports three angles of the house you build. It is true that we often had different opinions and separate views about the organisation, but finally everyone did his or her important bit in creating the final picture.

Borut Zemljič, Dr.Vet.Med., MSc., PhD
President of the Organising Committee

12th INTERNATIONAL SYMPOSIUM ON LAMENESS IN RUMINANTS
9-13 January 2002
Marriott World Center, Orlando, Florida

Minutes of the Business Meeting held in Orlando on Sunday 13, January 2002 12:45 PM

Chairman:	Professor A. David Weaver
Recording Secretary:	Dr. J.K. Shearer
Present:	~ 50 (estimated)

1. Meeting was called to order by A. David Weaver at approximately12:45 PM.

1.1 Professor Weaver opened the meeting with a request for "Apologies for Absence":

1) Dr. Dorte Dopfer was unable to attend the Symposium as she is pregnant and near parturition.
2) Dr. David Logue was also unable to attend as a result of work-related demands.

1.2 First order of business was review of the Minutes from the 11th International Symposium held in Parma. Minutes were accepted as presented in the proceedings. Professor Weaver acknowledged that having minutes from the previous meeting published in the proceedings was useful and recommended that this be considered by the organizers of future Lameness Symposia.

2. Review of Matters Arising from the 11th International Symposium in Parma

2.1 Lameness Data Capture - no action at this time

2.2 Locomotion Scoring - no action at this time

2.3 Proceedings of the Rebild 1992 meeting - Professor Weaver indicated that there has been no response from Dr. Karen Mortensen (organizer of the Rebild Symposium) regarding her promise to publish the proceedings from this meeting. It appears that all hope for having these published is lost.

2.4 Communication between meetings via email or newsletter - The issue of a newsletter was raised but as in times past no decisive action for preparation of such was taken. Discussion continued on the topic of sharing or publishing the contact information of symposium participants. It was acknowledged that some consider this confidential information and preferred not to have such information published. Therefore, it was agreed that contact information remain confidential and used only by Symposium organizers for the purpose of communications relative to preparation and promotion of upcoming Symposia. Contact information, such as mailing lists or email addresses, will not be shared with commercial interests.

3. Matters Arising

3.1 Disposition of Proceedings from the Lameness Symposium - There was general agreement that this group represents the expertise of the world in terms of lameness in ruminants. Therefore, the proceedings of these meetings are of great value, not only to participants of this Symposium, but to scientists, veterinary clinicians, hoof trimmers, and others throughout the world. There was much discussion about how to make information from the proceedings available to larger audience. Dr. Guard suggested offering a copy of the proceedings or a CD to veterinary college libraries throughout the world. Dr. Greenough inquired about the publishing of material from the proceedings on his web site. All agreed that these were good ideas but no official action was taken.

Professor Weaver suggested that we have the proceedings listed with Index Veterinarius and that he would contact the British Cattle Veterinary Association to make an announcement about how interested persons could obtain a copy of the proceedings. Dr. Shearer agreed to contact the American Association of Bovine Practitioners (AABP) and the Journal of the American Veterinary Medical Association (JAVMA) with contact information for persons desiring a copy of the proceedings.

Dr. Sarel van Amstel suggested that the group consider refereeing the proceedings. General consensus of the group was not favorable toward this idea. Most preferred instead to publicize information about how to purchase copies of the proceedings or CDs.

Dr. Roger Blowey offered a comment that it might be useful to separate the duties of preparation of the proceedings from organization of the Symposium. Continued growth of the Symposium makes the management of both a very time consuming effort.

4. Maribor, Slovenia -Site for the 13th International Symposium in 2004

4.1 Dr. Borut Zemjlic reported on the venue for the 13th Symposium in Slovenia. The meeting will be held February 11-15, 2004, in Maribor, Slovenia at the Habakuk Hotel and Convention Center. The hotel is located at the base of a mountain with great skiing all around. He promises it will be a meeting to remember. Although he did not elaborate due to time constraints, Dr. Zemjlic said he is planning a meeting with a different format than that held in Orlando, one that will encourage greater interaction.

5. Proposed Venues for the 14th International Symposium in 2006

5.1 Professor Weaver opened the floor for offers to host the 2006 International Symposium. There were 2 offers; Dr. Jouni Nemi, from Finland and Dr. Roberto Acuna from Uruguay. Vote by a show of hands was taken - Uruguay received 30 votes to Finland's 17. Therefore, site for the 2006 Symposium will be Uruguay most likely in November or December.

6. Other Business

6.1 A question was raised as to a possible web site for information relative to the 13th Symposium. Dr. Mike Socha proposed that the web site used for the 12th International Symposium be maintained and officially licensed for use by future Symposium organizers and participants. Hearing no dissenting opinions, Dr. Socha's proposal was accepted. Therefore, the official web site for information concerning the International Symposium on Lameness in Ruminants shall be: ruminantlameness.com

6.2 Dr. Paul Greenough inquired about official transfer of the "cow bell". Dr. Shearer explained that the official transfer of the bell would occur later in the program following Dr. Borut Zemjlic's formal presentation on the 13th Symposium in Maribor, Slovenia.

6.3 As it was time to begin the afternoon sessions, the business meeting was adjourned by Professor Weaver.

Respectfully,

J.K. Shearer
Recording Secretary

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3. Session: Biomechanics of the ruminant claw

6. Session: Infectious diseases of acropodium in ruminants

7. Session: Animal welfare and claw diseases

8. Session: Nutrition and claw health

10. Session: Cotnrol of lameness in smal ruminants

11. Miscellaneous

RESULTS OF LAMENESS STUDIES AND GETTING ADVICE TO DAIRY PRODUCERS

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"We are not exercising the vision needed or being sufficiently proactive to put that knowledge to work on the farm" said Paul Greenough in Orlando in 2002. Putting it more simply, our research and investigational results are not arriving on the dairy farm, either as preventive measures -the theme of our Maribor meeting - or as therapy. Why ?

The symposia of our meetings are abstracted by CABI (Commonwealth Agricultural Bureaux International), many of the papers are submitted shortly after oral presentation here for publication in refereed journals, and some symposia papers may be highlighted in lay publications (e.g. "Hoards Dairyman" in the USA and "Dairy Farmer" in the UK)and in sundry non-refereed publications. Some papers are put onto personal (e.g. Paul Greenough www.cow.doc.net) and paraprofessional websites such as the UK foot trimmers group (www.nacft.co.uk). Several other countries will have similar arrangements. Some material is filtered out for use by commercial companies, for example by Zinpro, a major sponsor of this meeting.

This organisation has biennial meetings and has twice unsuccessfully tried to establish a regular newsletter for communication between meetings. Now the arrival and widespread adoption of the internet has altered the vision of rapid communication between interested parties in that the printed word or hard copy has been overtaken as the desirable medium for the dissemination of new information. But is it effective down on the farm in that herd of lame cows ?

Thinking of the future, and as far as the UK is concerned, unfortunately today most veterinary and dairy science undergraduates are unlikely to hear much about the advances (for example in the control of digital dermatitis) unless fortunate enough to have some extramural education ("seeing practice") with a Maribor participant. Also, in the UK anyway, the government veterinary service has little time or manpower available for farm advice, since their numbers are still lower than at the time of the 2001 FMD outbreak. So expert advice on lameness problems in dairy herds is unlikely to come from this source, but rather from informed cattle practitioners. We must find ways of getting the current (2004) knowledge across to farmers, and it can only come from enthusiastic colleagues in practice, some of them, but too few, here today. They are ideally placed to utilise this information on the farm next week.

It has been repeatedly shown that on UK dairy units farmers fail to recognise the true prevalence of lame cows in their herds. A typical estimate, based on Becky Whay's published data, may be 6% by the farmer, but 22% by an independent observer who looks at every cow exiting the milking parlour one afternoon. The reasons for this discrepancy have been much discussed and remain unclear, but psychological reasons cannot be ruled out. These possibilities include a natural reluctance to check all the cows systematically and an acquired desensitisation to the real situation. Clearly pressure caused by the increased herd size and shortage of labour, and therefore of time, also play their part. Also, despite numerous statements on the financial costs involved, many farmers appear not to appreciate the milk production loss and the increased calving interval. This latter information is readily available in the long-established recording schemes such as DAISY, started by Dick Esslemont and his colleagues in Reading. But such schemes were not designed to incorporate the latest research information.

But there is some light, and hope, in the gloom of problem lameness herds, and getting information across. One UK veterinary group (Kingfisher, Ilminster, Somerset) takes a stand at a major South West English agricultural show in order to teach farmers about lameness prevention and hooftrimming. They employ their own practice trimmer, and he is there on the stand with his veterinary colleagues. This practice is one of several in the UK to run regular training courses for dairy farmers. The Milk Development Council, supported by a levy from all dairy farmers, is happy to pay for groups to learn about lameness management from a speaker, usually a vet, of the participants' own choice. But again , does the latest information get put across, and get onto an action plan ? Farmers discussion groups, dairy farm walks and evening veterinary meetings to which farmers are invited, rarely present new information relevant to the huge problem of lameness prevention.

The role of the foot-trimmer has been a major concern in the UK over the last years. A paper presented to its national association (NACFT) in 1999 entitled "When the trimmer stops and the vet takes over" caused heated discussion. Some months ago DEFRA (Department of the Environment, Food and Rural Affairs, the successor to the Ministry of Agriculture, Fisheries and Food) invited national discussion about a potential new Veterinary Surgeons Act (VSA) in 3-4 years time to replace the current VSA of 1966. One topic on which suggestions and proposals are sought is the role of the paraprofessional in veterinary work. The NACFT has proposed to DEFRA that it should be licenced by DEFRA under any new VSA, and that only qualified trimmers should be licenced. Today it is estimated that only one quarter of active trimmers are NACFT members (about 70). They are bound by a constitution, code of conduct, training courses and examinations, and subsequently by regular assessment every two years.

The NACFT claims, I believe correctly, that "foot trimmers have superseded vets as the main carers of cows feet, including the lame cows" . Yet currently, under UK legislation in the VSA of 1966, the trimmer cannot legally diagnose foot disease as it is "an act of diagnosis", the prerogative of the veterinarian. Similarly the NACFT also believes the act of treatment, currently in veterinary hands, should be extended to the trimmer, in so far as he would no longer be breaking the law in putting aa block on the medial hind claw to relieve the lateral claw with its painful sole ulcer.

Regardless of the eventual form of the new VSA, it seems vital that there be ever closer links between foot trimmer and veterinarian, preferably by joint visits, examination of good farm records, and joint recommendations, even though it seems that most trimmers are unwilling to work financially under direct veterinary control. The NACFT members are happy to work alongside vets, and I envisage a routine veterinary herd health visit coupled with foot trimming. Both interested parties should first take the opportunity initially, with the dairy personnel of looking at the entire milking herd walking past. This can be repeated every three months..The routine individual cow work is then done. At the end, with the problem cases checked and managed by the veterinarian, a half hour session round the the kitchen table, with the ubiquitous mugs of hot tea, permits discussion of further preventive measures, Which should always be written down (3 copies !) For action before the next joint farm visit. This half hour could be the most important step in pushing the prevalence of lameness down below 5%.

Conclusions

Current information and recommendations from our biennial international symposia rarely reach the dairy farmer. Yet we have increasingly sophisticated recording systems, email and internet "indigestion". The farm veterinarian should have easy access to this information, so as to digest it for the individual dairy farmer client, who finds it hard to institute necessary changes in management. More practitioners should attend our meetings ! Improved communication between foot trimmer ("the main carer of cattle feet"), veterinarian and farmer on a regular basis should be demonstrably good practice. The implementation of their joint control measures, agreed and noted down by the three parties, and naturally enhanced whenever necessary by other advisers, should become the cornerstone of good management.

HERD LAMENESS - A REVIEW, MAJOR CAUSAL FACTORS, AND GUIDELINES FOR PREVENTION AND CONTROL

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INTRODUCTION

Today's dairy cows face many different environmental and management challenges than the cows of the past. High energy rations, confinement on concrete, constant exposure to corrosive conditions, conformation defects, and perhaps even increased body size are some of the risk factors that increase the probability of lameness. However, despite an increased awareness of these factors, high levels of lameness in dairy herds have persisted into the 21st century, and continue to be one of the largest financial drains to the dairy industry. Worldwide research indicates that as many as 60% of a herd may become lame at least once a year. Lameness also presents a specific animal welfare issue of concern, since pain and discomfort associated with lameness can be prolonged. It has been recognised that lameness has a negative impact on milk yield, both before and after diagnosis of the insult. Furthermore, a growing body of research suggests that early-lactation lameness, i.e. lameness that begins during the first 30 days of lactation hurts reproductive performance. Therefore, prevention of lameness will have a big impact on production and sub-fertility.
Over the past 20 years, there have been a number of studies based on survey information, which have attempted not only to quantify the extent of the lameness problem, but also to identify the most important lameness-causing conditions, and to define the major causal factors. There is a multitude of suggested causal factors associated with bovine lameness, which can be broadly classified into environmental (e.g., nutrition, infectious agents, management) and genetic (e.g., conformation) factors. When attempting to control lameness on a herd basis, there is a need to consider and, if so required, to pay attention to all these factors.

THE LAMENESS PROBLEM - AN OVERVIEW

The Amount of Lameness
Worldwide, numerous surveys have been carried out determining the incidence rates of lameness in dairy herds. Lameness incidence is usually calculated on an annual basis from the records of individual lameness treatments. Farm records are sometimes limited to lameness cases that require only antibiotic therapy, and hose cases that are treated by claw trimming are often not recorded (Whay et al, 2002). Surveys based on cases treated by veterinarians have reported a lower incidence of lameness, indicating that not all lame cows receive veterinary treatment. For example, on many farms in Australia and New Zealand all or most of the lameness cases are dealt with by the farmer or herdsman, and therefore these numbers under-estimate the problem. Some reported incidence rates are:

- Australia: 7% (0-50%); Victoria (Harris et al, 1988)
  2.7% (vet treated only); Queensland (McLennan, 1988)
  2.5% (vet treated only); Victoria (Jubb & Malmo, 1991)
- New Zealand: 14%; (Dewes, 1978)
  20.7% (2-38%); (Tranter & Morris, 1991)
- United Kingdom: 36% (1-94%); (Esslemont & Spincer, 1993)
  55% (11-170%); (Clarkson et al, 1996)
  38% (4-69%); (Kossaibati & Esslemont, 1999)
  69% (32-112%); (Hedges et al, 2001)
- USA: 46% (40-52%); New York (Warnick et al, 2001)
  31% (one herd only); Florida (Hernandez et al, 2002)

These figures indicate that there is a large range between farms, districts and countries, that there is a lot of lameness about, and that the incidence of lameness is probably increasing. Certainly there is a heightened awareness, and appropriate steps are now being taken by veterinarians in many dairying countries in an attempt to reduce the existing lameness problem. This often entails a multi-disciplinary approach, with the involvement of a nutritionist and farm building engineer.

The Types of Lameness

Most cases of lameness are due to claw lesions (up to 90%), the remainder being associated with (upper) limb problems. On average, approximately 80% of lame cows are lame in the hind limb(s). Since the cow takes about 60% of her weight on her front limbs, one would expect a greater percentage of front limb lameness. However, the hind limbs are also involved in propulsion, which causes much more stress and friction compared with only weight bearing. Also, hind claws have a smaller ground area than front claws, further exacerbating any stress.

Almost 75% of all claw lesions in the hind limb are found on the outer claw. This is due to the fact that the outer claw commonly carries far more weight than the inner claw, that the outer claws are often larger, and that there is a need for the hind limbs to circumvent the udder during walking.

In Australia and New Zealand, a relatively larger number of cows (especially first-lactation heifers) may be lame in the front limb(s) due to lesions in the inner claw. This may reflect the low ranking of heifers in the herd's social hierarchy of dominance. Heifers are usually milked last and, therefore, spend more time in the holding yard. To avoid confrontation with the more dominant, older cows, heifers are constantly backing off. By doing so, excessive force will be put on the inner front claws. Soiled concrete surfaces are extremely abrasive, and movement of animals over these surfaces is accompanied by harsh grinding noises. These effects are exaggerated during pushing and crowding of the herd (excessive use of the backing gate, which is often electrified), and by bulling cows.

The Common Types of Claw Lesions

The various disorders of the ruminant digit have been ascribed Latin terms, though other names are more often used in the field. The most common types of lesions vary between countries. For example:

- United Kingdom (Murray et al, 1996) - sole ulcer (36%), white line disease (22%), digital dermatitis (8%), interdigital necrobacillosis or proper foot rot (5%)

- New Zealand (Tranter & Morris, 1991) - bruising/excessive wear (42%), white line disease (39%), septic pododermatitis (under-run sole or sole abscess) (9%)

- Victoria, Australia (Jubb & Malmo, 1991) - axial wall crack (22%), septic pododermatitis (21%), interdigital necrobacillosis (13%), white line disease (7%), sole ulcer (4%)

- Victoria, Australia (Malmo, 2002: unpublished data) - white line disease (34%), septic pododermatitis (29%), axial wall crack (17%), interdigital necrobacil losis (9%), sole ulcer (6%)

Thin soles due to excessive wear are one of the most prevalent lameness problems in extensive grazing systems, such as in New Zealand and parts of Australia, and in large free-stall barn systems in North America. In the latter, much of the problem is clearly due to excessively abrasive concrete, wet feet, and use of sand bedding. It may also be due to overzealous functional and corrective claw trimming (Van Amstel et al, 2002), which is routinely carried out once or twice each year. In year-round, pasture-based grazing systems (Australasia), a major factor is the distance that cows have to walk, especially in the all-important peri-partum period, when horn growth stops and wear increases. Also, more lameness occurs in seasons with high rainfall, and a high number of lameness cases usually follow periods of heavy rain.

During the last decade, axial wall cracks and lesions associated with laminitis (i.e. poor claw horn quality (yellowish, waxy and soft), sole haemorrhages, sole ulcer and white line disease) have become more prevalent, both in Australia and New Zealand.

The Cost of Lameness

Lameness in modern confine
ment dairy herds has joined infertility and mastitis to become the third most important health problem in dairy production (Dürr et al, 1997). Overall, reproductive problems are the major cause of culling, and lameness will contribute indirectly to those culls. Clinical lameness causes direct and indirect economic losses. Direct losses are associated with the costs of treatment and the withholding of milk due to antibiotic therapy, prevention of lameness, premature involuntary culling, and increased replacement costs. Indirect losses are incurred through decreased milk production, reduced body condition and reproductive performance, and increased risk of mastitis. The veterinary or treatment costs are only a minor item in comparison to reduced fertility, production and cull costs.

A recent UK study showed that clinical lameness had a significant impact on milk production both before and after the diagnosis of the insult (Green et al, 2002). The total mean reduction in milk yield per 305-d lactation was approximately 360 kg or 1.2 kg per day. Similarly, research from Cornell University (New York, USA) showed that milk production losses due to lameness can be as high 1.5 l per day (Warnick et al, 2001). In another American study (Florida), interdigital phlegmon was associated with a 10% decrease in milk production (Hernandez et al, 2002). Lame cows with claw lesions or digital dermatitis also produced less milk than healthy cows.

Researchers at Michigan State University developed a five-point system to evaluate herd lameness (Sprecher et al, 1997). This Locomotion Scoring System assigns a score from 1 to 5 depending on the cows' gait and back posture. University of California (Davis) research (Robinson, 2001) has shown the following milk losses per score:

- Locomotion Score 3 (moderately lame) - 5.1% milk loss;

- Locomotion Score 4 (lame) - 16.8% milk loss;
- Locomotion Score 5 (very lame) - 36% milk loss.

A survey of 13 Dutch dairy herds indicated that there exist no relationship between lameness and reproduction (Bakerma et al, 1994). However, other studies have contradicted these findings, i.e. cows experiencing lameness had more reproductive problems, i.e. more days open (= longer calving to conception interval), lower conception rates and more ovarian cysts. Overall, lame cows have between 11 and 28 more days open than cows that are not lame (Argaez-Rodriguez et al, 1997; Collick et al, 1989). A University of Florida study (Hernandez et al, 2001) found that lame cows with claw lesions were only 0.52 times as likely to conceive as healthy cows. The median time to conception was 40 days longer and the number of services per conception significantly higher, when compared with healthy cows. According to another University of Florida study (Melendez et al, 2002), lameness also impacts fertility by reducing first-service conception rates, increasing the incidence of ovarian cysts, and decreasing pregnancy rates. A growing body of research suggests that early lactation lameness hurts reproductive performance the most. More than 30% of the cows that were lame during the first 30 days post-partum were culled before any reproductive event, compared to only 5.4% of the control cows. Research also indicates a direct correlation between locomotion scores of greater than 2 and reproductive problems, i.e. higher scoring cows show fewer heat signs.

The proportion of cows culled for locomotor disease may be as low as 2.7% in studies in France (Seegers et al, 1998). However, after reviewing the literature, Malmo and Vermunt (1998) estimated that approximately 5% of cows were being culled because of lameness. A survey in the USA by the National Animal Health Monitoring System (1996) reported that 15% of all dairy cow culling was directly due to lameness or injury. Lameness also contributed indirectly to reproductive failure and subsequent involuntary culling.
Lame cows have been shown to be more susceptible to other diseases, such as mastitis (Peeler et al, 1994). This association is likely to be indirect, because lame cows are lying down more often and for longer.

Results of these studies indicate that the more severe the lameness, the greater the economic loss. Various authors have calculated the total cost of lameness, including costs arising from loss of milk, loss of condition, reduction in fertility, cost of treatment, and culling. Examples of the average total cost of a single case of lameness are:

- Australia - Victoria: Aust $43 (Harris et al, 1988)
- Australia - Victoria: Aust $200 (Malmo & Vermunt, 1998)
- United Kingdom: Ł250 (Kossaibati & Esslemont, 1997)
- USA - New York: US $302 (Guard, 1997)

Put another way, in the United Kingdom, annual losses caused by lameness have been estimated to be about Ł90 million annually (Bennett et al, 1999). In the Netherlands, lameness in dairy cattle accounts for an estimated loss of 4% to 5% of the typical dairy farm income (Enting et al, 1997).

The Animal Welfare Implications

There is a widespread belief amongst the general public that cattle are relatively insensitive to pain (O'Callaghan, 2002). However, clinically lame cows suffer from behaviour-modifying pain and prolonged discomfort, which is a major welfare issue associated with modern dairy farming (FAWC, 1997). For example, Tranter and Morris (1991) found the average duration of lameness to be 27 ± 19 days!
For lame cattle, indications of pain are obvious in the changed gait of an animal, and the greater the disruption of normal movement, the more intense the pain is likely to be. The degree of pain, however, remains unknown.
Welfare aspects are not easy to measure, but attempts have been made to highlight some of the changes in behaviour of lame cows. Studies have shown that:

1. Lame cows spend more time lying down and show abnormal behaviour patterns of eating, ruminating and interactions with other animals (Singh et al, 1993a). They spend less time eating and graze more slowly, as measured by the bite rate (Hassall et al, 1993). Also, lame cows have a significantly lower number of meals each day compared with non-lame cows (Margerison et al, 2002). All of this means that they will lose weight, which is illustrated by the strong negative correlation between locomotion scores and body condition scores; condition scores decrease as locomotion scores increase (i.e. lame cows are thinner).

2. Lame cows are more sensitive to pain (Whay et al, 1997; 1998), which suggests that they are suffering considerably.

These observations clearly indicate that lame cows are badly affected by lameness. In addition to causing considerable pain, it is detrimental to production and fertility.

THE CAUSAL FACTORS

A shift in the causes of lameness in cattle has occurred over the past two decades, but progress in understanding the various causes of lameness has been slow and is still incomplete. Management, housing and feeding systems have changed to accommodate an increasing herd size and the production potential of the modern dairy cow. Cubicles (free stalls) are favoured over tie stalls and straw yards, and concrete has taken the place of pasture.
There is a growing awareness of the importance of investigation, diagnosis and control of herd lameness. As is the case with most production diseases, the cause of the problem is likely to be multi-factorial and often difficult to identify with total conviction.

THE MULTI-FACTOR CONCEPT

It is only in the last 25 years that the principles of preventive medicine have been applied to control cattle lameness. Also, there has been recognition that little was known of the risk factors that are associated with lameness. Over the last two decades or so, several epidemiological studies have identified the key factors contributing to lameness. A number of causal factors are thought to influence, either alone or in conjunction with one another, the severity and the prevalence of lameness. Recognising which factor(s) may be causing problems in an individual herd requires a systematic approach to the on-farm investigation, so relevant data are going to be collected and analyzed (Greenough & Vermunt, 1994). The ideal epidemiological methodology would evaluate potential stress in the animal, as well as the degree of exposure to the causal factor.

The various causal factors and their order of importance vary somewhat between countries. For example, in the United Kingdom, claw shape, genetics (breeding), claw trimming and nutrition rank high, whereas farm tracks and behaviour are further down on the list. In Australasia, where dairy cows are pasture fed all-year-round, tracks (raceways), dairy shed design (especially the holding yard) and herd management are considered to be the most important causal factors. However, the role of nutrition is increasingly recognised as a contributing factor in the aetiology of herd lameness (Westwood et al, 2003).
The more important factors are discussed below and recommendations, which may be of assistance in the control and prevention of herd lameness, are listed.

COW COMFORT

Numerous authors have stressed the importance of housing in the initiation of claw lesions. Clinical studies of herds affected by lameness have allowed a number of predisposing factors to be elucidated. These include, amongst others, sudden introduction to cubicles and concrete walking surfaces, lack of bedding and exercise, and poor cubicle and housing design.

Concrete The concrete surface on which cows walk and stand has received a great deal of attention. When smooth, it is slippery making footing tenuous, and when rough enough to give reasonable grip, it is very abrasive and causes damage to the horn. Rough concrete has been associated with higher levels of lameness than well-textured concrete (Wells et al, 1995). Severe problems of excessive wear may arise on new concrete, which is often extremely abrasive. Also, compression of the sole corium is directly related to the amount of time that cows spend standing, in particular where surface conditions are unyielding, as with concrete. To counteract the problems associated with concrete, the installation of rubberised walking surfaces in feed alleys and passageways has found some favour in modern dairy barn design. However, the benefits of this have yet to be proven (Vokey et al, 2002).

For optimum slip-resistance, concrete floors for dairy cattle should be finished so that they have parallel grooves 10 mm wide running perpendicular to the main walking direction of the cattle and spaced at 40-mm intervals (Dumelow & Albutt, 1990). If the walking direction of the cattle is difficult to predict, a pattern of hexagons with 46-mm sides formed by 10-mm grooves is best. A simple and equally effective grooving widespread in the UK is a rectangular pattern about 50 to 60 mm square and 10 mm deep (Bickert & Cermak, 1997). A smooth, slippery concrete floor can be improved by either a new, roughened surface or by cutting grooves as mentioned above. A recent Dutch study showed that cows in straw yards had by far the lowest number of claw disorders when compared to cows housed on solid concrete floors, slatted floors or managed in a zero-grazing system (Somers et al, 2003).

Cubicle Design The use of cubicles is related to their comfort. Holstein-Friesian cows on pasture need 240 cm x 120 cm lying space and a lunging space for rising of at least 60 cm (Faull et al, 1996). By these standards of space requirements the majority of cubicles may be either too short, too wide or too narrow. Therefore, resting time will be adversely affected in situations where cubicle partitions are poorly designed and/or cubicle dimensions do not meet the space requirements of the animal. The occupancy rate of the Newton Rigg cubicles by in-calf heifers was less than observed for the Dutch Comfort cubicles and, after calving, claw health deteriorated less rapid in the animals housed in the latter (Leonard et al, 1994).
Proposals for adequate cubicle dimensions have been suggested by Bickert and Cermak (1997). Length of the cubicle is probably the most important dimension and should be relative to the size of the cow and her dynamic space requirement. These authors reported that a 600 kg Friesian dairy cow has a forward space demand of 0.7 to 1.0 m to allow her to lunge forward when rising. A large dairy cow needs a cubicle length of 2.4 m. Width requirements are, to a certain extent, dependent on the cubicle design, as narrow cubicles can be partly offset by divisions which allow "space sharing". Such cubicles provide three areas of free space, i.e. for the head, ribcage and the loin-rump area. Ideally, the width of the cubicle should be 115 cm for heifers and 120 cm for mature cows. The material used for the base and bedding of the cubicle also has a profound effect on the lying time of the cow (see below).

Bedding The importance of a soft resting area in relation to lameness has been well recognised. There is a growing body of evidence that increased lying times have a beneficial effect on lameness prevalence and claw health. If a cow can lie and rise easily, and its bed is comfortable, it will more likely use cubicles. Soft bedding results in longer resting times and less lameness, thus supporting the importance of the burden factor (compressive stress or loading). Broom and Galindo (1997) suggested that dairy cows should lie down for 9 to 14 hours per day, whether at pasture or housed in free stalls. More recently, it has been reported that lying times of cattle on pasture ranged from 10.9 to 11.5 hours per day.

Therefore, a lying time of around 11 hours per day would seem to be an appropriate target, and cows that lie down for shorter periods are more likely to become lame.

Cows lie down for longer in straw yards and on pasture than in cubicles, particularly if the cubicles have inadequate bedding (Singh et al, 1993a; 1993b; 1994). The use of straw bedding in cubicles has been abolished or greatly reduced on many farms, mainly because of its incompatibility with liquid-manure (slurry) handling. Rubber mats, mattresses, sand, sawdust, shavings, shredded newspaper and rubber tyres, or rice hulls are satisfactory alternatives to straw. With appropriate management, virtually any of the current cubicle bedding options will help to reduce lameness and encourage cows to get off their feet. In fact, cows will tolerate many inadequacies in cubicle design to lie on a cushioned surface.

A combination of straw yard and concrete area (70:30 ratio) adjacent to the feeding area has many advantages for cows' comfort, but is expensive in bedding and labour costs associated with daily topping-up and removal of old bedding one or more times during the winter period. At least 4.5 square metres of bedded area per cow should be provided (Bickert & Cermak, 1997).

Passageway Design The crucial issue is to ensure that animals are free to move around and pass each other unhindered. Areas that are particularly intensively used by cows are the alleys between rows of cubicles, around water troughs, and at other prime sites within the building such as feeding areas, collecting yards, and entries and exits from the milking parlour (Potter & Broom, 1990). Therefore, the space available in these "strategic sites" must be generous to ease movement and avoid aggressive confrontation. Very narrow passageways may create a problem for subordinate cows. The competitive use of space and an underlying level of social aggression, promoted by social hierarchy of cows, often results in sudden actions of avoidance, causing these animals to turn and twist on unyielding and abrasive surfaces. This can lead to claw horn damage, especially of the white line. The width of the passageways between feed bunk and curb of the cubicles should be 3 to 3.5 m when the feed fence is an integral part of the cubicle barn (Bickert & Cermak, 1997). This allows two cows to pass each other while others are feeding.

Most investigators believe that adverse housing conditions and changed patterns of behaviour result in an increased incidence of lameness. Certainly, some of this lameness is the direct result of injury. However, it is also thought that animals that are stressed are more prone to claw diseases. Careful observation of the animals for aggressive behaviour and the amount of time spent resting or standing, may provide a useful indicator of the importance of this environmental factor.

Exercise Locomotion maintains adequate blood circulation in the claws, supplying nutrients and oxygen to the keratin-producing tissues. In intensive dairy systems, cows are maintained in relative physical confinement (overcrowded) and have limited opportunity for exercise. A significant reduction in the amount of exercise decreases the rate of blood perfusion of the corium. This state reduces the rate of toxin removal, causes anoxia, and increases intra-ungular pressure.

Cows confined to tie stalls or pens move less than cows in loose housing systems (cubicles), and locomotion of cows in a cubicle facility is reduced when the walking space (loafing or exercise area) is 3.0 square metres or less per cow (Bickert & Cermak, 1997).

Recommendations:

- Keep concrete surfaces clean and in a fit state of repair; make sure they are non-abrasive, but not slippery.
- Provide comfortable (resilient and dry) resting areas; straw yards are better than cubicles. Cubicles must have plenty of bedding and the correct dimensions.
- Provide adequate numbers of cubicles (i.e. a cubicle for each cow in the herd) to prevent competition for lying space.
- Design cubicle rows and passageways in such a manner that cows can move and walk freely and avoid aggressive confrontation.
- Make sure that slurry does not accumulate in the passageways.
- Separate dry cows from the milking herd and keep them on dirt or grassed areas.
- Allow lactating cows as much exercise as practicable, preferably outdoors on pasture, as well as a loafing area or a dirt lot

NUTRITION

The role of nutrition and feeding management in the development of lameness has received much attention. Despite the possible overemphasis on nutrition, it has become clear that feed input is a factor in herd lameness. Feeding diets that result in a significant and prolonged drop in rumen pH will result in a dramatic increase in lameness. Laminitis is regarded as a major predisposing factor in lameness due to claw lesions such as white line disease, sole ulcers and sole haemorrhages. Laminitis is a multi-factor disease, nutrition supposedly being an important factor in its aetiology (Vermunt & Greenough, 1994). Understanding of causal factors is embryonic. It is known that a disturbed circulation is essential to the development of laminitis, but its exact role is still unclear.

Carbohydrate Subacute rumen acidosis (SARA) plays an important role in the initiation of laminitis and subsequent lameness. Excessive grain or non-structural carbohydrate (NSC) feeding, slug feeding of grain, feeding sources of NSC that are rapidly fermented in the rumen, and feeding finely chopped silage are common factors in the development of laminitis because of their propensity for inciting SARA. The risk of SARA developing is less when the concentrate to forage (C:F) ratio of the diet is kept under 60:40.

Westwood and Lean (2001) examined the potential for nutritional factors to contribute to the high incidence of claw lameness in New Zealand. They proposed that the high digestibility, high concentration of rumen degradable protein (RDP) and the low effectiveness of neutral detergent fibre (NDF) in lush pasture diets result in suboptimal rumen function, which in turn increases the risk of laminitis / lameness.

Fibre Dairy cows require a minimum amount of effective fibre and forage in their diet for proper chewing and rumination activity, for proper rumen function, and to maintain rumen pH above 6.2. They need to chew (masticate and ruminate) 10 to 12 hours/day to maintain normal rumen function (Shaver, 1997). The effective fibre of a feed is directly related to the chewing time and, therefore, saliva production associated with that particular feed (Allen, 1997). High-fibre diets, e.g. hay and coarsely chopped silage, stimulate rumination, which in turn increases saliva flow. Saliva is rich in bicarbonate, which acts as a buffer by neutralising the acid produced in the rumen. Fine chopping reduces the effective fibre content of forages. Adding buffers to rations containing finely chopped silage may help if saliva production is low. The addition of buffers at 0.75% of total ration dry matter is common with maize (corn) silage-based rations. Sodium bicarbonate should not be fed at levels greater than 1% of the ration otherwise its palatability will be affected.
Under US feeding systems, it is recommended that the diet contains a minimum of 25% NDF. This recommendation, however, may be inadequate for diets in which pasture is the predominant forage (Westwood et al, 2003). One reason rumen pH may be low on high quality pasture is that NDF in pasture is not as effective as NDF in silage and hay. In this situation, adding a small amount of straw to the diet may be beneficial.

Protein It has been suggested that feeding excess protein (particularly RDP or degraded intake protein) to dairy cattle may cause laminitis (Vermunt, 1992). However, little research information is available to indicate the level of dietary protein that may be of concern or the mode of action that protein plays in the disease development process. It is assumed by some that products of protein degradation in the rumen may be responsible for the increased incidence of lameness. Offer et al (1997) found that the source of dietary protein (either a proprietary protein supplement of animal origin or soybean meal) in the concentrate from week 3 to week 27 of lactation had no effect on locomotion, lameness, lesion formation, or any other claw measurement. Whether or not the high protein content of pasture is contributing to increased lameness in pasture-fed cattle needs further investigation. It is known that sulphur-containing amino acids contribute to the sulphur bonds that give horn tissue the strength and resilience needed to minimize lameness. However, studies in which the amino acid L-methionine was fed to improve horn flexibility and claw durability, and to reduce lameness found no advantage from the use of such proteins (Logue et al, 1989).

Silage Poor quality silage has long been recognised as a potential risk factor for lameness. A recent study investigated the effect of forage type on claw horn lesions in dairy heifers from 3 months of age until 6 months after calving (Offer et al, 2003). Both white line and sole lesions were significantly worse in animals that were fed a wet, fermented grass silage-based diet compared to those that were receiving a dry, unfermented straw and concentrate-based diet.

Recommendations:

- Provide a steam-up ration 2 before calving, with cows receiving concentrate up to 0.5% to 0.75% of BW or 3.5 to 5.0 kg per cow daily.
- Gradually increase concentrate (grain, maize silage) intake during the first 6 weeks of lactation. Never feed more than 4 kg of concentrate at one time.
- Do not exceed 35% NSC in the ration.
- Be careful with feeding NSC sources with high rumen degradability, such as barley, wheat and wet, finely ground high-moisture corn. The latter should not be fed if the moisture content is greater than 35%.
- Ensure that there is enough fibre in the diet. Rations should contain a minimum of 21% NDF from forage.
- Provide adequate, "effective fibre" in the diet; long-stem roughage is best. Formulate rations to include 30%-40% forage in the dry matter.
- Silage should be chopped to contain 25% of the par ticles more than 5 cm long. If chopped too finely, feed 2 to 4 kg of long orcoarsely chopped hay per cow daily.
- Supplement dietary buffers in early lactation; e.g., sodium bicarbonate at 0.75% to 1% of total ration DM.
- Consider feeding a complete-diet or total mixed ration feeding to control the C:F ratio.
- Avoid excesses of the energy (starch or carbohydrate) and protein components in the ration.
- Always make all feed changes slowly.
- Young stock diets should not be heavily based on wet grass silage (< 25% DM).

HERD MANAGEMENT/STOCKMANSHIP

In early lactation, dietary intake is unable to meet the demands of high milk production. Dairy cattle, therefore, enter a period of negative energy balance (NEB), which leads to mobilisation of body reserves to balance the deficit between energy intake and milk energy production (Bauman & Currie, 1980). Consequently, body condition scores (BCS) decrease to compensate for the NEB. Wells et al (1993) reported that, phenotypically, increased lameness was associated with decreased BCS. In a study that evaluated clinical lameness in 24 herds, it was found that lameness was most common during the first 50 days of lactation (Boettcher et al, 1998), when NEB would be most severe. Similarly, in a more recent study, increased locomotive problems were found to be associated with longer and more extreme periods of NEB (Collard et al, 2000). The implications from these studies are clear. Cows with the greatest dry matter intakes in early lactation are those that produce more milk, loose less weight, and have fewer lameness problems.

A study on lameness in dairy cows conducted by the University of Liverpool considered amongst others the factor of stockmanship in claw lameness (Clarkson et al, 1993). When herding cows, some farmers let the animals cows amble along at their own speed, while others encourage them by shouting, using sticks, dogs, or motor bikes. It has been shown that the more the cows get pushed the more lameness there will be (Chesterton et al, 1989; Clarkson & Ward, 1991). When the stockman was impatient, lameness was higher and in the majority of the high lameness incidence herds a biting dog was used to bring in the cows for milking.

Another study found that the amount of lameness was closely related to each of the following aspects: knowledge, training and awareness (Mill & Ward, 1993). Those farmers who knew most, had most training, were most aware of lameness and consulted their veterinarians, had the least lameness problems. From this, it can be concluded that there is a need to improve the level of knowledge that farmers have about lameness. This includes recognition of lameness, the causes of lameness, recording and early treatment of lameness cases, and the principles of prevention of lameness in dairy cattle.

Recommendations:

- Start providing a transition ration 2 to 3 weeks before calving to encourage food intake.
- Provide a well-balanced, palatable diet that meets the cows' meta bolic needs as soon as possible after calving.
- Use patience while assembling and herding cows, and drive cows gently over tracks and through gate ways; the herd should be allowed to drift to and from the milking shed (a herd walking speed guideline is 45 m/min or 2.7 km/h).
- Do not use a biting dog, motorbike, or tractor to herd cows.
- Use separate herds for heifers and older cows, espe cially on farms with large herds (more than 200 cows).
- Encourage farmers and stockpersons to acquire extra training to increase their awareness and to enable them to recognise and deal with lameness problems.

FARM TRACKS/RACEWAYS

During the summer when cows are at pasture and in countries such as New Zealand and Australia, where year-round grazing is practised, housing is not a factor in the aetiology of lameness. However, lameness is still a major cause for concern. In a ground-breaking epidemiological study in New Zealand, factors associated with the movement of animals along farm tracks to the dairy shed explained 40% of the variance with regard to the lameness prevalence level (Chesterton et al, 1989). The importance of the track may be due to the fact that in New Zealand (as well as in Australia) cows have to walk the distance between the paddocks and the shed 4 times a day. In these situations, lameness is associated with the length, width, site, quality, construction, maintenance and use of the farm track, with the handling and movement of animals on the track, and with herd size (Harris et al, 1988; Chesterton et al, 1989; Clarkson & Ward, 1991; Hemsworth et al, 1995). The two factors most strongly linked to lameness are:

1. The maintenance state of the track, and
2. The patience of the herdsman handling the cows on the track.

Proper construction and regular maintenance of the track, especially the first 300 to 500 metres closest to the milking shed, are important in reducing lameness in dairy herds (Bridges, 1985; Malmo & Vermunt, 1999). Farms where track maintenance is poor or where the herdsman has less patience with the herd on the track are more likely to have a high number of lame cows. A poorly maintained track will not only cause injury to the claws of the cows (direct effect), it will also slow the herd down, making the herdsman less patient (indirect effect). Furthermore, lame cows most often walk at the back of the herd and therefore are more affected by an impatient stockman.

Recent observations show that dairy cows have a very strong preference for a softer (woodchips) track surface in comparison with a conventional hard-core track surface (Gregory & Taylor, 2002).

Recommendations:

- Ensure that the width of the main track is at least 5 m for herds of 200 cows or more.
- Use fine, non-abrasive or easily crushable material (e.g., sand, pumice, limestone, sandstone or wood chips) rather than coarse gravel on the surface of the track.
- Ensure that the track is firm, correctly crowned to pro mote drainage from the centre of the track, and well drained along the sides.
- Fill holes and repair any broken section by grading, rolling, or both.
- Avoid steep slopes and eliminate any areas of poten tial bunching of the herd (e.g., sharp corners, nar row entrance into the yard, narrow bridges and underpasses).
- Ensure that excellent underfoot conditions are main tained year-round at gateways and drinking troughs.
- Remove adjacent hedges or keep them well trimmed to enable the sun and wind to dry out the track.
- Direct track expenditure toward those parts nearest to the shed when improvements are necessary.
- Avoid the use of farm machinery (including motor bikes) on the cattle tracks.
- Physically walk all the farm tracks as part of a herd lameness investigation.

THE DAIRY SHED/HOLDING YARD

Milking as such should be a pleasant experience for cows, and - everything else being normal - cow flow to the holding yard and from the yard into the dairy shed should be smooth. Positive incentives for cows to come into the shed will further improve cow flow. If cows do not come forward willingly to be milked, a shed problem may exist.

Factors associated with characteristics of the milking process explained 24% of the variance with regard to the lameness prevalence level (Chesterton et al, 1989). Among the risk factors for lameness in the dairy shed, the presence of a biting dog was the most important one. A poorly designed shed will also affect the normal flow of cows through the yard and in to the shed. Cows do no like physical contact, and a lot of pushing, turning and shuffling may occur if the yard is too small. This may cause cows to slip and to have less control over where they place their feet, thereby increasing the risk of claw injury - especially if stones and gravel are carried in to the yard.

Recommendations:

- Have the entrance to the holding yard wide enough, and avoid acute turns into the yard.
- Keep concrete surfaces clean and in a fit state of repair; make sure that they are non-abrasive, but not slippery.
- Eliminate factors that make cows reluctant to enter the holding yard and milking shed (e.g., shadows, slip pery concrete, electrified backing gate, a dog chained to the gate, and stray voltage).
- After every milking, clean the yard and the concrete apron linking the main track with the yard; ensure that slurry does not accumulate at the yard entrance.
- Ensure the holding yard is big enough (i.e.1.5 m2/cow or more).
- Consider bail feeding to improve cow flow into the shed.

CONFORMATION

Much of the variability in claw health is associated with environmental effects, including differences in housing, nutrition and management. However, research has also revealed genetic sources of variation (Huang & Shanks, 1995), indicating that selection can be used indirectly to select for resistance to lameness. It has been shown that the daughters of some bulls were more likely to suffer claw lameness than those of other bulls (Russell, 1988), and it would be sensible to select bulls on the basis of clinical lameness. A large UK study found that cows with long toes or with abnormally low or high heels had a higher prevalence of lameness (Clarkson et al, 1993). McDaniel (1994) concluded from three separate comprehensive studies that higher claw angles were positively correlated with increased herd life. Boettcher et al (1998) reported that among the various conformation type traits analyzed, claw angle and rear leg - rear view had the strongest associations with lameness. Low claw angle and hocking-in were associated with clinical lameness. In other words, bulls that transmitted higher claw angles and straighter limbs from the rear view had fewer daughters with clinical lameness. Rear leg - rear view evaluates the straightness of the rear legs when viewed from behind and is measured by the degree of inward deviation of the hocks and the corresponding degree to which the toes point outward. There appears to be a strong relationship between the rear leg - rear view and the general health and soundness of the claw.

In contrast to popular belief, it was also found that the correlation between clinical lameness and rear leg - side view was essentially zero (Boettcher et al, 1998), indicating that neither posty legs nor straight hocks are strongly associated with lameness. Similarly, McDaniel (1997) reported that cows with mildly straight legs had longer lives (i.e. greater survival). In another study, it was found that cows with a steeper claw angle, straighter rear leg angle, downward sloping rump and wide pins were less likely to develop lameness and had better herd longevity (Van Dorp et al, 1998).

There are indications that certain breeds have a predisposition of lameness. For example, sole ulcers occur predominantly in very large-framed, pure-bred, Holstein-Friesian cows (Jubb and Malmo, 1991). Also, the claws of cows in high lameness prevalence herds are more likely to be less pigmented (Chesterton et al, 1989), which is common in Holstein-Friesian cattle. White horn is about 30% softer than dark-pigmented horn and, consequently, may be more prone to damage. This provides a good example of fashion conflicting with considerations of health.

Recommendations:

- Select bulls that sire offspring with shorter, steeper claws (in addition to high milk yield).
- Select bulls with straight legs (rear view) and hock angles (side view) of about 170o in breeding pro grammes for herd replacements.
- Consider the use of crossbred cows (dark-coloured horn).

BEHAVIOUR

The mean lying time for cows housed indoors is less than at pasture (Galindo & Broom, 1993). A similar observation has been reported by Singh et al (1993b). Several authors have suggested that behaviour is a factor to consider in lameness (see Vermunt & Greenough, 1994).

Modern, intensive dairying systems have important consequences for the social behaviour of the animals. Several studies (e.g., Galindo & Broom, 1993) have shown that aggression is increased and the synchrony of behaviour disrupted when cows are housed at high density. Reduced space and constant regrouping of cows causes increased aggression, partly because cows have to compete more for eating and lying places (Wierenga, 1991). Under these conditions, some animals will be more successful than others at gaining access to feeding or lying places. A behavioural study found that low-ranking animals, such as heifers, whose movement is restricted by social factors, spend less time lying and more time standing still in passageways and standing half in cubicles (Galindo & Broom, 2000). As the total time spent standing increased, so did the number of cases of lameness. The survival rate to lameness for low-ranking cows was significantly lower than for higher-ranking animals. Similarly, Chaplin et al (2000) reported that more severe claw lesions in early lactation heifers were associated with reduced lying, less idling, increased standing in cubicles, and more disturbed lying behaviour. A New Zealand study showed that a relationship exists between dominance ranking and lameness incidence in year-round, pasture-grazed dairy cows (Sauter-Louis et al, 2004). The dominance ranking of lame cows is significantly lower in herds that have a high incidence of lameness compared with herds that have a low incidence.

The cubicle to cow ratio generally recommended is 1:1. However, this does not mean that all cows in a group will have a lying place guaranteed and that they will be able to lie for the length of time they want. Therefore, the number of cubicles should exceed the maximum number of cows in the herd (Vermunt & Greenough, 1997).

Recommendations:

- Ensure at least one cubicle per cow and, if passage fed, one feed space per cow. This minimises con frontation between dominant cows and those lower in the social hierarchy (mainly heifers).
- Always have a spare number of cubicles to provide options for lying to those cows reluctant to use specific cubicles or that are displaced more frequently from certain areas.
- Run separate herds of similar classes of cattle (such as first-calving cows), especially in large herds on pasture.
- Handle first-calf cows carefully during the first 60 days of lactation.

FUNCTIONAL CLAW TRIMMING

The primary purpose of claw trimming is to re-establish normal function by correcting claw horn overgrowth, thereby restoring appropriate weight bearing within and between the claws of each limb. Studies have shown that horn wear is decreased and horn growth increased by claw trimming (Manson & Leaver, 1988; 1989). The explanation may be that artificially removing horn from the sole stimulates a natural compensatory reaction of increased horn production, thereby balancing wear. The new horn may even be of better quality than that removed. Therefore, regular claw trimming to stimulate the growth of healthy horn may help in the control of lameness (Vermunt, 1999). For example, a recent Swedish study found that autumn-trimmed cows at spring trimming had significantly lower odds of lameness, haemorrhages of the sole or white line, sole ulcer and white line disease or double sole (Manske et al, 2002). Also, urgent claw treatment between claw trimmings was less common in the autumn trimmed cattle. "Functional claw trimming", carried out once or twice yearly according to the Dutch standard (Toussaint-Raven et al, 1989), is now regarded as an integral part of any lameness management and control programme.

On the other hand, inadequate or unskilled claw trimming are recognised factors, which may cause lameness. In the University of Liverpool study it was found that claw trimming may be a risk factor of lameness unless done correctly and at the correct time (Clarkson et al, 1993). The conclusion reached was that: "Foot-trimming can be beneficial, but not always. It would seem that correct training in the correct technique is essential".
In a recent study (Paulus & Nuss, 2002), the sole thickness on the medial and lateral claw was compared. It was found that if the two claws were trimmed to equal size (conventional wisdom for Dutch claw trimming), then the sole of the lateral claw was on average 1.6 mm thinner than on the medial claw and in some areas up to 4.1 mm thinner. This almost led to exposure of the corium. Therefore, to achieve equal sole thickness it is necessary to leave the lateral claw larger than the medial.

Recommendations:

- Provide regular claw care (i.e. inspection and trimming).
- People who trim claws should be trained to use the correct technique.
- Do not trim just before turn-out or calving; one recommended time is at drying off.
- Heifers should have their claws trimmed only lightly before entry into a loose-housing system or confinement on concrete.
- Dairy cattle veterinarians should attend short courses or workshops to learn proper claw care (including claw trimming).

ENVIRONMENT (especially rainfall and heat stress)

In New Zealand, the peak incidence of lameness occurs during the late autumn/winter in autumn-calving herds and during the late spring in spring-calving herds (Tranter & Morris, 1991). Practical experience from both New Zealand and Australia tells us that more lameness occurs in seasons with high rainfall, and that a high incidence of lameness usually follows prolonged periods of heavy rain. Also, numerous studies have shown an association between wet weather conditions and the onset of lameness. Wet underfoot conditions will result in:

- An increase in claw horn moisture ==> horn becomes softer (esp. the sole) ==> greater wear and more chance of sole penetration.
- Concrete being much more abrasive ==> more wear of the weight bearing surface.
- The soil being washed off the surface of the tracks ==> exposing stones and other sharp material ==> increased risk of trauma.
- Tracks getting very muddy ==> cases of infectious conditions causing lameness (e.g., interdigital necrobacillosis).

In North America, heat stress has also been associated with an increased incidence of lameness (Spencer, 2001). Heat stress alters the animals' breathing rate (it may double), heart rate, immune response (reduced) and behaviour (e.g., cows are standing for longer periods of time, which promotes pooling of blood in the digits). Reduced feed intake, a preference for concentrates rather than forage, a loss of salivary buffering from increased respiratory rates and drooling, and a reduction in the total buffering pool all contribute to a greater potential for subacute rumen acidosis (SARA) during periods of hot and humid weather. This may explain in part, why some herds experience more acidosis and lameness despite being fed properly formulated rations.

OTHER FACTORS IN LAMENESS:

Management of Replacement Stock Studies on the effect of age on lameness have shown that young, first-calving heifers are more likely to be severely affected both in term of the lesions observed as well as in their reaction to it (Logue et al, 1993). Early in the housing period, first-lactation animals (heifers) lie down for a significantly shorter period than adult cows. Such a shorter lying time is significantly related to sole lesions (Singh et al, 1993c). Heifers housed in free stalls during the year before parturition - having already experienced the use of cubicles - will likely demonstrate a higher rate of cubicle use as first-lactation cows. Segregating first-lactation cows from older cows in a separate group will result in less bullying by the older animals. This practice also makes sense from a nutrition point of view, because adequate dry matter intakes are more likely being maintained, with animals experiencing fewer health-related problems, including laminitis.

Growth rate is another factor that has been considered. Greenough and Vermunt (1991) concluded that rapid growth (greater than 800 grams per day) in young heifers during their second year and prior to calving at 24 months, coupled with the sudden mixing of these pregnant heifers with older cows, played an important role in the occurrence of sole haemorrhages, which often preceded sole ulceration.

Recommendations:

- Start at breeding age to train heifers to the system that they will join after parturition.
- Heifers should be put in a separate area of the build ing with deep- strawed cubicles where they can learn to use the cubicles.
- In systems requiring housing, allow heifers to adapt (for about 8 weeks) to reduced exercise and to walking on a concrete surface prior to introducing them to the main herd.
- Avoid introducing single heifers to the main herd; transferring heifers in groups of four or five may reduce the amount of bullying.
- Monitor the growth rate of heifers, particularly during their second year.

Previous Lameness -- Cows with a history of prior incidents of lameness are more likely to go lame again (Alban et al, 1996). A French study showed that cows lame in one lactation had an increased risk of becoming lame in the next (Calavas et al, 1996). Therefore, prevention of lameness in first and second-calf cows is of paramount importance, and detailed records should be kept of lame cows, lesions, treatment etc.

Production (Milk Yield) High production is often associated with undesirable conformation, which indicates that breeding solely for production traits will increase the risk of lameness. For example, Alban et al (1996) reported a significant association between average herd yield and lameness. In another study, breeding values for first-lactation claw trait scores were generally negatively correlated with those for milk production (Brotherstone et al, 1991). Analysis of the Liverpool data (Clarkson et al, 1993) showed that cows, which were lame in their second lactation had yielded more milk in the first than cows that were not lame in the second lactation (Ward & French, 1997). The risk of lameness increased in each lactation from the first/second up to the seventh. A recent Dutch study found that high milk yield on the first herd test after calving was a reliable indicator of an increased risk of lameness (Heuer et al, 1999). As part of a biotin intervention study, it was found that cows that went lame were, on average, higher yielding than cows that never went lame (Green et al, 2002). However, when cows did go lame they lost all that yield advantage of 350 litres. Interestingly, the yield reduction started four months prior to the onset of lameness. It is tempting to speculate that the initial reduction in yield is associated with the original insult (e.g., subclinical laminitis), which could be from a variety of causes. This is then later translated into a claw horn defect or lesion.

Age and Stage of Lactation The influence of age on the incidence of lameness varies, depending on the lesion. Excessive wear (thin soles) and bruising occur most commonly in young cows (heifers) and natural service bulls. White line disease and sole ulcer are more often observed in older cows, and for these lesions the risk of lameness increases with age.

The prevalence of lameness is highest during the first 3 to 4 months of lactation, which coincides with a high level of energy intake and negative energy balance. Vaarst et al (1998) found a strong positive association between 61 and 120 days post-partum and the presence of claw disorders.

Body Weight and Body Condition Score (BCS) Both these factors have been associated with clinical lameness in some dairy herds (Wells et al, 1993). Each 100-kg increase in body weight was associated with a 1.9-fold increase in the odds of clinical lameness. A lower BCS was correlated strongly with clinical lameness. However, this may be the result of, rather than a cause of, lameness.

ADDITIONAL RECOMMENDATIONS

- All lame cows should be examined and treated as soon as possible.
- The claws of all cows should be examined and trimmed (if necessary) at least once annually.
- Footbath use as part of a regular claw care programme is essential, because it reduces a number of infectious conditions affecting the area between the claws.
- Consider using a Treponema vaccine (if available) to control digital dermatitis in problem herds, rather than relying on footbaths containing chemicals.
- Clean floor surfaces and alleyways as required, to remove collections of urine, water and manure that contribute to constant wetting of the claws.

Early diagnosis and treatment of lesions is essential for controlling lameness in dairy herds by minimising the severity and potential long-term implications of lameness. Most of this can be done at the time of routine claw trimming. However, surveys have found that only about 20% to 30% of healthy cows have their claws trimmed regularly.

Footbaths are used to remove dirt and abrasive material, and to bring claws and interdigital skin in contact with a disinfecting, astringent chemical solution. The use of permanent (concrete) footbaths has declined in favour of portable equipment. Formalin, copper sulphate and peracetic acid are the most widely used agents, all showing equal efficacy in treating digital dermatitis (Raven & Hunt, 2002). Other chemicals such as iodides and creosols rapidly fail owing to the organic matter in the washing liquid.
A large survey of dairy herds across North America concluded that 43.5% of herds were affected with papillomatous digital dermatitis (Wells et al, 1999), and it appears that the incidence of this condition is increasing rapidly (Cook, 2002). Footbaths containing antibiotic solutions are also used in the control and treatment of digital dermatitis. The antimicrobial activity and concentration of these products decrease significantly after a herd has used the bath, possibly because of absorption by faeces and soil particles (Van Keulen et al, 1992).

- Use Locomotion Scoring as an integral part of any lameness prevention programme.

Locomotion Scoring centres on prevention rather than treatment, because the system will detect many lesions in the very early stages. Therefore, it can be used to identify at-risk cows for possible treatment. Early detection and intervention is essential for minimising the severity of lameness. Lame cows receiving prompt treatment loose less than 1% of lactation potential compared with cows neglected for two or three days, which can lose up to 20% of production/lactation. Locomotion Scoring is an easy, sensitive and relatively accurate method of detecting subclinical lameness within a herd. The cow's gait (locomotion) is assessed visually with special emphasis on her back posture. Researchers at the University of Liverpool showed that the curvature of a cow's spine can be used to predict the presence of a claw lesion (O'Callaghan et al, 2002). With this system, cows are ranked from 1 (normal) to 5 (very lame). Scores of two or three indicate subclinical lameness. Identifying these animals allows for early intervention (examination) and correction (trimming) before lameness becomes more severe and costly. Once lame animals have been identified and treated, preventative strategies can be implemented for the types of lameness common to a particular herd.
In the future, detecting lameness may well become more automated. For example, by using a force-plate system positioned at the entrance or exit of the milking shed, lame cows can be recognised early and the affected limb(s) identified (Rajkondawar et al, 2002).

- Consider feeding biotin in problem herds.

Biotin (vitamin H) is a water-soluble vitamin, which is essential for the formation and integrity of keratinised tissues such as skin and horn. Biotin supplementation (above requirement) is of value for improving the quality of claw horn in pigs, hoof horn in horses, and foot pad epithelial tissue in poultry. Under normal conditions, the developed rumen is considered capable of synthesizing adequate amounts of biotin. However, it has been suggested that rumen synthesis of biotin is not significant in cattle (Frigg et al, 1994). Evidence exists that acidic rumen conditions can reduce microbial biotin synthesis (Da Costa et al, 1998). Therefore, cattle fed high grain or high-quality pasture diets may develop a subclinical biotin deficiency. Cows suffering from laminitis have lower blood biotin concentrations and a higher moisture content of sole horn than normal cows (Huguchi & Nagahata, 2001). Furthermore, the biotin demand is increased during periods of stress and blood biotin levels found in lame cows are lower than normal (Smart & Cymbaluk, 1997).
Recently, biotin supplementation has received much interest for improvement of claw health in dairy cattle. Several controlled studies have shown that lameness is significantly decreased in cows supplemented with biotin (Rovimix H-2; Hoffmann-La Roche Ltd, Basel, Switzerland) at a rate of 20-40 mg of active biotin/cow per day. Midla et al (1998) reported significant improvements in claw health in primi-parous dairy cows following biotin supplementation. In a recent study, the risk of lameness caused by white line disease was halved (Hedges et al, 2001). These positive effects of biotin supplementation on white line disease appeared to be greater in cows than in heifers. Fitzgerald et al (2000) reported that the response to supplemental biotin was a reduction in claw lesions and better locomotion scores. In another study, a significant improvement in histological horn quality was found in biotin-treated animals, indicating that biotin exerts a positive influence on the healing of sole ulcers (Lischer et al, 2002a).

- Consider feeding trace mineral complexes in at risk cows or problem herds.

Trace minerals are essential for the production of good-quality claw horn, and an inadequate intake may compromise claw health. Zinc (Zn) and copper (Cu) are the "popular players", but cobalt (Co), selenium, molybdenum and manganese (Mn) are also important in claw horn formation. Zn and Cu deficiencies have been implicated in lameness, and supplements are commonly used worldwide. Trace minerals have traditionally been added to ruminant diets in the form of inorganic salts. However, in recent years, there has been considerable interest in organic forms of trace minerals, which are claimed to have superior bio-availability (Miles & Henry, 1999). These organic forms, or chelates, are stable in the digestive tract and are protected from forming complexes with other dietary components that would otherwise inhibit their absorption (Spears, 1996).

Improvements in claw integrity have been observed when Co-glucoheptonate and specific amino acid complexes of Cu and Mn were added to diets of cows already containing specific amino acid complexes of Zn (Nocek et al, 2000). A recent US study found that replacing Zn, Mn, Cu and Co from sulphates with complexed sources (Availa-4; Zinpro Corporation, Eden Prairie, Minnesota, USA) decreased the percentage of dairy cows with claw disorders at 75 d after calving (Ballantine et al, 2002).

SO, WHERE TO FROM HERE?

It is well recognised that herd lameness is a problem associated with high production, intensive feeding and confined conditions, and that the increasing incidence of lameness needs to be addressed and reduced. However, herd lameness is a multi-factor problem; the causal factors are complex, interrelated, and not always well understood. Finding the ideal control strategy is like piecing together a jigsaw puzzle, with each piece revealing only a small part of the complete picture. Innovative research has filled in some areas of the puzzle, but other parts still remain unclear.

Most studies report a high incidence of lameness in early lactation (2 to 16 weeks after calving). The event of calving and its associated problems during the early postpartum period appear to be particularly important, but their exact role is not clear as yet. The many changes in management, feeding, environment and social grouping to which animals are subjected during the peri-partum period are likely to trigger a considerable level of stress. This occurs at a time when body metabolism is also changing rapidly during the transition from late gestation to peak lactation and beyond, perhaps more so than at any other point during lactation. These processes are of particular significance in heifers calving for the first time. Presently, a large body of international research is working on the hypothesis that systemic events associated with calving and the onset of lactation may set in motion the chain of events that leads to development of claw horn lesions in dairy cows.

Laminitis is generally regarded as a major predisposing factor for many lameness-causing claw lesions, particularly sole haemorrhages, sole ulcers and white line disease. The precise aetiology and pathogenesis of laminitis are incomplete. However, it appears that, similar to equine laminitis, a systemic and local digital inflammatory process occurs in bovine laminitis (Belknap et al, 2002). For many years, and in analogy to the horse, the most commonly accepted hypothesis has been that of insults (vasoconstriction and ischaemia) to the peripheral vascular system of the corium, resulting in a reduced digital capillary perfusion, particularly through the lamellae and papillae (see Vermunt, 1992; Vermunt & Greenough, 1994; Ossent & Lischer, 1998). It is hypothesised that the ischaemia results because blood is shunted away from the capillary circulation through dilated arteriovenous anastomoses (AVAs).

However, recent research has provoked some debate regarding the role that AVAs and the micro-circulation play in the initial stages of laminitis and the subsequent development of claw lesions. For example, it has been suggested that loss of integrity of the epidermal-dermal junction does not play such a significant role in the development of laminitis-associated lesions (Lischer & Ossent 2002; Lischer et al, 2002b). It is more likely that other biomechanical mechanisms are involved as a result of changes in the connective tissue of the distal phalanx' suspensory apparatus (Tarlton, 2003). It has been shown that there is an increased laxity of the distal phalanx' suspensory apparatus around the time of calving, leading to a decreased load bearing capacity (Tarlton & Webster, 2002). This means that the distal phalanx can move up and down in the claw to a certain degree, potentially causing trauma to the corium (dermis). It has been proposed that, if the properties of the connective tissue in this area are sufficiently disturbed, the fibres may stretch enough to allow the third phalanx to sink within the claw capsule, subsequently producing the characteristic lesions of laminitis at the sole surface (Ossent, 2000). Some people suggest that this phenomenon may be the causal link between calving and lameness.

Based on these recent findings, several theories have been advanced regarding the origin of the increased laxity of the connective tissue. One favours the central role of matrix metalloproteinases and their activation by a novel 52kDA protease, called "hoofase". This gelatinolytic p52ase is present at high levels in heifers up to two weeks pre-calving, then declining at four and 12 weeks post-calving (Tarlton, 2003). Another theory favours the effects of certain hormones, particularly relaxin (Holah et al, 2000), which are present in the peri-partum period. Neither of these theories, however, can explain the frequent occurrence of laminitis-associated lesions in bulls and steers. Also, these typical lesions are relatively rare in year-round, pasture-grazed dairy cows in New Zealand and Australia, even more so in first-lactation heifers. Therefore, could it be more likely that the breakdown of the dermal-epidermal lamellar connection is facilitated by the action of a Streptococcus bovis exotoxin (Mungall et al, 2002), released during an acidotic event? Alternatively, could it be that histamine - produced by ruminal conversion of the amino acid histidine - is a bigger contributing factor to perturbed supply of nutrients to the horn-producing tissues than is commonly thought? A histamine-producing bacterium has been identified that may be implicated in the pathogenesis of laminitis (Russell & Garner, 2003). Clearly, these hypotheses warrant further investigation.

It has become clear that calving and early lactation are important as far as the initiation of lameness is concerned. The best prospects for control lie in a preventative management programme, which addresses amongst others cow comfort, nutrition, management, behaviour, environment, claw care and conformation. Minimising external stresses to the claws during these critical periods may significantly reduce the development and severity of lameness-causing lesions.
Continued research into identification of causal factors of lameness will be invaluable in providing useful tools for more effective control of herd lameness. It is essential that future funding in the subject of lameness is maintained, not only because of an increasing consumers' awareness that it is a major welfare issue associated with modern dairy production systems, but also because it will help to increase our understanding of causes and mechanisms involved in herd lameness. This in turn will allow us to develop sound, research-based recommendations for prevention and control.

REFERENCES

1. Alban I, et al (1996). Lameness in tied Danish dairy cattle: The possible influence of housing systems, management, milk yield, and prior incidents of lameness. Preventive Veterinary Medicine 29:135-49
2. Allen MS (1997). Relationship between fermentation acid production in the rumen and the requirement for physically effective fiber. Journal of Dairy Science 80:1447-62
3. Argaez-Rodriguez FJ, et al (1997). Papillomatous digital dermatitis on a commercial dairy farm in Mexicali, Mexico: Incidence and effect on reproduction and milk production. Preventive Veterinary Medicine 32:275-86
4. Bakerma HW, et al (1994). The effects of lameness on reproductive performance, milk production and culling in Dutch dairy herds. Preventive Veterinary Medicine 20:249-59
5. Ballantine HT, et al (2002). Effect of feeding complexed zinc, manganese, copper and cobalt to late gestation and lactating dairy cows on claw integrity, reproduction and lactation performance. The Professional Animal Scientist 18:211-8
6. Bauman DE, Currie WB (1980). Partitioning of nutrients during pregnancy and lactation: a review of mechanisms involving homeostasis and homeorhesis. Journal of Dairy Science 63:1514-29
7. Belknap EB, et al (2002). Expression of pro-inflammatory mediators and vasoactive substances in laminitic cattle. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). P 387
8. Bennett RM, et al (1999). Estimating the costs associated with endemic diseases of dairy cattle. Journal of Dairy Research 66:455-9
9. Bickert WG, Cermak J (1997). Housing considerations relevant to lameness of dairy cows. In: Lameness in Cattle, 3rd edition. W.B. Saunders Company, Philadelphia. Pp 300-7
10.Boettcher PJ, et al (1998). Genetic analysis of clinical lameness in dairy cattle. Journal of Dairy Science 81:1148-56
11. Bridges, DJ (1985) Dairy farm race construction. Research Publication Series No. 10, Massey University Agricultural Research Foundation, Palmerston North, New Zealand
12. Broom DM, Galindo FA (1997). Behavior. In: Lameness in Cattle, 3rd edition. W.B. Saunders Company, Philadelphia. Pp 297-9
13. Brotherstone S, et al (1991). Dairy life in relation to linear type traits and production. 2 Genetic analyses for pedigree and non-pedigree cows. Animal Production 53:289-97
14. Calavas D, et al (1996). Analysis of associations among diseases in French dairy cows in two consecutive lactations. Preventive Veterinary Medicine 27:43-55
15. Chaplin SJ, et al (2000). A comparison of hoof lesions and behaviour in pregnant and early lactation heifers at housing. The Veterinary Journal 159:147-53
16. Chesterton RN, et al (1989). Environmental and behavioural factors affecting the prevalence of foot lameness in New Zealand dairy herds. New Zealand Veterinary Journal 37:135-42
17. Clarkson DA, Ward WR (1991). Farm tracks, stockman's herding and lameness in dairy cattle. Veterinary Record 129:511-2
18. Clarkson MJ, et al (1993). An epidemiological study to determine the risk factors of lameness in dairy cows. Liverpool University, Leahurst, Neston, Cheshire CH64 7TE
19. Clarkson MJ, et al (1996). Incidence and prevalence of lameness in dairy cattle. Veterinary Record 138:563-7
20. Collick DW, et al (1989). Associations between types of lameness and fertility. Veterinary Record 125:103-6
21. Collard BL, et al (2000). Relationships between energy balance and health traits of dairy cattle in early lactation. Journal of Dairy Science 83:2683-90
22. Cook NB (2002). The influence of barn design on dairy cow hygiene, lameness and udder health. Proceedings of the AABP 35th Annual Convention, Madison (Wisconsin). Pp 97-103
23. Da Costa GC, et al (1998). Effect of varying hay/barley proportions on microbial biotin metabolism in the rumen simulating fermenter (Rusitec). Proceedings of the Society of Nutrition and Physiology. 7:30 (abstract)
24. Dewes HF (1978). Some aspects of lameness in dairy herds. New Zealand Veterinary Journal 26:147-8 & 157-9
25. Dumelow J, Albutt R (1990). The effects of floor design on skid resistance in dairy cattle buildings. Proceedings of the VI International Symposium on Diseases of the Ruminant Digit, Liverpool, UK. Pp 130-4
26. Dürr JW, et al (1997). Culling in Quebec Holstein herds. 2. Study of phenotypic trends in reasons for disposal. Canadian Journal of Animal Science 77:601-8
27. Enting H, et al (1997). Economic losses due to clinical lameness in dairy cattle. Livestock Production Science 49:259-67

28. Esslemont RJ, Spincer I (1993). The incidence and costs of diseases in dairy herds. DAISY Report 2. Department of Agriculture, University of Reading, Earley Gate, Reading RG6 2AT
29. Farm Animal Welfare Council (1997). Report on the welfare of dairy cattle. FAWC, Government Buildings, Hook Rise South, Tolworth, Surbiton, Surrey KT6 7NF
30. Faull WB, et al (1996). Epidemiology of lameness in dairy cattle: the influence of cubicles and indoor and outdoor walking surfaces. Veterinary Record 139:130-6
31. Fitzgerald T, et al (2000). The influence of long-term supplementation with biotin on the prevention of lameness in pasture fed dairy cows. Journal of Dairy Science 83:338-44
32. Frigg M, et al (1994). Biotin kinetics in serum of cattle after intravenous and oral dosing. International Journal of Vitamin and Nutritional Research 64:36-40
33. Galindo F, Broom DM (1993). The relationships between social behaviour of dairy cows and the occurrence of lameness. Cattle Practice BCVA 1:360-5
34. Galindo F, Broom DM (2000). The relationships between social behaviour of dairy cows and the occurrence of lameness in three herds. Research in Veterinary Science 69:75-9
35. Green LE, et al (2002). The impact of clinical lameness on the milk yield of dairy cows. Journal of dairy Science 85:2250-6
36. Greenough PR, Vermunt JJ (1991), Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. Veterinary Record 128:11-7
37. Gregory NG, Taylor OD (2002). Dairy cow preference for a soft track surface. New Zealand Veterinary Journal 50(2):83
38. Guard C (1997). Proceedings of the North American Veterinary Conference, Orlando (Florida)
39. Harris DJ, et al (1988). The incidence, cost and factors associated with foot lameness in dairy cattle in south-western Victoria. Australian Veterinary Journal 65:171-6
40. Hassall SA, et al (1993). Effect of lameness on behaviour of cows during summer. Veterinary Record 132:578-80
41. Hedges J, et al (2001). A longitudinal field trial of the effect of biotin on lameness in dairy cows. Journal of Dairy Science 84:1969-75
42. Hemsworth PH, et al (1995). The welfare of extensively managed dairy cattle: a review. Applied Animal Behaviour Science 42:161-82
43. Hernandez J, et al (2001). Effect of lameness on the calving-to-conception interval in dairy cows. Journal of the American Veterinary Medical Association 218:1611-14
44. Hernandez J, et al (2002). Effect of lameness on milk yield in dairy cows. Journal of the American Veterinary Medical Association 220:640-4
45. Heuer C, et al (1999). Postpartum body condition score and results from the first test day milk as predictors of disease, fertility, yield and culling in commercial dairy herds. Journal of Dairy Science 82:295-304
46. Higuchi H, Nagahata H (2001). Relationship between serum biotin concentration and moisture content of the sole horn in cows with clinical laminitis or sound hooves. Veterinary Record 148:209-10
47. Holah DE, et al (2000). The histology and histopathology of the support structures in the laminated region of the bovine hoof in maiden heifers and around the time of first calving. Proceedings of the XI International Symposium on Disorders of the Ruminant Digit, Parma, Italy. Pp 109-12
48. Huang YC, Shanks RD (1995). Within herd estimates of heritabilities for six hoof characteristics and impact of dispersion of discrete severity scores on estimates. Livestock Production Science 73:513-31
49. Jubb TF, Malmo J (1991). Lesions causing lameness requiring veterinary treatment in pasture-fed dairy cows in East-Gippsland. Australian Veterinary Journal 68:21-4
50. Kossaibati MA, Esslemont RJ (1997). The costs of production diseases in dairy herds in England. Veterinary Record 154:41-51
51. Kossaibati MA, Esslemont RJ (1999). The incidence of lameness in a group of dairy herds in England. Proceedings of the British Society of Animal Science, p 221
52. Laven RA, Hunt H (2002). Evaluation of copper sulphate, formalin and peracetic acid in footbaths for the treatment of digital dermatitis in cattle. Veterinary Record 151:144-6
53. Leonard N, et al (1994). Effect of different housing conditions on behaviour and foot lesions in Friesian heifers. Veterinary Record 134:490-4
54. Lischer ChJ, Ossent P (2002). Pathogenesis of sole lesions attributed to laminitis in cattle. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 82-9
55. Lischer CJ, et al (2002a). Effect of therapeutic dietary biotin on the healing of uncomplicated sole ulcers in dairy cattle - a double blinded controlled study. The Veterinary Journal 163:51-60
56. Lischer CJ, et al (2002b). Suspensory structures and supporting tissues of the third phalanx of cows and their relevance to the development of typical sole ulcers (Rusterholz ulcers). Veterinary Record 151: 694-8
57. Logue DN et al (1993). Lameness in dairy cattle. Irish Veterinary Journal 46:47-58.
58. Malmo J, Vermunt JJ (1998). Lameness in dairy cattle - A review (part 1). Journal of the Australian Association of Cattle Veterinarians 8:23-31
59. Malmo J, Vermunt JJ (1999). Lameness in dairy cattle - A review (part 2) -Farm track construction. Journal of the Australian Association of Cattle Veterinarians 9:15-8
60. Manske T, et al (2002). The effect of claw trimming on the hoof health of Swedish dairy cattle. Preventive Veterinary Medicine 54:113-29
61. Manson FJ, Leaver JD (1988). The influence of dietary protein intake and of hoof trimming on lameness in dairy cattle. Animal Production 47:191-9
62. Manson FJ, Leaver JD (1989). The effect of concentrate:silage ratio and of hoof trimming on lameness in dairy cattle. Animal Production 49:15-22
63. Margerison JK, et al (2002). Effect of locomotion score and lameness on dry matter intake, feeding and

general behaviour. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 407-10
64. Mcdaniel BT (1994). Feet and leg traits of dairy cattle. Proceedings of the 8th International Symposium on Disorders of the Ruminant Digit, Banff (Canada). Pp 102-9
65. McDaniel BT (1997). Genetics of conformation. In: Lameness in Cattle, 3rd edition. W.B. Saunders Company, Philadelphia. Pp 75-8
66. McLennan MW (1988). Incidence of lameness requiring veterinary treatment in dairy cattle in Queensland. Australian Veterinary Journal 65:144-7
67. Melendez PJ, et al (2002). Relationship between lameness, ovarian cysts and fertility in Holstein cows. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 339-42
68. Midla LT, et al (1998). Supplemental dietary biotin for prevention of lesions associated with aseptic subclinical laminitis (pododermatitis aseptica diffusa) in primiparous cows. American Journal of Veterinary Research 59:733-8
69. Miles RD, Henry PR (1999). Relative trace mineral bioavailability. Proceedings of the California Animal Nutrition Conference, Fresno (California). Pp 1-24
70. Mill JA, Ward WR (1993). Lameness in dairy cattle and farmers' knowledge, training and awareness. Veterinary Record 134:162-4
71. Mungall BA, et al (2002). In vitro evidence for a bacterial pathogenesis of equine laminitis. Veterinary Microbiology 65:209-23
72. Murray RD, et al (1996). Epidemiology of lameness in dairy cattle: description and analysis of foot lesions. Veterinary Record 138:586-91
73. National Animal Health Monitoring System (1996). Dairy 1996. Part I: Reference of 1996 Dairy Management Practices. USDA-Animal Plant Health Inspection. Serv-Vet. Serv., Fort Collins, Co
74. Nocek JE, et al (2000). Digital characteristics in commercial dairy herds fed metal-specific amino-acid complexes. Journal of Dairy Science 83:1553-9
75. O'Callaghan K (2002). Lameness and associated pain in cattle - challenging traditional perceptions. In Practice 24:212-9
76. O'Callaghan KA, et al (2002). Behavioural indicators of pain associated with lameness in dairy cattle. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 309-12
77. Offer JE, et al (1997). The effect of protein source on lameness and sole or lesion formation in dairy cattle. Animal Science 65:143-9
78. Offer J, et al (2003). Effect of forage type on claw horn lesion development in dairy heifers. The Veterinary Journal 165:221-7
79. Ossent P (2000). Bovine laminitis: the lesions and theories on their pathogenesis. Proceedings of the XI International Symposium on Disorders of the Ruminant Digit, Parma, Italy. Pp 31-3
80. Ossent P, Lischer C (1998). Bovine laminitis: the lesions and their pathogenesis. In Practice 20:415-27
81. Peeler EJ, et al (1994). Inter-relationships of periparturient diseases in dairy cows. Veterinary Record 134:162-4
82. Phillips CJC, et al (2001). The effects on production and behaviour of mixing uniparous and multiparous cows. Journal of Dairy Science 84:2424-29
83. Potter MJ, Broom DM (1990). Behaviour and welfare aspects of cattle lameness in relation to the building design. Proceedings of the VI International Symposium on Diseases of the Ruminant Digit, Liverpool, UK. Pp 80-4
84. Paulus N, Nuss K (2002). Claw measures at defined sole thickness. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 428-30
85. Rajkondawar P, et al (2002). A system for identifying lameness in dairy cattle. Applied Engineering in Agriculture 18(1):87-96
86. Robinson PH (2001). Locomotion scoring cows. California Dairy
87. Russell AM (1987). The influence of sire on lameness in cows. Proceedings of British Cattle Veterinary Association 1986/7. Pp 213-9
88. Russell JB, Garner MR (2003). Allisonella histaminiformans, a novel histamine-producing bacterium that may play a role in rumen disorders and bovine laminitis. In: Nutritional Biotechnology in the Feed and Food Industries. Nottingham University Press, Nottingham. Pp 309-13
89. Sauter-Louis CM, et al (2004). A longitudinal study of the relationship between the occurrence of lameness and herd dominance related behavioural factors in New Zealand dairy cows. New Zealand Veterinary Journal (in press)
89. Seegers H, et al (1998). Reasons for culling in French Holstein cows. Preventive Veterinary Medicine 36:257-71
91. Shaver RD (1997). Nutrition. In: Lameness in Cattle, 3rd edition. W.B. Saunders Company, Philadelphia. Pp 293-7
92. Singh S, et al (1993a). Behaviour of lame and normal dairy cows in cubicles and on a straw yard. Veterinary Record 133:204-8
93. Singh S, et al (1993b). Behaviour of first lactation and adult dairy cows while housed and at pasture and its relationship with sole lesions. Veterinary Record 133:469-74
94. Singh S, et al (1993c). Aetiology and pathogenesis of sole lesions causing lameness in cattle: A review. Veterinary Bulletin 133:469-74
95. Singh S, et al (1994). Behaviour of dairy cows in a straw yard in relation to lameness. Veterinary Record 135:251-3
96. Smart M, Cymbaluk NF (1997). Role of nutritional supplements in bovine lameness - Review of nutritional toxicities. In: Lameness in Cattle, 3rd edition. W.B. Saunders Company, Philadelphia. Pp 149-61
97. Somers JGCJ, et al (2003). Prevalence of claw disorders in Dutch dairy cows exposed to several floor systems. Journal of Dairy Science 86:2082-93
98. Spears JW (1996). Organic trace minerals in ruminant nutrition. Animal Feed Science and Technology 58:151-63
99. Spencer E (2001). Heat stress and lameness.

Newsletter of the Hoof Trimmers Association Inc. Pp 8-9
100. Sprecher DJ, et al (1997). A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. Theriogenology 47:1179-87
101. Tarlton J (2003). Biochemical, histopathological and biochemical mechanisms of lameness associated with first calving. Cattle Practice 11(2):81-7
102. Tarlton JF, Webster AJF (2002). A biochemical and biomechanical basis for the pathogenesis of claw horn lesions. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 395-8
103. Toussaint-Raven E, et al (1989). Cattle Foot Care and Claw Trimming. Farming Press, Ipswich, UK
104. Tranter WP, Morris RS (1991). A case study of lameness in three dairy herds. New Zealand Veterinary Journal 39:88-96
105. Vaarst M, et al (1998). Sole disorders in conventionally managed and organic dairy herds using different housing systems. Journal of Dairy Research 65:175-86
106. Van Amstel SR, et al (2002). Anatomical measurement of sole thickness in cattle following application of two different trimming techniques. The Bovine Practitioner 36(2):136-40
107. Van Dorp TE, et al (1998). Genetic parameters of health disorders and relationships with 305 day milk yield and conformation traits of registered Holstein cows. Journal of Dairy Science 81:2264-70
108. van Keulen KAS, et al (1992). The antimicrobial activity of two tetracyclines and lincomycin in walk-through footbaths for dairy cattle: A longitudinal study. Proceedings of the VII International Symposium on Disorders of Ruminant Digit, Rebilt, Denmark. (Abstract No 16)
109. Vermunt JJ (1992). "Subclinical" laminitis in dairy cattle. New Zealand Veterinary Journal 40:133-8.
110. Vermunt JJ (1999). Regular claw trimming for the control of lameness - good or bad? The Veterinary Journal 157:109-10
111. Vermunt JJ, Greenough PR (1994). Predisposing factors of laminitis in cattle. British Veterinary Journal 150:151-64
112. Vermunt JJ, Greenough PR (1997). Management and control of claw lameness - an overview. In: Lameness in Cattle, 3rd edition. W.B. Saunders Company, Philadelphia. Pp 308-15
113. Vokey FJ, et al (2002). Effects of alley and stall surfaces on indices of claw and leg health in dairy cattle housed in a freestall barn. Journal of Dairy Science 84:2686-99
114. Ward WR, French NP (1997). (Abstract) Proceedings of Annual Conference of Association of Veterinary Teachers and Research Workers
115. Warnick LD, et al (2001). The effect of lameness on milk production in dairy cows. Journal of Dairy Science 84:1988-97
116. Wells SJ, et al (1993). Individual cow risk factors for clinical lameness in lactating dairy cows. Preventive Veterinary Medicine 17:95-109
117. Wells SJ, et al (1995). Some risk factors associated with clinical lameness in dairy herds in Minnesota and Wisconsin. Veterinary Record 136:537-40
118. Wells SJ, et al (1999). Papillomatous digital dermatitis and associated risk factors in US dairy herds. Preventive Veterinary Medicine 38:11-24
119. Westwood CT, Lean IJ (2001). Nutrition and lameness in pasture-fed dairy cattle. Proceedings of the New Zealand Society of Animal Production 61:128-34
120. Westwood CT, et al (2003). Review of the relationship between nutrition and lameness in pasture-fed dairy cattle. New Zealand Veterinary Journal 51:208-18
121. Whay HR, et al (1997). Associations between locomotion, claw lesions and nociceptive thresholds in dairy heifers during the post-partum period. The Veterinary Journal 154:155-61
122. Whay HR, et al (1998). The influence of lesion type on the duration of hyperalgesia associated with hind limb lameness in dairy cattle. The Veterinary Journal 156:23-9
123. Whay HR, et al (2002). Farmer perception of lameness prevalence. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando (Florida). Pp 355-8
124. Wierenga HK (1991). Behaviour of dairy cows under modern housing conditions. PhD thesis, Agricultural University, Wageningen, the Netherlands

EVALUATION OF A DUTCH CLAW HEALTH SCORING SYSTEM IN DAIRY CATTLE.

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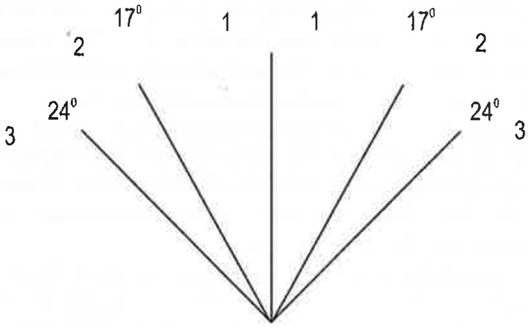
Abstract

To determine the optimal moment for claw-trimming of dairy cows, a scoring system was developed by the Faculty of Veterinary Medicine of Utrecht University, The Netherlands: the leg score. The leg score is a three-point scoring system, based on the external rotation of the hind feet relative to the spinal column. By calculating the percentages of leg score 1 to 3 per herd, herd claw health is summarized in a single variable. Per scoring event and dependent on the cut-off values used in practice, the advice is given to have the herd claw trimmed or not. Although the leg score is widely used in practice, there has not been a quantitative evaluation of its repeatability and its reproducibility. The study was performed as a first step towards evaluation of this diagnostic tool.
To assess the repeatability, the score was performed twice with a scoring-scale and twice without such scoring scale by 11 observers on 52 cows in a dairy herd and kappa values were calculated.

To assess reproducibility, the score was performed twice, once with and once without a scoring scale on 2 dairy herds with 62 and 50 cows by 8 observers.
Concerning the reproducibility, the kappa value varied from 0.170-0.658 for scoring with the scoring scale and from 0.121-0.577 for scoring without the scoring scale and only 11% of cows were assigned the same score by all 8 observers.
Evaluating the advice to the farmer: in the investigated herd the trimming advice to the herdsman, based on over 20% of the animals showing score 3 and when scoring with the scale, was consistent for 3 out of 11 observers. When scoring without a scale, the advice to have the dairy cows trimmed was consistent for 4 of 11 observers. The advice to have the dairy cows not trimmed, when scoring with a scale, was consistent for 6 of 11 observers. When scoring without a scale, the advice to have the dairy cows not trimmed was consistent for 5 of 11 observers. The advice of 2 observers was inconsistent for both scoring with as scoring without a scale.
It was concluded that the repeatability of the leg score performed with or without a scoring scale,
varied from good to bad depending on the observer and that the scoring scale had no additive value.
In a first step toevaluating the leg-score further research is needed about the reasons for differences of scoring in relation to the (in)consistency of the exo-rotation based score of the hind legs and what changes are needed to improve the quality of this diagnostic instrument.



The present study was undertaken as a first step in evaluating the leg-score, to assess the repeatability, defined as the degree of agreement between sets of observations made on the same animals at the same moment by the same observer, and the reproducibility, defined as the degree of agreement between sets of observations made on the same animals at the same moment by different observers (Thrusfield, 1995). Observers were professionals (practitioners) and one well-trained student and the result was a herd-score that was related to the advice for herd trimming. That herd advice is discussed in relation to three different cut-off values: score 1 $\leq$ 40%, score 3 $\geq$ 20% and score 2+3 $\geq$ 50%.

Introduction

Lameness and claw-health are of increasing importance in herd-health management. Lameness is an increasing problem, comparing recent studies on the prevalence of lameness in dairy cows (e.g. Somers et al, 2003). In The Netherlands it is estimated that in about 60% of the dairy herds all lactating cattle are trimmed twice a year by a professional claw-trimmer. These trimmings are usually performed at the end of the winter period and at the end of the summer/ grazing period, and areindependent of the claw health condition.

For assessing the optimal moment of "herd trimming", the leg-score was developed (J.J. van Amerongen, personal communication).
The leg-score is determined during square standing by the angle of the spinal column and the interdigital space of the hind claws. The leg-score is divided into 3 classes. If the observed angle is between 0-17 degrees, it is scored as 1, an angle between 18-24 degrees is scored as 2 and an angle scored >24 degrees and any lame cow is scored as 3 (Fig.1).
The distribution of the percentages of scores 1, 2 and 3 (= the herd score) is recommended as a tool for an "objective" determination of the best moment for claw trimming the whole dairy herd.

Material and methods

Animals and data collection
For the estimation of the reproducibility, the leg-scores were recorded twice in two dairy herds (farm A and farm B, with respectively 62 and 50 cows > 2 years, breed Holstein Friesian). When recording, the cows were fixed to the feed bunk. Between recordings cows were re-mixed. The leg-scores were performed once with a scale and once without by 8 trained observers at the end of the housing period (March 2003), about 2 months after the whole milking herd was trimmed.
For estimation of the repeatability of the leg-score, the scores of all cows > 2 years were recorded twice with and twice without a scoring scale in farm B by 11 trained observers, one month after the scoring for estimation of the reproducibility (April 2003).
Statistical analysis
At animal level, the reproducibility of the leg score was estimated by comparing the 2 results (score once with and once without a scoring scale) on 2 herds (farm A and farm B) of the different observers. As a proportion of agreement between scores the kappa value was estimated (Noordhuizen et al.,2002). The analyses were performed using Winepiscope 2.0 and Statistix 7.
The repeatability at animal level was assessed by comparing the 2 results of scoring with scale and the 2 results of scoring without a scale. This was again expressed by computing the kappa value. In addition, the absolute differences (0, 1 or 2) of the leg-scores between the duplicates were calculated for the recordings with and without the scale. These differences were reduced to one dependent variable y: y = 0 if the absolute differences were null and y = 1 if the absolute differences were 1 or 2 (binom-

inal distribution). The probability p of y = 0 was estimated taking the factors: cow (random, n = 52), observer (fixed or random, n = 11) and scoring method (fixed, n = 2) into account. The dispersion was kept fixed at 1. For this analysis the GLMM-procedure (for single effect observer and method) was used whereas the IRREML-estimation technique was used for the interaction of observer*method. The analyses were performed using Winepiscope 2.0 and Genstat.

The repeatability of the claw score at herd level was assessed by the McNemar symmetry test based on different cut off values for leg score 1 to 3, while the Chi-square was used to evaluate the effect of the method and observer (Statistix 7).

Results

Reproducibility: the resulting equal outcomes of the leg score (with and without a scale) and the numbers of animals involved are presented in Table 1. Kappa values for all possible pairs of observers are presented in Table 2.

Table 1: Reproducibility of the leg-score based on the unanimous and different scores estimated with and without the scale as performed by 8 veterinarians.

Herd	Scoring-scale	Same score (%)	2 different scores (%)	3 different scores (%)	N
1	Yes	9.1	61.8	29.1	55
2	Yes	10.9	54.3	34.8	46
1	No	5.2	60.3	34.5	58
2	No	19.1	55.3	25.5	47

Table 2: Reproducibility outcomes (kappa's) between the different observers on herd 2; at the left side of the table the values for scoring with a scale and at the right side for scoring without a scale

	1	2	3	4	5	6	7	8
1	xxx	0.216	0.382	0.367	0.251	0.485	0.247	0.156
2	0.181	xxx	0.351	0.276	0.257	0.182	0.124	0.228
3	0.343	0.165	xxx	0.221	0.285	0.198	0.227	0.072
4	0.239	0.181	0.204	xxx	0.300	0.267	0.114	0.081
5	0.264	0.197	0.239	0.343	xxx	0.334	0.180	0.100
6	0.101	0.105	0.142	0.133	0.345	xxx	0.114	0.209
7	0.314	0.027	0.089	-0.081	0.107	0.083	xxx	0.481
8	0.354	0.178	0.177	0.114	0.110	0.082	0.430	xxx

Repeatability: the estimated kappa of the leg-score, estimated on herd 2 in April (twice scoring with and without the scale) and the number of animals involved are presented in Table 3.

Table 3: Repeatability of the leg-score estimated for scoring with the scale (kappa 1) and estimated for scoring without the scale (kappa 2). Nr. of animals is the number of animals scored twice with or without with the scale.

		With scale		without scale	
Observer	profession	kappa 1	N	kappa 2	N
1	vet	0.26	43	0.41	45
2	vet	0.18	46	0.33	46
3	student	0.66	43	0.43	45
4	vet	0.28	43	0.39	45
5	vet	0.39	45	0.37	46
6	vet	0.17	43	0.33	42
7	vet	0.51	42	0.31	43
8	vet	0.41	41	0.24	41
9	vet	0.33	43	0.58	45
10	vet	0.43	40	0.12	31
11	vet	0.37	45	0.37	44

Reasons for missing values in the data set were "double I&R numbering" in the same herd, missing scores, miscoding of numbers and other human errors.

In general kappa values are interpreted as no agreement between observations when kappa is equal to 0 and perfect agreement when kappa value is equal to 1. For practical use kappa values between 0.4 and 0.5 are indicated as moderate, values between 0.5 and 0.6 as sufficient and values between 0.6 and 0.8 as good.

The probability of the same result for each observer is presented in Table 4. These probabilities were comparable for scoring with and without a scale. On average, the probability of a same result was 0.61. The probability of the same result was not significantly different between the methods of scoring, the observers scoring or the interaction between observer*method (χ^2-tests, all P> 0.10).

Table 4. The probability of a same result betweem duplicates)n= 52 cows)

Observer		Probability on a same result between duplicates
1	Vet	0.58
2	Vet	0.58
3	Trained student	0.72
4	Vet	0.57
5	Vet	0.60
6	Vet	0.53
7	Vet	0.63
8	Vet	0.60
9	Vet	0.70
10	Vet	0.58
11	Vet	0.61

Advice at herd level.

The derived advice for the herdsman to have the dairy-cows trimmed or not is presented in Table 5 for the option leg score 3 $\geq$20% (with and without scale). Evaluating the advice to the farmer: in the investigated herd the trimming advice to the herdsman, 2 months after the last herd trimming and when scoring with the scale, was consistent for 3 out of 11 observers. When scoring without a scale, the advice to have the dairy cows trimmed was consistent for 4 of 11 observers. The advice to have the dairy cows not trimmed, when scoring with a scale, was consistent for 6 of 11 observers. When scoring without a scale, the advice to have the dairy cows not trimmed consistent within 5 of 11 observers. The advice of 2 observers was not consistent when scoring with as scoring without a scale.

Table 5. The advice for the herdsman to have or have not the milking-cows trimmed, based on the results of the leg score with and without the scale.

Border value/ Observer	Leg score 3 ≥ 20% with a scale		Leg score 3 ≥ 20% without a scale	
1	No (14.0)	Yes (22.2)	Yes (25.5)	No (8.9)
2	No (13.0)	No (12.8)	No (10.9)	No (12.5)
3	Yes (37.0)	Yes (36.6)	Yes (34.8)	Yes (30.4)
4	Yes (20.5)	Yes (29.8)	Yes (38.3)	Yes (41.3)
5	Yes (29.8)	Yes (28.9)	Yes (25.5)	Yes (28.3)
6	No (8.5)	No (15.9)	No (17.0)	Yes (23.3)
7	No (13.3)	No (19.1)	Yes (22.7)	Yes (20.5)
8	No (9.8)	No (11.9)	No (14.6)	No (11.9)
9	No (8.9)	No (4.7)	No (11.1)	No (6.5)
10	No (15.2)	No (14.3)	No (5.6)	No (12.5)
11	No (11.1)	Yes (24.4)	No (17.8)	No (11.4)
Results (unanimous)	Yes= 3, No= 6		Yes= 4, No= 5	

Discussion

As a first step in evaluating the leg-score, the repeatability and the reproducibility were estimated. The results of the repeatability (Table 2) are in agreement with the results of Boisot et al. (2002). In that study the repeatability of objective measurements (length and angles) on the rear legs of dairy cows was estimated to see whether higher repeatability estimates could be obtained compared to the corresponding linear score. In the study of Boisot, the repeatability for the angles was even lower (0.00-0.24). The probability of a same result was in our study 0.61 and this should be increased before this method of scoring is suitable for practical application about having a good impression about the claw condition. One possibility for improvement could be the period between standing at the feed-bunk and moment of scoring; another could be the relation between the observer and the scored dairy-cows (more or less restless).
Based on a single observation, the resulting advice of the leg-score (Table 4) to the herdsman to have the milking herd trimmed or not (3 to 6 when scoring with the scale and 4 to 5 when scoring without the scale) it can be concluded that this system is not yet suitable as a diagnostic herd aid. For having good impressions about the soundness of the claws locomotion score or gathering information at herd trimming is advisable, as long as those parameters are tested also in relation to reproducibility and repeatability.

To conclude, further longitudinal research is needed to investigate the reason for different scoring results within the same cows within 30 minutes (time in this investigation measured between 2 scores), and to investigate additional criteria (mentioned above) that can improve the quality of this instrument to have objective criteria for the optimal moment for herd trimming and to quickly have information about the claw's condition.
Above that, research to investigate the variations of the leg-score in relation to different claw-lesions is advisable.

Literature

Boisot, P.O., Rodriguez-Zas, S.L. and Shanks, R.D. 2002. Repeatability of objective measurements on the rear legs of dairy cows. J. Dairy Sci. 85:2344-51.
Brand, A., Noordhuizen, J.P.T.M. & Schukken, Y.H. 1996. Herd health and production management in dairy practice. Wageningen Pers. Chapter 7 part 1.
Noordhuizen, J.P.T.M., Frankena, K., Hoofd, van der, C.M. and Graat E.A.M. Application of Quantitative Methods in Veterinary Epidemiology. Wageningen Pers, Wageningen, The Netherlands, 2002.
Somers J.G.J.C., Frankena K, Noordhuizen-Stassen E.N. en Metz J.H.M. 2003. Prevalence of claw disorders in Dutch dairy cows exposed to several floor systems. J. Dairy Sci. 86: 2082-93.
Thrusfield, M. Veterinary Epidemiology, second edition 1995, page 135

MEASURING THE INITIAL CONCENTRATION OF FORMALIN IN WALK-THROUGH FOOTBATHS IN DAIRY HERDS.

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Abstract

In a field study the changes in formalin concentration in walk-through footbaths in 18 dairy herds (n = 66.8, [40-140]) were determined by sampling 3 consecutive days. The footbaths were sampled just after preparation and every time, immediately after the dairy cows had passed the footbaths. The concentration was estimated by sampling, immediately filtering and analysing (gas-chromatography) the formalin-concentration in the samples from the walk-through footbaths. The initial concentration was reduced by 50% after 2,4 days and this is in this study comparable with 300-320 cow passages. The milking cows' enthusiasm for walking through the footbaths did not influence the decrease of the formalin concentration.

Introduction

Healthy feet and legs are of paramount importance to the cow for optimal productivity, health and animal welfare (Brand et al.,1996). Lameness is foremost associated with claw-problems (Weaver, 2000). Epidemiological research on claw disorders in dairy cattle indicates that the main infectious claw diseases resulting in hoof lesions and lameness, are digital and interdigital dermatitis (e.g.Somers et al., 2003). Since the use of antibiotics in footbaths is banned, many farmers are advised to use chemical disinfectants in footbaths like Copper Sulphate, Zinc Sulphate, formalin and their combinations for the prevention and treatment of (infectious) claw-problems. In spite of some disadvantages (for example irritation of the conjunctivae and epithelium of the respiratory tract of the farmer/milker) formalin-footbaths have been advised ever since Toussaint-Raven (1989). A lot of these original recommendations, especially the walk-through baths are still practised although the herd management has changed and the size of the dairy herds has increased enormously.
The objective of this field study was to estimate the changes in formalin concentration in walk-through footbaths in dairy herds by sampling before and every time immediately after the last dairy cows had passed the footbaths. The influence of the milking cows' enthusiasm of walking through the footbaths was also evaluated.

Material and methods

Selection and sampling.
Between December 2000 and September 2001,18 dairy herds with cubicles and a walk-through footbath with a formalin disinfectant, near the Animal Health Service (AHS) in Deventer, were visited and sampled 7 times. During the herds' first visit the capacity of the bath was calculated, the number of animal passages and the behaviour of the cows while walking through the footbath were observed. A first sample for measuring the formalin concentration was taken immediately after preparation by the farmer and before cows had passed through the footbath.
In all herds the footbath was situated in the cows' route on the exit lanes from the milking parlour to the free stall barns. The cows walked twice a day through the footbath, filled to a depth of 100 -120 mm and after the passage the cows were kept on scraped floors. The claws were not systematically cleaned before passing the footbath and the bath was not filled up during the field study. The farmers used the footbath both as a preventive and curative for digital infections during this experiment.
Analysis of the samples.
Before taking the samples out of the bath, the contents of the footbath were homogenised as much as possible. The sample-bottle was filled completely, to minimise possible gas production. As the footbaths contain many particles (faeces, sawdust and so on), which may influence the concentration, the samples were filtered immediately. In spite of removing the solid matter, the formaldehyde could still react with other agents present, which might also influence the concentration. To inhibit the production of formaldehyde radicals, 1 ml methanol was added to the filtered sample. While the morning samples were delivered within 2-3 hours to the laboratory for further analyses, the evening samples were stored at room temperature and delivered the next morning (after 14-16 hours).
Developing the method of analysis.
The method used for analysis and determination of formaldehyde concentration was developed in the AHS laboratory. With a gas-chromatographic technique, the present components in the samples were separated at an apolair capillary column and were shown and quantified with a flame-ionisation with an auto sampler. Before the experiment this method was validated for the field study. Eventually disturbing components that would influence the analysis were not found.
Validation of the technique included, for example, the production of para-formaldehyde and the possible conversion of para-formaldehyde into formaldehyde during analysis. These phenomenon were checked in the analytical method and could not be measured.
Statististical analysis.
The data were analysed by using Sigma Plot (Sigma Plot for Windows 4.01, SPSS Inc.). The half value time (t = ") was estimated based on the equation: y = f(a x e (-bx)), of which y is the formalin concentration in % and x is the time since preparation of the bath. f, a and b are constant values based on the results.

Results

The contents and type of footbaths (disposable (d) or not disposable (nd)) and the behaviour of the cows while going through the baths (very quiet (v), quiet (q) and unquiet or restless (u)) is shown in Table 1. Table 2 shows the formalin concentration measured in the different samples and time (in min.) after preparing the footbath. Figure 1 demonstrates the formalin concentration (with deviation) related to the moment of sampling. Based on this figure, a half-value time can be estimated as 2,40 days (=3460 min.). This means that after this time under average conditions the initial concentration had decreased to under 50%. Table 3 demonstrates the formalin concentration related to the behaviour of the cows through the footbath. In relation to the low number of herds (u and v) it was not possible to draw further conclusions.

Table 1. An overview of the contents (in litres) and type (d= disposable and nd= not disposable) of the used footbaths, the number of cows passing the footbath and the behaviour of the cows while walking through the footbath (Q= quiet, U= unquiet and V= very quiet) in the different dairy herds.

Herd	Contents of bath (l)	Kind of bath	Number of cows passing the bath	Behaviour
1	200	nd	40	Q
2	400	nd	51	U
3	600	d	50	Q
4	370	d	60	U
5	270	nd	65	Q
6	382	nd	64	Q
7	240	d	57	V
8	168	d	65	Q
9	200	nd	50	Q
10	930	nd	100	U
11	257	d	58	Q
12	170	d	58	Q
13	160	nd	73	Q
14	290	nd	85	Q
15	110	nd	140	Q
16	150	nd	62	Q
17	144	nd	40	Q
18	131	d	85	Q

Table 2. The measured formaline concentration in the different samples in relation to the time after preparing the footbath.

	Time of sampling (in min.)						
Herd	1	2	3	4	5	6	7
1	0,9/ 0	2,7/ 105	2,2/ 810	1,7/ 1545	1,4/ 2370	1,1/ 3105	0,8/ 3930
2	1,9/ 0	2,6/ 75	2,7/ 870	2,3/ 1480	2,1/ 2250	1,8/ 2925	1,6/ 3690
3	1,8/ 0	3,1/ 120	3,1/ 900	3,1/ 1560	2,7/ 2325	2,8/ 3000	2,9/ 3765
4	6,5/ 0	6,9/ 105	6,5/ 745	6,3/ 1440	6,4/ 2175	6,2/ 2880	5,1/ 3615
5	9,6/ 0	3,3/ 210	3,2/ 990	3,0/ 1650	2,8/ 2490	2,6/ 3090	2,4/ 3870
6	2,0/ 0	1,2/ 600	1,6/1260	1,3/ 2070	1,8/ 2700	2,2/ 3480	2,1/ 4140
7	4,3/ 0	3,8/ 180	3,5/ 900	3,3/ 1560	3,0/ 2340	2,1/ 3015	2,3/ 3780
8	3,2/ 0	2,6/ 90	1,9/ 930	1,0/ 1545	1,0/ 2370	0,7/ 2970	0,4/ 3810
9	1,8/ 0	3,4/ 120	3,1/ 855	2,8/ 1560	2,9/ 2295	2,4/ 2985	2,3/ 3720
10	3,4/ 0	2,6/ 180	2,1/ 630	1,5/ 1185	0,9/ 1560	1,0/ 2010	0,8/ 2580
11	3,8/ 0	3,7/ 78	3,5/ 798	3,2/ 1488	3,0/ 2268	2,0/ 2913	2,4/ 3678
12	2,7/ 0	2,8/ 75	2,2/ 765	2,4/ 1485	1,9/ 2235	1,7/ 2895	1,4/ 3645
13	4,0/ 0	3,4/ 135	2,8/ 885	1,6/ 1575	1,1/ 2325	0,3/ 3030	0,5/ 3765
14	5,5/ 0	5,0/ 120	3,9/ 810	3,4/ 1560	2,6/ 2250	2,3/ 3000	1,7/ 3660
15	5,5/ 0	3,3/ 180	1,7/ 960	1,0/ 1620	0,5/ 2400	0,1/ 3060	0,8/ 3840
16	2,5/ 0	2,6/ 150	2,2/ 900	1,9/1560	1,6/ 2325	1,5/ 3000	1,2/ 3765
17	5,0/ 0	4,7/ 145	4,3/ 905	3,7/ 1580	3,4/ 2370	3,0/ 3035	2,4/ 3805
18	4,5/ 0	3,5/ 120	2,9/ 970	2,0/ 1580	1,4/ 2405	1,2/ 3045	1,0/ 3850

Table 3. The formalin concentration related to the behaviour of the cows while walking through the footbath.

	Mean values.						
	1	2	3	4	5	6	7
Quite (n=14)	3,6	3,2	2,8	2,4	2,2	2,0	1,9
Unquiet (n=3)	3,9	4,0	3,8	3,4	3,1	3,0	2,5
Very Quite (n=1)	4,3	3,8	3,5	3,3	3	2,1	2,3

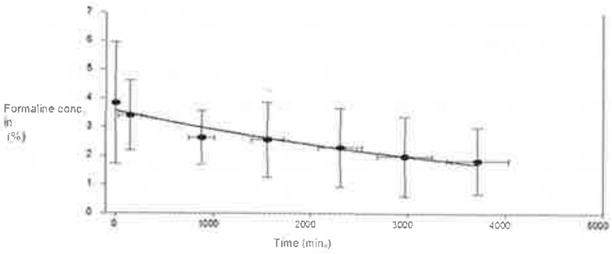

Fig. 1 The mean formalin concentration with the deviation related to the time (in min.)

Discussion

The results of this field study, with a 50% decrease of the initial concentration after 2,40 days (300-320 cow passages) are comparable with other studies (e.g. Berry et al., 1997), which also advised renewal of every footbath after 200-300 passages. This means that under normal circumstances in the Netherlands (medium herd size 57 cows (CBS, 2002)), every 2 days (4 cow passages) the bath has to be renewed, to guarantee enough activity.

Based on the results of the footbath volume (Table 1) and the concentration in the first samples just after preparing the bath (Table 2) much attention must paid to the dimensions and the initialconcentration. Fifty percent of the footbaths had a capacity of less than 250 litres, which means the baths were usually too short or not high enough..

The great differences in decrease of the footbath concentration could probably be related to the cleanliness of the hooves at the start of the footbath and the amount of defecation in the bath. In the Netherlands it is uncommon to make use of two footbaths (e.g. Blowey,1993), when a first bath can clean the claws and the cows can defecatet. When cows go through the second bath, disinfection is more effective and there is less change in the concentration. So a dual foot-bath is probably advisable.

To conclude, under the present Dutch circumstances (medium herd size of 57, cows on a slatted floor) attention must be paid to the initial concentration and it is advisable to refresh the footbaths after 200-250 cow passages.

References

Berry S.L. and Maas J. 1997. Clinical treatment of papillomatous digital dermatitis (footwarts) on dairy cattle. Proceedings of the 1997 Hoof Health Conference, Batavia, NY, 4-7.

Blowey, R. 1993. Cattle lameness and Hoofcare. An illustrated Guide. Farming Press, Ipswich IP1 4LG, United Kingdom, p. 76.

Brand, A., Noordhuizen, J.P.T.M. and Schukken, Y.H. 1996. Herd health and production management in dairy practice. Wageningen Pers. The Netherlands. Chapter 7 part 1.

CBS. 2002. Agricultural Census 2002. Statistics Netherlands, ed. Elsevier, Voorburg, The Netherlands.

Somers, J.G.C.J., Frankena, K., Noordhuizen-Stassen, E.N. and Metz, J.H.M. 2003. Prevalence of claw disor-

ders in Dutch dairy cows exposed to several floor. J. Dairy Sci. 86:2082-2093.
Toussaint Raven, E. 1989. Cattle Footcare and Claw Trimming. Farming Press, Ipswich IP1 4LG, United Kingdom.
Weaver, A.D, 2000. Lameness.. Pages 149-202 in The Health of Dairy Cattle. A.H. Andrews, ed. Blackwell Science, Oxford, United Kingdom, p. 149-202.

A CROSS-SECTIONAL STUDY OF CLAW LESIONS AND RISK FACTORS IN DANISH HOLSTEINS

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Introduction

Danish dairy production is going through dramatic changes from small tie stall herds to large loose housed herds. There was an increase in loose housing systems from 37,5% to 47,8% during 2001. New barns are being built, production increases and the herd size increased from 98 to 107 cows during 2001 (Lauritsen and Lind 2003). Intensive production and loose housing presumably result in an increased occurrence of claw lesions. (Enevoldsen et al 1991a,Enevoldsen et al 1991b,Murray et al 1996,Rodriguez-Lainz et al 1999,Stanek 1997,Vermunt and Greenough 1994,Wells et al 1993). Pododermatitis is associated lameness detected more easily in systems where cows can move more freely Consequently, we need to update our knowledge on the relationship between claw lesions and housing or management factors in Danish dairy herds. The objectives of this study were to estimate the prevalence of claw lesions in Danish Holsteins kept in loose housing systems and to identify predisposing herd-level factors. The study was performed in the winter of 2002-2003 because the claw lesions generally are expected to increase during the cold and wet season when animals are kept indoor at all times.

Material and methods

This cross-sectional study was performed during the winter from November to March 2002-2003 in Denmark. We wanted to obtain information on the predominant herd typ of the future within dairy production in Denmark. Therefore we chose Holstein in larger loose housing systems.
In order to get the most reliable data we wanted trimmers to volunteer for the study. Unfortunately only four trimmers wanted to participate. The claw trimmers collected data from all the herds they trimmed and which fitted the required characteristics during the study period. Only the routine trimmings of a large part of the herd were included. This resulted in a total of 52 herds and 6500 cows. The datasheet used for collecting claw data included lameness score, but only severely lame cows were recorded because of the conditions and limited space around the trimming chute. The trimmers recorded all skin and horn lesions related to the distal part of all four limbs. This included heel horn erosion (HHE), solar haemorrhage (SH), sole ulcer (SU), interdigital dermatitis (ID), digital dermatitis (DD), white line disease (WLD), abscess, interdigital hyperplasia (IH), and double sole (Dbs) . The lesions and abnormalities were evaluated during and at the completion of trimming. We thus obtained records of both new and older lesions.
In each of the 52 dairy herds, milk quality advisors employed by the Danish Dairy Board collected farm level information at herd visits. The information collected on the farm was largely detailed. However, only limited information could actually be used in the analysis because of too little variation in the data. Housing and management factors included in the analysis were: type and amount of bedding, hygienic condition of the floor and the cubicles, feeding routines, the size of the herd, footbathing routines, trimming routines, and whether the cows were grazing or not.
Housing factors included were; new housing system build within the last 5 years, uncomfortable cubicles, and type, structure and quality of flooring. Data were analysed by means of a multilevel random effects logistic regression model and common factor analysis.

Results and discussion

The main results of our study are presented in tables 1 to 2.

Table 1. Across-herd prevalence of claw lesions in 6240 loose housed Danish Holsteins

Variable	Proportion
Lameness	0.02
Heel horn erosion	0.54
Solar haemorrhage	0.59
Sole ulcer	0.06
Interdigital dermatitis	0.25
Digital dermatitis	0.22
Interdigital dermatitis	0.05
Abscess	0.01
White line disease	0.05
Double sole	0.06

Table 2. The prevalence of claw disorders in Danish Holsteins - the distributions of the no. of recordings and herd-level prevalence and variance components associated with the herd level (random herd effect estimated from logistic regression.

Variable	10th Pctl	25th Pctl	50th Pctl	75th Pctl	90th Pctl	Random herd effect, %
Herd size	48	83	101	125	191	...
Lameness	0.00	0.00	0.02	0.04	0.06	13
Heel horn erosion	0.04	0.16	0.44	0.80	0.92	65
Solar haem-orrhage	0.23	0.31	0.57	0.70	0.89	30
Sole ulcer	0.00	0.02	0.05	0.08	0.14	9
Interdigital dermatitis	0.02	0.08	0.20	0.42	0.54	29
Digital der-matitis	0.00	0.07	0.16	0.28	0.38	21
Interdigital hyperplasia	0.00	0.01	0.03	0.06	0.12	14
Abscess	0.00	0.00	0.00	0.01	0.01	37
White line disease	0.00	0.01	0.03	0.06	0.11	14
Double sole	0.00	0.02	0.04	0.07	0.12	9

The random herd effects (Table 2) indicate the contributions from the herd factors including claw trimmer to variability in the occurrence of claw lesions. The table shows that the occurrence of heel horn erosion is particularly influenced by risk factors on herd-level and that horn related lesions like sole ulcer primarily are related to cow-level risk factors. In this analysis the cow characteristics have not been included. A factor analysis of the data showed a moderately strong relationship between slippery, wet floors and heel horn erosion. There was a relationship between new stalls and solid concrete floors and between large herds (>121), zero grazing and automatic concentrate feeding.

The prevalence of herds with digital dermatitis in this study is 79% (7 of 52 herds did not have any cows with clinical DD lesions). This shows that digital dermatitis has spread throughout Denmark since the early eighties (Blom 1996,Enevoldsen et al 1991a). Infectious diseases like digital dermatitis and interdigital dermatitis are becoming more common and cause serious lameness in Danish herds.

We expect that the type of flooring in loose housing systems affects the prevalence of claw lesions. However, the results from this study do not show significant and clear relationship between diseases and between diseases and all herd-level risk factors. Consequently, most of the claw diseases probably are more affected by cow-level factors than by herd-level factors. This indicates a need for analysis of cow-characteristics like lactation stage, age and production level.

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References

Blom JY. Prevalence of digital dermatitis in Danish dairy herds. 9th International Symposium on disorders in the Ruminant Digit. . [Jerusalem 1996], 291-292. 1996.
Enevoldsen C, Grohn YT, Thysen I (1991a), Heel Erosion and Other Interdigital Disorders in Dairy-Cows - Associations with Season, Cow Characteristics, Disease, and Production, Journal of Dairy Science 74: 1299-1309
Enevoldsen C, Grohn YT, Thysen I (1991b), Sole Ulcers in Dairy-Cattle - Associations with Season, Cow Characteristics, Disease, and Production, Journal of Dairy Science 74: 1284-1298
Lauritsen U, Lind T (2003), Ydelseskontrollen 2001-2002, Kvoginfo 1069:
Murray RD, Downham DY, Clarkson MJ, Faull WB, Hughes JW, Manson FJ, Merritt JB, Russell WB, Sutherst JE, Ward WR (1996), Epidemiology of lameness in dairy cattle: Description and analysis of foot lesions, Veterinary Record 138: 586-591
Phillips CJC, Morris ID (2001), The locomotion of dairy cows on floor surfaces with different frictional properties, Journal of Dairy Science 84: 623-628
Rodriguez-Lainz A, Melendez-Retamal P, Hird DW, Read DH, Walker RL (1999), Farm- and host-level risk factors for papillomatous digital dermatitis in Chilean dairy cattle, Preventive Veterinary Medicine 42: 87-97
Stanek C (1997), Housing and nutrition related claw diseases of dairy cattle, Israel Journal of Veterinary Medicine 52: 80-87
Vermunt JJ, Greenough PR (1994), Predisposing Factors of Laminitis in Cattle, British Veterinary Journal 150: 151-164
Wells SJ, Trent AM, Marsh WE, Robinson RA (1993), Prevalence and Severity of Lameness in Lactating Dairy-Cows in A Sample of Minnesota and Wisconsin Herds, Journal of the American Veterinary Medical Association 202: 78-82

EFFECT OF PRESENCE OF CLAW LESIONS IN HEIFERS PRIOR TO FIRST PARTURITION ON RISK OF DEVELOPING CLAW LESIONS DURING LACTATION

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INTRODUCTION

According to the 2002 National Animal Health Monitoring Systems (NAHMS) survey, 16% of cattle are culled due to lameness. However, this survey may underestimate this number as cows culled for low production (19%) or reproductive failure (27%) may actually have been lame. Lameness has been shown to reduce milk production (Guard, 1997; Robinson et al., 2003) and fertility (Sprecher et al., 1997; Hernandez et al., 2000: Melendez et al., 2002). Furthermore, dairy producers tend to underestimate extent and severity of lameness within their herd (Whay et al., 2002). Literature also indicates that previously lame cattle are more prone to future reoccurrences (Peterse, 1986; Raven, 1989; Enevoldsen et al., 1991). Therefore, preventing animals from becoming lame must be a key management objective. However, there is limited data on incidence and severity of claw lesions in calves and heifers. Data is also limited on the impact of claw lesions during the rearing phase on reoccurrences of claw lesions during lactation. The objective of this study was to determine the incidence and severity of claw lesions in heifers from 12 months of age to calving and the impact of claw lesions during the rearing phase on reoccurrence of claw lesions.

MATERIALS AND METHODS

Claws of 572 dairy heifers at a commercial heifer rearing facility were evaluated at 12 months of age, one month prepartum and two months after parturition. Heifers originated from one of four source dairies and were housed at the commercial heifer rearing facility in groups of approximately 100 animals in open, earthen-mounded lots without overhead protection. Concrete feed platforms and manure alleys integral to the open mound lots were scraped twice per week and mounds were bedded with coarse bark when needed as determined by the operator of the commercial heifer rearing facility.

During the rearing phase, heifers were fed a TMR consisting of (DM basis) 42% corn silage, 30.2% haycrop silage, 25.5% hay, 0.4% soybean silage, 0.5% corn gluten meal, 0.2% high moisture corn, 0.3% urea and 0.9% vitamins and minerals. Two dietary treatments were used: a control diet, and a treatment diet with the same ingredient composition as the control diet except for the addition of a complexed trace mineral additive. Results regarding dietary treatments will be reported in another paper at this conference. Mean chemical composition of diets is reported in Table 1.

Table 1. Average chemical composition of diets fed to heifers

Chemical component, DM basis	
Crude protein, %	15.3
Acid detergent fiber, %	29.8
Neutral detergent fiber, %	40.3
Calcium, %	1.0
Phosphorus, %	0.36
Magnesium, %	0.28

After completion of the claw evaluation at one month prepartum, heifers were returned to the source dairy. At the source dairy, heifers were housed in naturally ventilated free stall barns, fed similar total mixed rations (TMR) with respect to the source dairy and were milked thrice daily.

Claws were evaluated by one claw trimmer using a clean, light grind. The claw trimmer was a graduate of the Dairyland Hoof Care Institute (Baraboo, WI). Lesions were noted in the 7 zones of the claw (adapted from Greenough, 1997) and each lesion was scored for severity on a scale of 1 to 3 (1=minor, 2=moderate, 3=severe). To assess both incidence and severity of claw lesions, a claw lesion incidence and severity (CLIS) index was calculated. This index was the average number of zones affected per cow multiplied by the average severity score of the lesion multiplied by 10.

The CLIS index was analyzed using the MIXED procedure of SAS (1999) with the effects of dietary treatments and source dairies as discrete class variables, and CLIS index in a prior phase and its interaction with dietary treatments as continuous variables. Effects were declared significant at $P < 0.05$.

RESULTS AND DISCUSSION

The CLIS index integrates frequency and severity measurements. At 12 months of age, sole hemorrhage, white line separation, and heel erosion were the predominant disorders (Table 2).

Table 2. Least square means of claw lesion and severity (CLIS) indexa of dairy heifers at 12 months of age, one month prepartum and two months postpartum.

Claw Disorder	12 Months of Age	1 Month Prepartum	2 Months Postpartum
Dorsal wall ridges	0.4	0.0	1.2
Heel erosion	6.4	31.9z	35.0z
Abaxial wall lesions	0.0	0.2	0.0
Abaxial wall fissures	1.0	0.8	2.5z
Double soles	0.2	0.2	0.1
White line separation	8.6	2.9	6.3
Sole hemorrhages	9.7	13.2z	26.4z
Sole ulceration	0.0	0.0	0.2
Digital dermatitis	0.0	1.5	1.9z
Interdigital dermatitis	0.0	0.0	0.2
Foot rot	0.0	0.0	0.0

[a] Calculated using the following formula (number of zones affected per cow x average severity score x 10); Severity score ranged from 1 (minor) to 3 (severe)
[z] Significant effect of claw status in prior phase ($P < 0.05$)

One month prepartum, heel erosion, sole hemorrhage, and to a lesser extent white line separation were the predominant disorders. More importantly, the effect of the claw status at 12 months of age had a significant effect on the CLIS index one month prepartum for both heel erosion and sole hemorrhage, indicating that the incidence and severity of these two diseases at 12 months of age influence the incidence and severity of the same diseases near the end of the rearing period. In early lactation, heel erosion, sole hemorrhage, and to a lesser extent white line separation, abaxial wall fissure, and digital dermatitis were the predominant disorders. The CLIS status one month prepartum had a significant effect on the CLIS status two months postpartum for heel erosion, abaxial wall fissure, sole hemorrhage, and digital dermatitis.

Heifers that had at least one claw lesion at 12 months of age were 27.7 times more likely ($P < 0.05$) to have a claw lesion two months postpartum than those that did not have a claw lesion at 12 months of age (Table 3). Heifers with abaxial wall fissures and sole hemorrhages at 12 months of age were more likely (odds ratio 5.3 and 2.0, respectively, $P < 0.05$) to have these lesions present two months postpartum than those who did not have these lesions (Table 3).

Table 3. Effect of the presence of a claw lesion during the rearing phase on the risk of having a claw lesion in early lactation.

Claw Disorder	Risk of a claw lesion at 2 months postpartum when animals have a claw lesion at 12 months of age, odds ratio	Risk of a claw lesion at 2 months postpartum when animals have a claw lesion at 1 month prepartum, odds ratio
Claw lesion	27.7y	15.1y
Dorsal wall ridges	3.5	0.3
Heel erosion	1.1	1.7y
Abaxial wall fissures	5.3y	1.7
Double soles	--z	--
White line separation	1.2	1.0
Sole hemorrhages	2.0y	1.2
Sole ulcer	--	--
Digital dermatitis	--	4.0y
Interdigital dermatitis	--	--

[y] Differs from one ($P < 0.05$)
[z] Insufficient frequencies to calculate odds

Heifers that had at least one claw lesion one month prepartum were 15.1 times more likely ($P < 0.05$) to have a claw lesion two months postpartum than heifers that had no claw lesions one month prepartum. The presence of heel erosion or digital dermatitis one month prepartum increased the odds of having the same disorder two months postpartum (odd ratio 1.7 and 4.0, respectively, $P < 0.05$). Presence of lesions such as dorsal wall ridges, white line separation and double soles at either 12 months of age or one month prepartum did not affect the incidence ($P > 0.15$) of these lesions two months postpartum.

Results of this study indicate that even mild claw lesions during the rearing phase can substantially increase incidence and severity of claw lesions during lactation. This confirms previous research (Peterse, 1986; Raven, 1989; Enevoldsen et al., 1991) that cows with claws lesions are more prone to reoccurrences.

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FOOT BATHING IN THE HOOF HEALTH MANAGEMENT

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Introduction

Hoof diseases and lameness in bovines account for a big portion of the income loss due to diseases. Various management tools are available that could help reduce these losses. Hoof problems during the animal's productive life are mostly related to infectious diseases, feeding, housing including the type of floor surface and improper hoof trimming practices and combinations of these factors. Foot bathing is the most commonly used method in the attempt to prevent hairy warts and other hoof diseases. There is a lack of well documented and reported trials under farm conditions that show the effect of foot bathing when accompanied by proper hoof trimming. Additionally, the available trial data does not always address the full range of foot diseases. Concern has been expressed (4) about procedures and products used with foot baths and that any perceived benefits must be weighed against direct economic costs, potential risk to human and animal health and environmental considerations (7). Foot bathing procedure recommendations including product selection, product concentration, frequency of use, and number of cow passes before cleaning and refilling are rarely supported by documented clinical trials. The objective of this study was to evaluate the overall effect of a well maintained foot bathing solution, containing no antibiotic, heavy metals or other environmentally hazardous substances on the health and condition of the hooves.

Material and Method

The trial was performed on a dairy farm located in Mt. Vernon, WA (USA). The target population of this study consisted of 190 dairy cows, Holstein breed, that were milked twice a day in a double ten herringbone parlor. The foot bath solution used prior to the trial consisted of 25 pounds of copper sulfate diluted in 50Gal of water. The housing consisted of free stalls with kiln dried shavings as bedding, cleaned every two days and concrete floors. There were no changes in the feeding or management practices throughout the length of the trial. Weather during the trial had the average rainy days and temperature found in this region of WA in late winter and spring. A 55 gallon footbath was located in the alley at the exit of the parlor, preceded by a wash bath containing fresh water. The foot bath containing Double Action® (DeLaval) at 5% and the wash bath were emptied and recharged daily after all 190 cows passed through the bath.

Hooves were evaluated at the start and the end of the trial for incidence of hoof disease. The pre-trial protocol consisted of an examination of all the cows of the herd and trimming as needed. The cows walked through the foot bath solution once a day from Monday through Friday after the evening milking. During the weekends both baths remained clean and empty in their place. The length of the trial was 12 weeks beginning in January 2003 and finishing in April 2003. Cows were surveyed again at the end of the trial and the results recorded. Both surveys were performed by a veterinarian skilled at evaluating hoof condition with the assistance of a professional hoof trimmer. . All the cows in the herd were scored but for analysis purposes only those that stayed in the herd throughout the trial period were included in the final results. Temperature and weather data was collected during the course of the trial. Statistical analysis was done using Chi Square test with Statistica software

Results

Heel erosions, hemorrhages and digital dermatitis (DD) were the most prevalent hoof problems at the start of the trial. A total of 37.5% of hooves were determined to be unhealthy. Foot bathing with 5% Double Action for 12 weeks reduced the number of cows affected with digital dermatitis and interdigital dermatitis. A significant reduction in hooves affected by Heel Erosion, Hemorrhages and Ulcers was observed. These results are summarized in the chart below. The total prevalence of hoof problems was reduced to 17.9%. Weather conditions, temperature and rain, during the trial were average for the season.

Survey	Initial		Final		Statistics	
Disease	% of hooves	Incidents	% of hooves	Incidents	χ^2	P
Heel Erosion (HE)	16.3	85	11.5	60	5.009	0.02500
Hemorrhages (H)	10.0	52	1.3	7	36.386	0.00001
Digital Dermatitis (DD)	5.0	26	3.7	19	1.138	0.28600
Interdigital Dermatitis (IDD)	0.8	4	0.6	3	0.144	0.70400
Foot Rot (FR)	0.4	2	0.4	2	0.000	0.99999
White Line Disease (WLD)	1.3	7	0.0	0	0.704	0.00790
Ulcer (U)	3.7	19	0.4	2	14.046	0.00010
Healthy Hooves	62.5	325	82.1	427	49.96	0.00001
Total	100.0	520	100.0	520	-	-

Discussion

Previous trials (3,6) have shown that Double Action is effective at reducing the incidence of Digital Dermatitis in herds with a high incidence of DD. The reduction in the prevalence of Digital dermatitis shows that even in a herd with a low number of hooves affected (5%) there is a good chance of reducing the incidence of this disease. In the current trial, Double Action helped control a range of hoof problems when used in a well maintained foot bath. Prior to the trial, hoof care on this farm had consisted of foot bathing with copper sulfate three times a week. The improved hoof health is attributed to the specific preventative product as well as the implementation of an improved foot bathing regime.
The diseases surveyed could be divided into infectious diseases and other hoof problems.
The infectious agents responsible for Digital and Interdigital Dermatitis are also believed to play a role in the etiology of Heel Erosion (1, 5). The significant reduction in the number of hooves affected with Heel Erosion along with the reduction in the number of DD lesions would tend to support that belief.

Sub solar hemorrhaging, White line disease (WLD), and sole ulcers are primary indicators of a previous laminitis. While a nutrition factor like rumen acidosis seems to be a key in the development of laminitis, different observations suggest that additional factors must be involved (2). During this trial there was a significant reduction in the prevalence of sub solar hemorrhaging, WLD and sole ulcers. Although there was no change in the feeding routine or diet during the trial, no specific nutritional parameters were monitored and therefore we cannot determine how nutrition affected this reduction. Locally acting factors influence the hoof directly. Overgrowth of the horn is a result of environmental factors exacerbated by predisposing disease factors (2). Functional hoof trimming restores the normal shape of the claw, the angle of the toe and the equalizes weight distribution between the two claws. Claw trimming does not replace other appropriate disease control measures but it is an important component indicating that hoof trimming probably played a significant role in reducing the level of sub solar hemorrhaging, WLD and sole ulcers during this trial.

Conclusion

The results of this study show that Double Action is effective at improving overall hoof condition when used 5 days per week in a herd with a relatively high incidence of hoof disease. Additional studies are suggested to determine if this product can be used less frequently as part of a hoof health maintenance program. Specific recommendations on frequency and concentration for other hoof bath agents like copper sulfate or formaldehyde would need to be determined through clinical trials.

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References

1.Bargai, U: Digital Dermatitis, Interdigital dermatitis and Heel Erosion - Are these separate diseases?, Proceedings of the 10th International Symposium on Lameness in Ruminants, Lucerne, Switzerland, 1998
2.Greenough, PR., et al: Lameness in Cattle. 3rd (ed), WB Saunders Co., Philadelphia, PA, 1997
3.Hammock, N., Hammock farms, VA, Personal communication. 2003
4.Hoblet, K.: Foot baths: separating truth from fiction and clinical impressions, 12th International Symposium on Lameness in Ruminants, Orlando, Florida, 2002
5.Toussaint, Raven E: Cattle foot care and claw trimming, Ipswich, Farming Press, UK, 1989
6.Seymour, J, et al. Foot bathing in the management of Digital Dermatitis. Proceedings 12th International Symposium on Lameness in Ruminants. 2002
7.Zemljic, B, Digital Dermatitis: Where we are after 30 years. Proceedings 12th International Symposium on Lameness in Ruminants. 2002

THE EFFECT OF SIRE ON THE FOOT AND HOOF HORN OF THE DAIRY HEIFER

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INTRODUCTION

To test the hypothesis that the sire of a first lactation heifer has a significant effect on the development of hoof horn haemorrhages and other factors predisposing to lameness, the dataset accumulated over a period of six years from DEFRA- and MDC-funded studies conducted at ADAS Bridgets research centre was interrogated and analysed to search for such correlations.

MATERIALS AND METHODS

Design and statistical analysis
The hypothesis to be tested was that the sire of a first lac-

tation heifer has a significant effect on the development of hoof horn haemorrhages and other factors predisposing to lameness. The pertinent records for all of the heifers on previous studies of cattle lameness at ADAS Bridgets were inserted onto a spreadsheet. This contained all the relevant data from these studies, so that the effect of sire could be examined independently of study treatments. The data were investigated using preliminary descriptive statistical techniques, Pearson correlations and Principal component analysis.

Inclusion of assessments

The foot lesion assessments included were:

i. The worst lesion score for each heifer: based on severity and severity/extent

ii. The lesion score at 12 weeks post partum for all heifers: based on severity and severity/extent

White line haemorrhage scores and sole haemorrhage scores were included separately for all animals

For hoof wear, the average wear rate between 0 and 12 weeks post partum were included. For hoof growth, the table included the average growth rate between 0 and 6, 6 and 12, 0 and 12 weeks post partum

The angles of the toe at calving and 12 weeks post partum were included.

The presence or absence of slurry heel at calving and 12 weeks post partum was included.

Sires

Five parameters of sire conformation and performance were included in the analysis. These were toe angle score, locomotion score, legs and feet score, PIN and PLI. In total, 21 sires were represented in the data set of 131 cow cases. For three sires, only one heifer case was present in the data set, whereas four sires were represented by 10 or more daughters.

RESULTS

Pearson correlations for key cow and sire parameters are shown in Table 1. There were no high correlations between any of the cow and sire parameters, indicating the complete absence of any useful prognostic factors.
Subsequently, principal component analysis was done in a further attempt to identify potential correlations between cow and sire traits in these data. Pearson correlations of the first eight cow and first three sire principal components are shown in Table 2 None of these correlation coefficients had a magnitude greater than 0.290 and most were less than 0.1, and the resultant conclusion was that there was no significant correlation between sire and cow factors relating to lameness.

Table 1. Pearson Correlations between cow foot lesion assessments and sire conformation parameters

	Cow factors						
	Foot lesions				Hoof wear	Hoof Growth	Toe angle
	Slurry heal		White line				
Sire factors	Worst case	At 12 weeks PP	Worst case	At 12 weeks PP	0-12 wks PP	0-12 wks PP	At 12 weeks PP
Foot angle	-0.126	-0.063	-0.047	0.024	0.045	-0.006	0.066
Locomotion score	-0.212	-0.165	-0.136	-0.050	0.011	0.001	0.126
Legs and feet score	-0.239	-0.187	-0.151	-0.066	-0.027	-0.033	0.079
£PIN*	0.114	0.018	0.088	0.077	0.048	-0.007	0.028
£PLI**	0.127	0.038	0.072	0.028	0.034	-0.022	0.039

Table 2. Pearson Correlations between the first eight cow (PCC) and first three sire (PCS) principal components

	Cow factors							
Sire factors	PCC1	PCC2	PCC3	PCC4	PCC5	PCC6	PCC7	PCC8
PCS1	-0.042	0.067	0.079	0.185	-0.061	0.017	0.156	0.028
PCS2	-0.057	0.070	0.066	0.028	0.149	0.290	0.031	0.053
PCS3	-0.008	-0.089	-0.033	0.122	0.003	0.089	0.100	0.102

CONCLUSION

None of the sire factors investigated in this study had any significant correlation or association with the observed measures of cow lameness.

USE OF A NOVEL FOOT FOAM IN THE CONTROL OF DIGITAL DERMATITIS

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Introduction

Digital dermatitis is a bacterial infection of the epidermal skin of the bovine digit. First reported in Italy in the early 70s, the disease has now spread to most intensive livestock producing countries worldwide, and accounts for around 20% of all cases of foot lameness. This is one probable reason why, despite years of research, the incidence of bovine lameness has not decreased over the past 15 years.
Many publications have shown the value of footbaths, but the majority of products in current use have some disadvantages. Formalin is unpleasant to handle and may have carcinogenic properties. Copper salts do not

degrade in the environment and there is already concern about the increasing incidence of copper poisoning in UK dairy cattle. Antibiotic footbaths are still used in the UK and some other countries, but there are milk withhold and environmental disposal considerations. Initially antibiotic footbaths were used when there were a significant number of visible lesions present in the herd. More recently this view has been challenged (Blowey 2000) and there is now a growing trend among farmers and vets to disinfect the feet regularly in an attempt to prevent lesions from developing. Prevention of lameness is clearly better than treatment. It is known that there is a considerable increase in incidence of digital dermatitis in the 2-3 months post-calving, (Blowey and others 2004). This is thought to be partly associated with housing conditions and partly associated with immune suppression in the periparturient animal.
There is a perception among many farmers that cows do not like walking through footbaths and hence alternatives to a liquid bath have been sought. These include a foam mat, which becomes a footbath when foot pressure is applied to the mat. An alternative, described in this paper, is a novel foam that is sited at the entrance to the milking parlour. This paper describes the use of the foam and the monitoring of foot condition in one trial herd.

Materials and Method

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

The recommended dosage regime is for treatment to be carried out at each milking for the first two weeks and thereafter at six consecutive milkings every two weeks. Increased frequency of foam application may be necessary for farms where there is a heavy challenge.

The effectiveness of the foam was monitored in a 140-cow unit in the author's general veterinary practice. There had been a longstanding problem of digital dermatitis in this herd. Foot bathing had been tried but was found to be very laborious, was not practised frequently and hence was not effective. Before the foam system was instigated, a whole herd foot score was carried out. Feet were examined in a herring bone parlour while the cows were standing to be milked. The heels were cleaned using a volume water hose and then scored according to the following criteria:

	Size	Colour	Adjacent skin edge
0	0	0	0
1	0-15mm	Dark/black	Flush
2	>15mm	Dark/black	Flush
3	>15mm	Red	Raised
4	>25mm	Red	Raised

A lesion would be given a score on achieving any two or three criteria. Hence if a lesion were greater than 15mm and raised above the adjacent skin surface, it would score as a 3. A lesion that was red and scored greater than 25mm would be scored 4, whether it was a raised proliferating lesion or flush with the adjacent skin, or eroded and hence depressed below adjacent skin. Scoring was carried out prior to the instigation of the foam, then at approximately six-week intervals following the use of the foam. The foam was applied at slightly higher frequency than the manufacturers recommended rate, being used continuous for the first two weeks, then once daily for 5 days each week thereafter.

Results

Mean herd scores based on the average score per foot are given in Table 1. There was a high incidence of digital dermatitis prior to the instigation of the foam, with 33% per cent of cows showing lesions with score 2 and above. Many other cows had mild, hyperkeratinised areas of skin with a score of 1.0. The herd score decreased over the period of the application of the foam, although it was noticeable that individual animals that were badly affected, i.e. with a high score, did not improve significantly. These animals had to be restrained and individually treated. This is in the Manufacturer's instruction for the foam; i.e. the foam is a preventive, not a treatment.

Discussion

There was a reduction in the incidence of digital dermatitis in this herd in association with the use of the Kovex ™ foam. This was not, of course, a controlled trial and hence the reduction in dermatitis may have been due to environmental or other conditions which may have led to an overall reduction in dermatitis. The lesion scoring system did not allow for the scoring of interdigital lesions, nor anterior coronary band lesions. The scores may also have been influenced by the posture of the cow, in that it is less easy to visualise the interdigital cleft area of a cow if the pastern angle is shallow and fetlock is close to floor level. However, it was the subjective feeling of the author that the cows were walking better at the end of the scoring period than they had done for many years beforehand. Whilst individual animals still needed treatment, it was noticeable that heifers entering the herd remained relatively free of the disease. Other studies (Blowey et al, 2004 Maribor Conference proceedings) have previously shown that digital dermatitis is more common both in heifers and in early lactation animals and hence the finding that heifers are able to enter the herd and remain free of the disease may well be significant. The owner and herdsman felt that the system was easy to use and there was very little adverse effect or fear on the part of the cows. The system currently costs around 2140 Euros (Ł1500) to install and the chemical cost is approximately 1.42 Euros (Ł1) per cow/month., or 4.7cents (3.3p) per cow per day when used at the recommended rate of 6 consecutive milkings every two weeks.

Although the equipment is expensive to install, the reagent costs are less expensive than some of the alternative forms of footbath. For example although 150 litres of 5% formalin, enough to foot bath 150 cows and made up fresh daily, would cost 1.4 cents (2 p) per day, a proprietary glutaraldehyde/copper sulphate foot bath on sale in the UK costs 13.1 cents (9.2 p) per cow per day. Formalin is unpleasant to handle and copper salts may have adverse environmental effects The Kovex foam used in the current study was pleasant to handle and there was no evidence of any adverse effect on the cows. Many farms would pay a little extra for ease of handling, and the installation of the foam system represents an alternative method for assisting in the control of digital dermatitis.

date		% cows with score 2 and above	Herd mean score
17-07-03	pretreatment	33	1.6
20-08-03	5 weeks of treatment	19	0.94
08-10-03	12 weeks of treatment	15.5	0.75

Figure One: Digital dermatitis foot scores over time

References

Blowey RW, , Green LE, Collis VJ and Packington AJ, (2004) The Effects of Season and Stage of Lactation on Lameness in 900 Dairy Cows, Proc Int Ruminant Digit Symposium, Slovenia, 2004

Blowey RW (2000) Control of Digital Dermatitis, Vet Rec vol 146, page 10

CAN EPIDEMIOLOGY INFLUENCE OUR UNDERSTANDING OF LAMENESS IN CATTLE ?

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Part I

Where shall we start?

We want cows that are not lame. We know that lameness reduces milk yield and longevity and affects the health and welfare of cows. If it were easy to prevent lameness we would have already done so. Epidemiology is one of a multidisciplinary team of research approaches that can advance our understanding of lameness. It is potentially a powerful tool when used in combination with biology and may be of particular use to address complex situations.

We need to optimise our control of lameness to have an economically and socially sustainable system of production. To do this we need to identify factors that cause lameness using optimal research approaches that include correct study design with a defined hypothesis, defined disease outcome, correct duration for the study and time intervals between sampling events within a study, correct sample size for power and finally economic evaluation of the results. The results then need testing, first through qualitative discussions with the end users on their likely uptake and then in controlled intervention studies.

Optimal research approaches have to consider....

1. The many causes of lameness.
In a recent study when 900 cattle were studied for 18 months (Hedges, 2001), 15 causes of lameness were identified (Blowey et al., 2004). New 'causes' are reported quite frequently (e.g. heel ulcer, Blowey et al., 2000) and further study will elucidate further causes. It may be that some of these are different presentations of one aetiology. We are currently collecting data as part of an EU project (Scaif et al., 2004) and we have 16 listed causes of lameness plus an 'other' section (Amory et al., 2004). We anticipate that with results from other workpackages in the project and the risk factors that we identify that we will be able to combine some of these causes and increase our understanding of the biology of some of the pathological processes that are occurring. In turn we may then improve our understanding of the exter nal factors that precipitate disease.

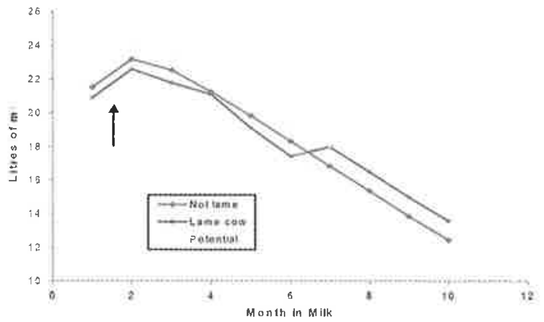
2. The diagnosis of lameness
Lameness is complex. There is no universal definition of lameness. Is it the presence of lesions visible on the outer surface of the foot? (What is the foot?). Is it an abnormal gait? What is abnormal? Is it a visible abnormality in leg movement or arching of the back or more complex than either of these? Maybe it is an abnormality detected using force plates or walking patterns? If so, what is the perfect cow's locomotion pattern, rate of foot placement and weight bearing? We need to decide what we are going to measure, why and how.

3. Some 'causes' of lameness are infectious (digital dermatitis and interdigital phlegmon) whilst others are not (sole ulcer, white line disease, 'laminitis').
The study of infectious disease requires a very different epidemiological approach in many circumstances, as we discuss below. A further complexity of all causes of lameness is that there are multifactorial risks that are associated with each cause. These include stage of lactation, age of cow, exposure to faeces, importation of new stock, type of flooring, production demands. These multifactorial risks change with time (season and time from calving), environment and management. These are matters that complicate the recording and interpretation of data during analysis.

How can we increase our understanding of a process?

1. A series of study types
We can further our understanding of any one factor through a series of study types (Martin et al., 1987). In the study of biotin (Hedges et al., 2001; Green et al., 2002; Potzsch et al., 2003) we provided strong evidence that supplementation with 20mg biotin reduced white line disease lameness by up to 50%. The strength of this association is biologically important and biologically plausible. There was previous experimental work (Mulling et al, 1999; Abel et al., 2001) that supported the study results and previous field work on lameness (Fitzgerald et al., 2000) and lesions without lameness (Zimerly and Weiss, 1997); although some apparent differences in lameness versus lesions may remain for heifer / parity 1 cattle (Potzsch et al, 2003 c.f. Zimerly and Weiss, 1997). Further research is being done to investigate the biomechanics of white line disease e.g. Hedges et al., 2002. Our study was done on five farms in Gloucestershire. We did not identify an interaction between biotin and farm, so farms did not behave differently in their response to biotin supplementation but it is not possible, with only five farms

studied, to conclude that all farms with white line disease would benefit to the same degree from biotin supplementation. A further study - or empirical use of biotin, is required to establish this.
We need to use this approach to understand many of the risk factors associated with lameness.



2. We can answer complex problems

Figure 1. Theoretical representation of loss of milk yield in lame cows.

Some of the most exciting advances occur when we conceptualise a process. One example of this was in the study of the impact of lameness on milk yield (Green et al., 2002) when it was necessary to create a variable to define whether a cow was lame in the lactation at all. This was done because there was evidence from previous research (Barkema et al., 1994), simulation models from the 1980s (Lucy et al., 1986) and genetic evidence that lame cows were usually high producers. The variable created captured that information and was used to describe the pattern of lost milk production around the lameness event.

One other example, which to our knowledge has not been used in lameness research, is that we can use epidemiology to create uncertainty (error) in our diagnostic ability and test the impact of this in our studies. The variability that we know exists between farms means that it is unlikely that factors that are successful at preventing lameness on one farm will work for all other farms. So we have to allow for variability, or stochasticity, in our studies and assess the impact of our recommendations on a farm-by-farm basis.

3. Conceptual epidemiology
Ironically, although epidemiology is often confused with statistics, it is when we put together all the evidence from ecology, pathology, histopathology, molecular biology, microbiology, experimental studies, intervention studies and create a new hypothesis that the most challenging and exciting advances may occur. For example, recent work on footrot in sheep (Wassink et al., 2003) has indicated that we may need a very different control programme in the UK than has been proposed to date which is based on work from Australia (see Green et al., 2004). This is a hypothesis but if correct would change our management of this disease radically.

4. Simulation models ~ chickens and eggs, which came first? (does it help to know?)
When thinking of infectious disease the occurrence of disease is dependent on the amount of pathogen present. The situation is believed to be circular: the more pathogens present the more severe the disease is likely to be. Interestingly, we may also identify steady states e.g. some cows that never become lame despite large amounts of pathogen present. These non-lame cows may even be the high yielding cows that were intuitively believed to be more likely to become lame. As a consequence, we need to consider these groups separately using a different, often interdisciplinary, approach.

Another circular situation is the occurrence of foot lesions in clinically sound cattle and in clinically lame cattle. When are foot lesions important? The controversy about lesions versus lameness is a good example of the difficulties in interpreting lameness data without having any or sufficient records about underlying foot lesions. There is no strong evidence for using presence of lesions as a proxy for lameness in all circumstances. We need studies that put the presence of lesions and the occurrence of lameness in a temporal setting.

This opens the discussion about self-organising systems without leaders e.g. lesions and lameness, pathogen load and disease, and complex dynamics that occur in lameness and foot lesions in cattle. An attempt should be made to describe the consequences of the complexity of lameness for herd health management aimed at reducing lameness and foot lesions.

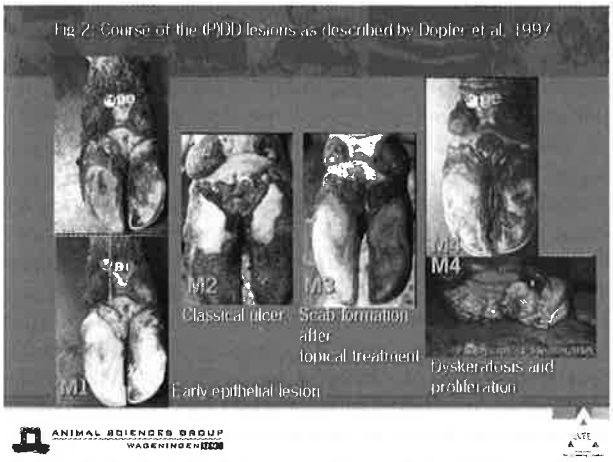
Part II

Host-pathogen interactions are self-organisational systems without leaders
Self-organizational systems are characterized by complex interactions without a leader where local information leads to global pattern formation (Carmazine 2001). The local information can be very simple (lesion/no lesion) and still generate patterns that are not understandable based on local information alone.

What does this have to do with lameness and foot lesions? Lameness or foot lesions could be such local information and the occurrence of outbreaks of foot lesions and lameness could be considered as global patterns. Host-pathogen interactions lead to states of infection or disease patterns that can be described on the cellular, individual, group and population level. Self-organising systems can be described on different levels and be intercalated, generating hierarchies of such systems. Furthermore, population dynamics of infection are the result of a hierarchy of complex interactions on different levels and may not be comprehensible by intuitive and instant evaluation alone. Despite this, herd health managers are routinely asked to intuitively understand complex animals husbandry systems and we wonder why they fail to eliminate lameness and foot lesions. One reason may be because of the complex nature of the systems they deal with. Conceptualising this process in an interdisciplinary approach on the cellular, individual and group level may aid in developing new preventive and therapeutic intervention strategies. The conceptualisation will result in the idea that there is variability, or stochasticity, demanding the assessment of the impact of interventions directed at reducing lameness and foot lesions on a farm by farm basis as explained above.

Most epidemiological studies about lameness and foot lesions have been designed as a knock-out approach. The knock-out approach wants to eliminate lameness and foot lesions by eliminating pathogens and risk factors. It is evident that elimination of lameness and foot lesions has failed until today; maybe because of a lack of awareness of ideas about complex interactions.

Complex interactions without leaders have certain features that need not be present simultaneously. Among those features are: feedback loops, threshold phenomena, multiple steady states or equilibria and transitions of the system between those equilibria. It was explained above that the "true or real risks" for lameness and foot lesions are dependent on the time frame for measurements, the statistical power of the study and biologically sound hypotheses for the interaction of host and pathogen or noxae leading to lameness and foot lesions. This multi-causality will be reduced to the interaction of host and pathogen in the following simulation model.



Using a simulation program called 'Starlogo' and a code originally developed for the aggregation of slime molds (http://www.media.mit.edu/Starlogo), the complex interactions of bovine host epidermis and imaginary proteolytic microbes during the pathogenesis of papillomatous digital dermatitis ((P)DD) will be described. The imaginary microbes shed a substance that attracts other microbes and destroys the epidermis at a rate dependent on the concentration of microbe and their duration of impact. The simulation yields examples for complex interactions without leaders and displays feedback loops, threshold phenomena, multiple steady states or equilibria and transitions of the system between those equilibria.

Figure 2 shows the course of disease for (P)DD as described by Döpfer et al., (1997). The abbreviations M1 to M4 will be used in the descriptions following this figure. Figure 3,4,5,6 illustrate examples for (P)DD lesions emerging from a simulation model using Starlogo.

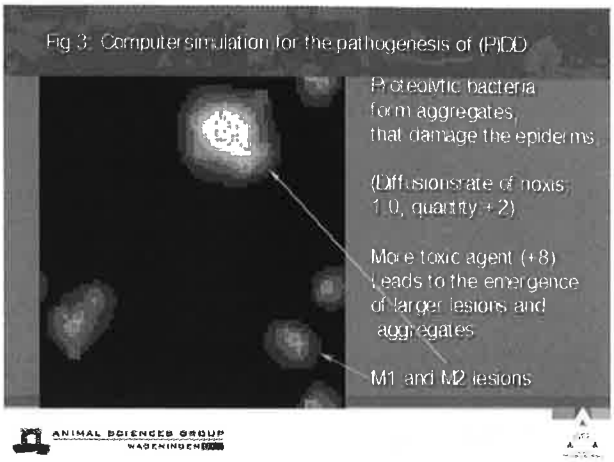
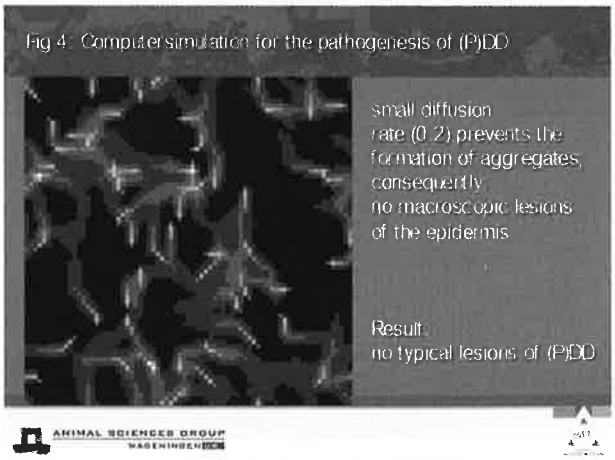


Figure 3 shows the outcome of a simulation for the interaction of host and imaginary pathogens where the combination of diffusion rate for the noxious agent together with the quantity of agent shed leads to the formation of stable aggregates that can be interpreted as the classical ulcerative lesions of (P)DD, abbreviated as M2, and the small epithelial damages, M1, as described by Döpfer et al. (1997). The M1 lesions are similar to the focal bacterial keratolysis (FBK) as described by Read et al., (1987). Aggregates develop due to feedback loops between pathogens on the host. Meanwhile, M1 and M2 lesions are interpreted as different equilibria or two 'attractors' of this dynamic system. Transition between these equilibria is possible as observed during the 'real' course of disease on the cow and during the simulation.



In Figure 4, the diffusion rate of the noxious agent was slightly diminished and no such aggregates are formed despite of the fact that the same amount of imaginary pathogens and quantity of noxious agent are present. This is an example of a transition of the system to a state where no equilibria such as M1 or M2 lesions can be found. The reason for this difference in diffusion rate could be a 'hardened' or less permeable imaginary epithelium of the host (imagine a really effective footbath applied to this cow's foot that has led to the hardening of the epithelium!!!) or the less efficient diffusive character of the noxious agent (see foot pathogens with low proteolytic index as described below).

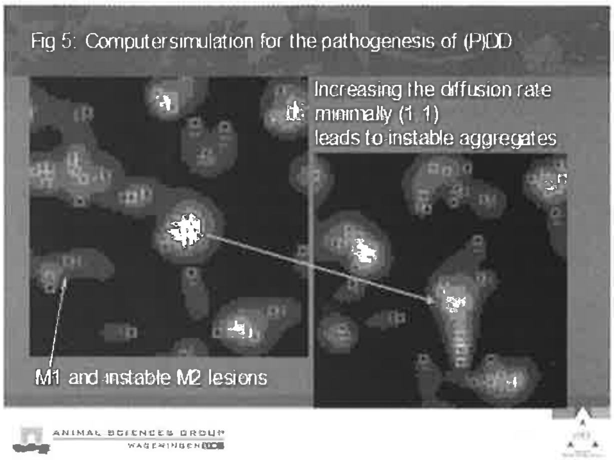
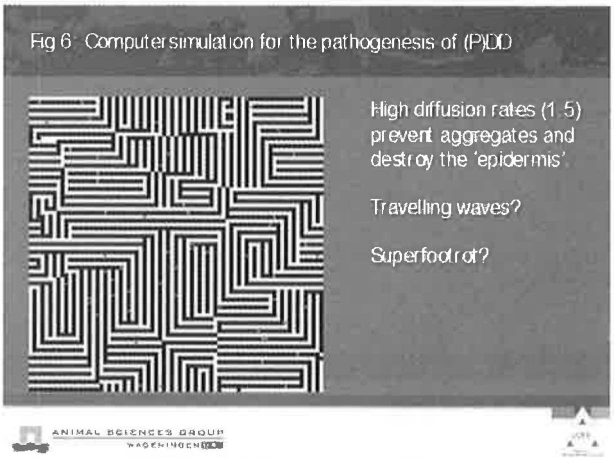


Figure 5 illustrates the situation where a slight increase in the diffusion rate of the noxious agent will result in unstable aggregates and temporary appearance of M2 lesions. This happens in the presence of the smaller M1 or focal bacterial keratolytic lesions. This simulation outcome is counter-intuitive, because we would have expected that increasing the diffusion rate would make the formation of aggregates more efficient. An imaginary treatment of this unstable lesion will lead to a clinical cure, but the aggregates are prone to recurrence. Periodic recurrence of (P)DD lesions after topical or systemic treatment is one of the big frustrations in preventing and treating (P)DD in larger groups of cattle. Increasing the amount of noxious agent while keeping the diffusion rate at the same level will result in larger M2 lesions (Figure not shown).



Increasing the diffusion rate any further will result in the imaginary destruction of this simulated epithelium without aggregates of pathogens nor visible lesions. This could be interpreted as the rarely described lesions of 'superfootrot' (Berger, Blowey personal communication) or very severe cases of (P)DD that lead to the destruction of the animals, because their digital skin seems to be literally eaten away.

This is to say that the classical ulcerations of (P)DD or M2 lesions may be described as a finely tuned equilibrium of host and pathogen that is not eliminated by temporarily applying local antibiotic or other disinfecting agents.

What is the equivalent of the imaginary pathogens in 'real' (P)DD or other diseases of the ruminant digital skin? Spirochetes of the Treponema spp. are candidates for the aetiological agents in the pathogenesis of (P)DD and they are known to produce proteases that harm the host's epithelium and form aggregates on and in their host's digital skin (Walker et al . 1998). Keratinocytes of ruminant digital epithelium on the other hand are known to be inflammatory modulators by producing messengers of inflammation, such as Interleukin 1 and Tumour necrosis factor ? (Jubb et al., 1993). It is postulated that the interaction of host keratinocytes and bacteria results in an inflammatory reaction that can lead to ulceration (M2 lesions), dyskeratosis and proliferation of the digital skin (M4 lesions, see Figure 2). There are more examples for the complex interaction of host and pathogens, such as in interdigital dermatitis in cattle or footrot in sheep.

During the pathogenesis of interdigital dermatitis, Dichelobacter nodosus and Fusobacterium necrophorum are believed to act in synergy including feedback loops. The normal ovine and bovine digital skin harbour D. nodosus with a low proteolytic index of serine and non-serine proteases facilitating penetration of micro-organisms. Hydropic maceration will facilitate the penetration of F. necrophorum and D. nodosus that produces elastases able to harm the host's epithelium and cause an inflammatory reaction. In addition, its exotoxin is destructive to leucocytes. The synergistic action of proteases, enhancing penetration and elastases causing inflammation and skin damage result in what is called 'interdigital dermatitis' (Jubb et al. 1993).

The current model is a simulation model, but mathematical algorithms (deterministic or stochastic) could be readily built to describe the structures of complex interactions that lead to such equilibria. Future intervention strategies should be aimed at modulating complex systems in order to cause transitions towards a manageable steady state, that is likely to be 'the endemic state' for (P)DD. This endemic or manageable state could be characterized by decreased incidence of lameness caused for example by classical (P)DD lesions that can be treated individually while avoiding antibiotic footbaths. It is not to be understood as a conventional 'knock-out' strategy that is currently applied. The same principle is proposed for the control of foot rot in sheep (Green et al., 2000).

Such modulating interventions should be routinely optimised using cost-benefit analysis. The modulation of equilibria originating from complex systems requires optimised insight into the dynamics of the system and may seem far-fetched to the current understanding of animal health management. The current animal health management has not been able to eliminate lameness and foot lesions though and a different concept for ruminant foot health should be explored.

Animal health managers are asked to influence such complex systems by management, treatment and prevention of disease. They are expected to confine those systems to states defined by 'animal health and welfare' under economic constraints (the 'sustainability' mentioned above). Under modern circumstances of intensive animal husbandry, these states of 'animal health and welfare' may be far away from equilibrium. Why is this a problem for the animal health manager?

The human brain has no intuitive insight into consequences of complex interactions, like periodicity, equilibria, transitions, threshold phenomena and chaotic behaviour. This is due to human cognition being conditioned by evolution and results in the 'paradox of modern animal husbandry'. During evolution of the human mind, survival did not require understanding of complex behaviours, because it evolved in partially predictable ecosystems at steady state (Vollmer 1986). Intensive animal husbandry systems are not balanced ecosystems and may develop dynamics that may not be predicted by intuition alone (Edelstein-Keshet 1988). This paradox of modern animal husbandry should lead to a change of consciousness for people engaged in herd health and production medicine who still aspire intuitive insight or immediate insight after "measuring" health or disease.

These complex dynamics ask for further visualization and illustration.

One possibility for the visualization of complex interactions is the modelling of such dynamics using methods developed in disciplines other than veterinary medicine and animals husbandry (Anderson and May 1991, Yodzis 1989). The development of mathematical methods for the analysis of complex data sets may help in structurising a complex process, such as lameness and foot lesions in ruminants on a cellular, individual and population level, but may be applied to other production disease such as mastitis or fertility problems. Applied mathematics may provide structures, which when interpreted in terms of real entities may result in a cognitive aid, that is a structurising algorithm, able to disentangle paradoxical situations and help to develop management strategies for animal husbandry.

It is therefore rewarding to apply analytic methods developed in other disciplines, such as mathematics, physics, chemistry, biology, engineering, sociology and economics and add them to the training of animal health managers.

Conclusions

Yes! Epidemiology can assist in our understanding of the complex process of lameness in dairy cows. Using a variety of epidemiological approaches and knowledge from many other research disciplines it provides the opportunity to consider large contrasts between systems high in a hierarchy of groupings e.g. between cattle keeping in a developed country and a developing country down to small differences between individual farms and cows where sophisticated non-linear or simulation techniques may be required to represent a situation.

References:

Abel H. J., I. Immig, C. da Costa Gomez, and W. Steinberg. 2001. Effect of increasing dietary concentrate levels on microbial biotin metabolism in the artificial rumen simulation system (RUSITEC). Arch. Anim. Nutr. 55:371-376.

Amory J. R., Z.E. Barker, N.R. Brassey, R.W. Blowey and L.E. Green (2004). A postal survey of the incidence of lameness and claw lesions in dairy cattle in the uk: a preliminary report 13th International ruminant lameness conference. Maribor.

Anderson, R.M. and R.M. May. 1991. Infectious diseases of humans - dynamics and control. Oxford University Press, Oxford, New York, Tokyo.

Barkema, H.W., Westrik, J.D., van Keulen, K.A.S., Schukken, Y.H. and Brand, A. (1994). The effects of lameness on reproductive performance, milk production and culling in Dutch Dairy Farms. Prev. Vet. Med. 20, 249-259

Blowey R. W., Ossent P., Watson C. L., Hedges V. J., Green L.E. and Packington A. J. (2000) A possible distinction between sole ulcers and heel ulcers as a cause of bovine lameness. Vet Rec. 147 110-112

Blowey R. W., Green L. E., Collis V. J., Packington A. J. (2004). The effects of season and stage of lactation on lameness in 900 dairy cows. 13th International ruminant lameness conference. Maribor.

Carmazine, S., J.L. Deneubourg, N.R. Franks, R. Sneyd, G. Theraulaz, and E. Bonabeau 2001. Self-organisation in biological systems. Princeton University Press. Princeton/NJ/USA.

Collis V.J. (nee Hedges), L.E. Green*, R.W. Blowey†, A.J. Packington‡, R.H.C. Bonser§** Testing white line strength in the dairy cow submitted, journal of dairy science.

Döpfer D., Koopmans A., Meijer F.A., Szakall I., Schukken Y.H., Klee W., Bosma R.B., Cornelisse J.L., van Asten A.J.A.M., ter Huurne A.A. 1997. Histological and bacteriological evaluation of digital dermatitis in cattle, with special reference to spirochaetes and Campylobacter faecalis. Vet Rec. 140(24):620-3.

Edelstein-Keshet, L. 1988. Mathematical models in biology. 1st edition, McGraw Hill, Boston/MA/USA.

Fitzgerald, T., B. W. Norton, R. Elliot, H. Podlich, and O. L. Svendsen. 2000. The influence of long-term supplementation with biotin on the prevention of lameness in pasture fed dairy cows. J. Dairy Sci. 83:338-344.

Green L. E., V.J. Hedges, Y. H. Schukken, R. W. Blowey, and A. J. Packington. 2002. The impact of clinical lameness on the milk yield of dairy cows. J. Dairy Sci. 85: 2250-2256.

Green L. E., Wassink G. J., Grogono Thomas R., Moore L.J. and Medley, G. F. (2004) Footrot in sheep: control versus elimination (2004). 13th International ruminant lameness conference. Maribor.

Hedges, V., 2001. Epidemiological and biomechanical studies into the role of biotin supplementation on lameness in dairy cows. Dissertation, University of Bristol, UK.

Hedges, V.J., Blowey, R.W., Packington, A.J.,

O'Callaghan, C.J. and Green, L.E. (2001). A longitudinal field trial of the effect of biotin on lameness in dairy cows. J. Dairy Sci. 84, 1969-1975
Jubb, K.V.F., P.C. Kennedy, and N. Palmer. 1993. Pathology of domestic animals. 4th edition. Academic Press, Harcourt Brace Javanovich, p. 531, p. 657-659.
Lucey, S., Rowlands, G.J. and Russell, A.M. (1986). Short-term associations between disease and milk yield of dairy cows. J. Dairy Res. 53, 7-15
Martin, S.W., Meek, A.H. and Willeberg, P. (1987). Veterinary Epidemiology. Edition 1, Iowa State University Press, Ames, Iowa, USA.
Midla, L. T., K. H. Hoblet, W. P. Weiss, and M. L. Moeschberger. 1998. Supplemental dietary biotin for prevention of lesions associated with aseptic subclinical laminitis (pododermatitis aseptica diffusa) in primiparous cows. Am. J. Vet. Res. 59 (Suppl. 6):733-738.
Mülling, Ch. K. W., H. H. Braguella, S. Reese, K. D. Budras, and W. Steinberg. 1999. How structures in bovine hoof epidermis are influenced by nutritional factors. Anat. Histol. Embryol. 28:103-108.
Pötzsch, C. J. V. J. Collis (nee Hedges), R. W. Blowey, A. J. Packington, L. E. Green (2003) The Impact of Parity and Duration of Biotin Supplementation on White Line Disease Lameness in Dairy Cattle. Journal of Dairy Science. 86 2577 - 2582
Read, D. and R.L. Walker. 1998. Papillomatous digital dermatitis (footwarts) in Califormia dairy cattle: clinical and pathological findings. J Vet Diagn Invest 10:67-76.
Scaife J.R., Ch. Bergsten, L. Green, Ch. Mülling, R. Pijl, Ch. Stanek, K. Urbaniak (2004) LAMECOW 2002-2006. A multidisciplinary approach to the reduction in lameness and improvement in dairy cow welfare in the European community. 13th International ruminant lameness conference. Maribor.
Vollmer, G. 1986. Was können wir wissen? Die Erkenntnis der Natur, vol. 2, Hirzel Verlag Stuttgart, Stuttgart/Germany.
Walker R.L., Read D.H., Loretz K.J., Nordhausen R.W. 1995. Spirochetes isolated from dairy cattle with papillomatous digital dermatitis and interdigital dermatitis. Vet Microbiol. 1995 Dec;47(3-4):343-55.
Wassink G. J., R. Grogono-Thomas, L. J. Moore, and Green L. E. (2003). Risk factors associated with foot rot in sheep The Veterinary Record 152 351 - 358
Yodzis, P. 1989. Introduction to theoretical ecology. Harper & Row Publishers Inc., New York/NY/USA.
Zimmerly C. A. and W. P Weiss. 2001. Effects of supplemental dietary biotin on performance of Holstein cows during early lactation. J. Dairy Sci. 84:498-506.

THE EFFECT OF LAMENESS ON FEED INTAKE, FEEDING BEHAVIOUR, LIVEWEIGHT CHANGE, MILK YIELD AND MILK LET DOWN AND MILKING DURATION OF HOLSTEIN FRIESIAN DAIRY CATTLE

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Abstract

This study assessed the effect of lameness on yield, milking duration, body condition and live weight. Over a 3 year period a total of 160 multiparous Holstein Friesian dairy cows were selected at random and paired retrospectively according to calving date, milk yield, body condition and live weight. Cows were allocated at 110 days postpartum into two groups, 80 lame (L) and 80 non lame (NL) cows according to the locomotion score (<3 = Non lame, 4 and above = Lame). Data was found to be normally distributed and analysed using ANOVA General linear model. Lame cows had a significantly greater (P<0.001) locomotion score compared with non-lame cows, NL 2.01, L4.47 (sem 0.445). Lame cows had a significantly (P<0.05) lower body condition score (scale 1 to 5), NL 3.24, L 2.87 (sem 0.064), a significantly (P<0.05) lower mean milk yield, NL 42.3, L 38.7 (sem 0.60) and significantly (P<0.05) lower milking duration (mins), NL 7.03, L 6.06, (sem 0.224) compared with non-lame cows. While, there was no significant difference was found between live weight per se, NL 654, L 646, (sem 5.5) the change in live weight was significantly different (P<0.05) with lame cows losing weight compared with non lame cows which gained weight. Lame cows had lower dry matter intakes (L 23.01, NL 25.5 kg/h/d DM), had fewer meal bouts (L 2.2, NL 4.1/d). In conclusion, lameness reduced feed intake, feeding bouts, milk yield, body condition score, milking duration and increased live weight loss. A model of the effect of locomotion score was created.

ASSESSMENT OF PREVALENCE, TREATMENT AND CONTROL OF LAMENESS-RELATED DISEASE IN DAIRY HEIFERS ON 30 FARMS IN SOUTHWEST BRITAIN

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Introduction

The prevalence of lameness in dairy cows in the UK remained high through the 1990s with estimates of 21% in 1989-91 (Clarkson and others 1996) and 22% in 2001 (Whay and others 2002). The Farm Animal Welfare Council report (1997) highlighted lameness as a major welfare problem. This study is being carried out to establish the level of improvement in lameness prevalence in primiparous dairy heifers that can be achieved through the implementation of lameness control plans tailored to the individual farm. The complete study will involve 60 herds, 30 calving during the winter housing period and 30 calving at pasture in spring and summer. This preliminary report presents results from the winter-housing group.

Materials and Methods

Sixty farms located in Southwest England, the Midlands and South Wales were nominated to take part in the study by their local veterinary practitioners. The main criterion for nomination was that either the local veterinary surgeon or the farmer had identified lameness as a problem. Of these 60 farms, 30 farms were visited through the winter housing period to assess lameness prevalence and the prevalence of lameness-related disease in the dairy heifers calved in winter. Potential lameness risk factors were identified by interview and direct observation.

Lesion scoring was carried out according to the techniques described by Leach and others (1998), using modified footmaps based on maps described by Greenough and Vermunt (1991). Only heifers that were 60-120 days in milk (DIM) were examined, as this stage of lactation has been shown to coincide with the peak prevalence of claw horn lesions in first calved heifers (Anon 2001).

Locomotion scores were recorded from heifers whose claws were examined and from additional primiparous heifers that were not lesion scored, so that up to a total of 20 heifers were examined. Heifers were locomotion scored using a 6-point system developed specifically for the project, derived from the system described by Whay and others (1997). The Manson and Leaver (1988) 9-point scoring system was considered impractical for the purpose of this study, as it required observation of animals rising from the lying position. The locomotion scoring system used in this project is summarised below (table 1).

Table 1 Locomotion-scoring system

Score	Score descriptors	Comments
0	Sound	No abnormality of gait detected
1	Limb abduction or adduction, but not lame or tender	Swinging limb gait with foot-fall outside or inside the line of fore-foot placement
2	Tender	Persistently shortened strides
3	Mild lameness	Limp barely detectable and requiring careful examination to identify affected limb
4	Lame	Lameness easily detected and affected limb easily identified
5	Severe lameness	Pronounced limp. Posture and motion affected

Results

In total 523 heifers were locomotion scored on a 0-5 scale. Of these, 282 heifers in early to mid lactation also had their feet examined. The overall prevalence of lameness (locomotion score 3 or higher) in the lactating heifers was 33.5% (n=175).

Only two farms had no evidence of digital dermatitis lesions and only one farmer had never observed digital dermatitis lesions in his herd. On the affected farms 40.6% (n=114) of heifers examined had digital dermatitis in the hind feet. Other lesions recorded in hind feet included: claw horn haemorrhage (97% heifers), white line disease (27% heifers), sole ulcers (7% heifers), white line abscess (0.4% heifers) and foul-in-the foot (0.4% heifers).

The observed treatment and control strategies varied considerably between farms. Twenty-five farms were actively foot bathing. The bathing agents used included antibiotics alone (9 farms), formalin alone (6 farms), or both antibiotics and non-antibiotic chemicals in rotation (10 farms). Twenty-nine farms had someone on farm able to inspect claws and treat lameness; only one farm relied entirely on the vet or claw trimmer for treatment of lame cows. Thirteen farms employed a claw trimmer periodically or intermittently.

Discussion

Primiparous heifers were the subjects of this study as they are the most sensitive group for measuring the acute affects of the herd environment and management, as it can be assumed that the prevalence of claw horn lesions is very low before joining the milking herd. Primiparous heifers are also thought to be more susceptible to claw disease than cows as heifers have a less well-developed digital cushion (Raeber and others 2002) and undergo the largest deterioration in claw health during their first lactation (Offer and others 2000). A lameness prevalence of 33.5% (compared with an estimated national lameness prevalence of 22% using a similar scoring system) confirmed the potential for improvements through the implementation of control measures specific to this age group. This is of particular importance given that heifers that are lame in their first lactation have been found to be twice as likely to be lame in a subsequent lactation as a clinically sound counterpart (Hirst and others 2002). Thus any lameness prevention in the first lactation could potentially prevent lameness in subsequent lactations. Furthermore, this finding confirmed that monitoring lameness in primiparous heifers is probably the most appropriate and immediate means of assessing the success or failure of any implemented control measures.

Nearly all the heifers in this study had claw horn or digital skin lesions in one or more foot and the majority of heifers examined had multiple lesions. The lameness treatment and control strategies on the 30 farms were variable and, in many cases appeared to be implemented too late or ineffectively. The next phase of this study will investigate the impact of lameness control plans targeted towards risks identified at the individual farm level.

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References:

ANON (2001) Strategies for reducing lameness in dairy cattle Phase 1:modelling risk factors for dairy cow lameness Vol.1 MDC, SERAD and MAFF end of project report

FARM ANIMAL WELFARE COUNCIL (1997) Report on the welfare of dairy cattle. London, FAWC PB 3426

CLARKSON, M.J., DOWNHAM, D.Y., FAULL, W.B., HUGHES, J.W., MANSON, F.J., MERRITT, J.B., MURRAY, R.D., RUSSELL, W.B., SUTHERST, J.E. & WARD, W.R. (1996) Incidence and prevalence of lameness in dairy cattle. Veterinary Record 138, 563-567.

GREENOUGH, P.R. & VERMUNT, J.J. (1991) Evaluation of subclinical laminitis in a dairy-herd and observations on associated nutritional and management factors. Veterinary Record 128, 11-17.

HIRST, W., MURRAY, R., WARD, W. & FRENCH, N. (2002) Generalised additive models and hierarchical logistic regression of lameness in dairy cows. Prev Vet Med 55, 37.

LEACH, K.A., LOGUE, D.N., RANDALL, J.M. & KEMPSON, S.A. (1998) Claw lesions in dairy cattle: Methods for assessment of sole and white line lesions. Veterinary Journal 155, 91-102.

MANSON, F.J. & LEAVER, J.D. (1988) The influence of concentrate amount on locomotion and clinical lameness in dairy-cattle. Animal Production 47, 185-190.

OFFER, J.E., MCNULTY, D. & LOGUE, D.N. (2000) Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. Veterinary Record 147, 105-109.

RAEBER, M., SCHEEDER, M.R.L., GEYER, H., LISCHER, C.J. & OSSENT, P. (2002) The influence of load and age on the fat content and the fatty acid profile of the bovine digital cushion. In: Proceedings of the 12th International Symposium on Lameness in Ruminants, Florida, Jan 9-13, 194-198.

WHAY, H.R., MAIN, D.C.J., GREEN, L.E. & WEBSTER, A.J.F. (2002) Farmer perception of lameness prevalence. In: Proceedings of the 12th International Symposium on Lameness in Ruminants, Florida, Jan 9-13, 355-358.

WHAY, H.R., WATERMAN, A.E. & WEBSTER, A.J.F. (1997) Associations between locomotion, claw lesions and nociceptive threshold in dairy heifers during the peri-partum period. Veterinary Journal 154, 155-161.

PRELIMINARY RESULTS OF PREVALENCE'S OF AND CORRELATION'S BETWEEN MAJOR REAR CLAW DISORDERS IN 348 DUTCH DAIRY HERDS

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Abstract

To improve claw health in dairy cattle the Dutch Animal Health Service has conducted a cross-sectional study to estimate the prevalence of claw-disorders and correlations between different claw disorders.
After a training to achieve uniform assessment of diagnoses, 20 professional hoof trimmers registered their findings of 21,478 animals on 348 herds on the presence (yes/no) of major hind claw disorders. During an one-year period (May 2002-July 2003) each hoof trimmer visited on average 17 dairy herds (SD: 13.5; Median:15 ; min 1 herd; max 53 herds). From the Dutch Cattle Syndicate (NRS) information was provided on age, breed, last calving date and parity of each registered animal.
The prevalence's of 7 claw disorders were calculated on herd and cow level. In addition, relations between claw disorders were estimated.
Of all recorded animals (21,359), 69% had at least one claw disorders on the rear claws. Of all animals 13.8%, 3 or more disorders were present. Most prevalent claw disorders measured on cow level were sole haemorrhages (38%), digital dermatitis (22%) and interdigital dermatitis (38%). These disorders were present in both hind claws in 70%, 31% and 68% respectively. Strong correlations at herd level were estimated between digital dermatitis and hyperlasie of the interdigital skin, interdigital dermatitis and sole haemorrhage andsole haemorrhages and white line disease. Significant correlations between sole haemorrhage and sole ulcer and digital and interdigital dermatitis were found at cow level.

Introduction

Sound feet and legs are of paramount importance to the cow for optimal productivity, health and animal welfare (Brand et al., 1996). The origin of most lameness problems is related to claw disorders (e.g. Weaver, 2000). Claw disorders can be distinguished at clinical level (i.e., being lame) and at subclinical level (i.e., digital disorders recognizable at hoof trimming).
Monitoring soundness of the feet can be based on scoring the cows locomotion (e.g. Sprecher et al., 1997), clinical lameness, or gathering information at claw trimming (e.g. Manske et al., 2002).
Registration of these findings are important as a tool for herd health management, in order to define a health problem), to direct management decisions and to evaluate the influence and approach of these decisions (e.g. Bargai, 2000). In addition, these investigations might also play a role in evaluating genetic influence of bulls (e.g. Groen et al., 1994).
The objective of this study was to estimate prevalence's and correlations between different claw disorders as part of an overall aim to improve claw health of dairy cattle in the Netherlands.

Material and methods

Data and case definition
Between May 2002 and July 2003, information about the presence or absence of claw disorders (digital dermatitis (DD), interdigital dermatitis (ID), sole haemorrhages (SH), sole ulcer (SU), chronic laminitis (CL), white line lesions (WLD), interdigital hyperplasia (HYP) and interdigital phlegmon (IP)) of rear claws of 21,478 dairy cows on 348 dairy herds were collected. This information was collected by twenty professional hoof trimmers at the time of routine trimming all eligible cows (adult and sometimes heifers at the end of their pregnancy). These trimmers were working in the Northern and Eastern parts of the Netherlands. Each trimmer recorded on average 17 herds (SD: 13.5; Median:15 ; min: 1herd; max: 53 herds) and trimmed on average 1263 animals (Median: 1048; min: 233; max: 3069). These trimmers were trained in diagnosing the most common claw disorders by studying photographs and by discussing claw diagnosis with the first author. In addition, diagnoses were compared regularly on location during hoof trimming as to guarantee the information standards in this study. Disorders were recorded as present or absent based on specific hoof lesions.
A cow was diagnosed as positive for a disorder if the disorder was found in at least one hind claw.

Selection of herds

In all included herds, cattle were on maintenance claw trimming schedules before the study. At the beginning of a routine herd hoof trimming, dairy farmers were approached by their own hoof trimmer if they were willing to participate in the project. The cooperation included an agreement in recording the main figures of the Identification and Registration (I&R) numbers, presence of all hind claw disorders and conducting a questionnaire on some housing and management aspects carried out by the hoof trimmer.

Participating animals, breed and parity.
Information on breed, parity and calving date was collected by the Royal Dutch Dairy Syndicate (NRS) by combining I&R-information with information of the Dutch herd book milk recording system (Table 1).

Statistical analysis

Prevalence's of and correlations between claw disorders were calculated using the statistical software package SAS 8.e (SAS Institute Inc., 2000). For the correlation at herd level the pearson correlation coefficient was calculated, for the correlation at cow level the spearman rho coefficient was calculated.

Results

Due to incompleteness of records information of 21,330 animals on 344 herds were used for the analysis. The average herd size of the included herds was 62.0 cows (median 60; SD, 23.1; min: 14, max: 207). Table 1shows the distribution of the cows for parity, breed and stage of lactation in numbers and percentages.

Table 1. Distribution of the cows for parity, breed and stage of lactation in numbers and percentages.

	Number of animals	Percentage	
Parity	Missing 942		
1	6326	30.8	
2	5094		24.8
3	3676		17.9
4	2403	11.7	
5 or greater	3040	14.8	
Breed	Missing 1065		
75 < HF ≤ 100 %	15816	77.5	
50 < HF ≤ 75 %	2833	13.9	
25 < HF ≤ 50 %	731	3.6	
MRIJ	302	1.5	
Other	731	3.6	
Days in lactation	Missing 1852		
< 30 days	1452	7.4	
30 - 60 days	1707	8.7	
60 - 90 days	1668	8.5	
90 - 120 days	1570	8.0	
120 - 180 days	2826	14.4	
180 - 360 days	7144	36.4	
> 360 days	1433	7.3	
Dry period	1826	9.3	

Holstein-Friesian (HF) cows were predominant (95%), reflecting the current proportion of that breed in the present cow population. Beside HF-cows other breeds that were found were Meuse Rhine and Yssel cows (Dutch red breed) (1.5%), Dutch Friesians (3.6%), Jerseys and Mont Beliardes (3.6%). Claw trimming was performed at least once per year or on irregular basis "when needed" (most times less than once a year) in 64 herds (=18.2%), twice a year in 284 herds (=81.8%). In 38 herds (11.2%), cattle had no access to pasture 12 months a year.

The overall herd and animal prevalence of different claw disorders and the deviation about the different prevalence classes due to between herd and claw trimmer variance is shown in Table 3 (a,b,c). In 31% of all investigated cows, no claw disorders were registered, 31.5% of all cows had 1 disorder and for 2, 3 and >3 disorders, this percentage was respectively 23.7, 10.4 and 3.4 % (Table 2a). The different prevalence classes for the different claw disorders is demonstrated in Table 2b. Digital dermatitis was not found on 7% of the farms and when present on a farm, it is most found in the prevalence classes 1-20% and 21-40%. The prevalence of ID in a herd is in general somewhat higher than DD and comparable with the prevalence classes of SH. Chronic laminitis, WLD, SU and HYP were mostly found in the lowest prevalence classes. IP was seldom diagnosed at herd trimming (not mentioned in the Table 2b). Table 2d shows the prevalences of the different claw lesions on herd, cow and claw level. The distribution of all lesions over the left and the right hind leg was equal.

When interdigital dermatitis was diagnosed in a cow, the lesion was found on both hind legs in 68.2% of the cows. When digital dermatitis was diagnosed in a cow, the lesion was found on both hind legs in just 30.5% and IP was diagnosed on both hind legs in 2% of the cows, when IP was diagnosed. SH, WLD, SU and HYP were diagnosed on both hind legs respectively in 70.3, 19.0, 16.7 and 26.7% of the cases (Table 2c).

Table 3 shows the correlations between lesions at different level. Some remarkable findings were a strong correlation between DD - HYP, ID - SH and SH - WLD at herd level. Significant correlations were found between SH - SU, DD - ID, DD - HYP and SH - WLD at cow level.

Table 2a. Percentage of cows with different kind of claw-lesions (n=21330).

Number of different claw disorders per cow	%
0	31.0
1	31.5
2	23.7
3	10.4
>3	3.4

Table 2b. Frequency of herds (numbers and %) with digital lesions divided in different animal level prevalence classes.

Prevalence classes	D.D.		I.D.		S.H		C.L.		W.L.D.		S.U.		H.Y.P.	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Herds with P=0	23	7.0	16	4.4	6	1.7	4	1.1	36	10.3	36	10.5	59	17.2
(0<P≤ 0.20)	163	46.8	94	27.0	98	28.2	318	92.4	305	87.6	333	96.8	333	96.8
(0.21<P≤ 0.40)	115	33.0	98	28.2	88	25.3	25	7.3	38	11.1	11	3.2	10	2.9
(0.41<P≤ 0.60)	42	12.1	83	23.8	73	21.2	1	0.3	1	0.3	0	0	1	0.3
(0.61<P≤ 1.00)	7	2.0	73	20.9	83	23.8	0	0	0	0	0	0	0	0

(DD=Digital dermatitis; ID= Interdigital dermatitis; SH=Sole haemorrhage; CL= Chronic laminitis; SU= Sole ulcer; WLD= White line disease; HYP= Hyperplasie of the interdigital skin; and IP=Interdigital phlegmon).

Table 2c. Prevalence's of different claw-lesions on the herds (n= 348, mean, SD, min-max) and at cow level (n= 21329) at the left and the right claw.

	DD	ID	SH	CL	WLD	SU	HYP	IP
Prev. at herd level	0.219	0.378	0.383	0.043	0.100	0.060	0.061	0.007
S.D.	0.164	0.244	0.243	0.080	0.081	0.054	0.059	0.017
Min.	0	0	0	0	0	0	0	0
Max.	0.783	1	0.943	0.426	0.438	0.281	0.408	0.143
S.D.	0.413	0.485	0.485	0.208	0.298	0.234	0.239	0.084
Prev. left claw	14.3	32.4	32.4	3.6	6.5	3.3	3.8	0.4
Prev. right claw	14.2	31.2	32.0	3.8	5.2	3.5	3.9	0.4
Both hind legs	30.5	68.2	70.3	??	19.0	16.7	26.7	2

(DD=Digital dermatitis; ID= Interdigital dermatitis; SH=Sole haemorrhage; CL= Chronic laminitis; SU= Sole ulcer; WLD= White line disease; HYP= Hyperplasie of the interdigital skin; and IP=Interdigital phlegmon).

Table 3. Correlations between different type of claw lesions at herd (n=348) and cow (n= 21.329) level (data from routine claw trimming in Dutch dairy herds, 2002-2003). Vertical correlations at herd level, horizontal correlations at cow level.

		cow level							
H	Lesion	DD	ID	SH	CL	SU	WLD	HYP	IP
E	DD	XXX	0.136*	0.062*	-0.018	0.005	-0.019	0.131*	0.054*
R	ID	0.319*	XXX	0.146*	0.119*	0.019	0.031*	0.121*	0.010
D	SH	0.215*	0.445*	XXX	-0.042*	0.110*	0.151*	0.078*	0.005
L	CL	-0.044	0.220*	-0.104	XXX	0.092*	0.091*	0.056*	-0.005
E	SU	0.212*	-0.023	0.184*	0.135*	XXX	0.116*	0.057*	0.020
V	WLD	-0.003	0.146*	0.428*	0.114*	0.228*	XXX	0.028*	-0.012
E	HYP	0.493*	0.273*	0.330*	-0.076	0.123	0.085	XXX	0.031
L	IP	0.283*	0.028	0.049	-0.022	0.113	-0.030	0.000	XXX

(DD=Digital dermatitis; ID= Interdigital dermatitis; SH=Sole haemorrhage; CL= Chronic laminitis; SU= Sole ulcer; WLD= White line disease; HYP= Hyperplasie of the interdigital skin; and IP=Interdigital phlegmon).
*P< 0.001

Discussion

The objective of this study was to estimate prevalence's and correlations between different claw disorders as part

of an overall aim to improve claw health of dairy cattle in the Netherlands.
The average herd size in this study is somewhat higher than the average herd size in the Netherlands in the period our study was performed (56.3 cows/ milking herd, CBS 2003). Farmers that were willing to cooperate might have been more interested in hoof-health matters, and therefore their cows might have had a better hoof-health than other herds in the population (Mill and Ward, 1994). All herds did participate in a claw trimming schedule for trimming the hind hooves, so there has been no random sampling. As found in most field investigations of lameness in dairy cattle (e.g. Manske et al., 2002; Somers et al., 2003), the prevalence of claw disorders was highly variable between herds.
Due to the disproportionate representation of herds from the north-eastern part of the country, the study might not be representative for the soundness of the claws of all cows in dairy herds in the Netherlands. On the other hand, cubicle housing with a concrete floor is the predominant housing type for dairy cattle in the Netherlands. Approximately 90% of the dairy cows are kept in cubicle houses with a slatted floor (Braam and Swierstra, 1997), while about 10% are kept on solid concrete floors. Where the floors of the housing are hypothesised as a crucial factor in locomotion, little difference in claw health between solid and slatted floors was found by Somers et al. (2003), and the distribution of the herds in our study are representative for the Dutch dairy herds, the outcome of this study has reflected the current condition of the hind claws in the Netherlands at the moment of routine corrective trimming.
In our study we found a lower percentage of total claw disorders and especially for DD and SH (21.9% and 37.8% on slatted floors) in comparison with Somers et al. (2003), who found respectively 30.0% and 47.1%. These differences might be a result of the different objectives of the studies, the different way in which claw disorders were defined, scored and registered and the selection of farmers. Besides that, much attention has been paid (different media and lectures for farmers) on treatment and prevention since the last 2 years.
In our study it was found that in the majority of the cases (67%) that if interdigital dermatitis was diagnosed that this was found on both hind legs. For the presence of digital dermatitis we found just in 33% that this was diagnosed on both hind legs. These remarkable finding between both infectious claw disorders, where it was found in general on the right foot as often as on the left, could possibly be declared out of the differences in expression of the two disorders: DD: acute illness with an acute lameness and painful and ID: sub-acute illness with sub-clinical lameness and less painful.

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Literature

Bargai, U. 2000. Lameness in a dairy herd: an epidemiological model. Comput. Cont. Educ. Pract. 22, 58-67.
Braam, C.R. and Swierstra, D. 1997. Developments in design of concrete floors in dairy cattle houses; impact of environmental demands. Pages 11-22 in Proc. Concr. For a Sustain. Agric., Stavanger, Norway.
Brand, A., Noordhuizen, J.P.T.M. & Schukken, Y.H. 1996 Herd health and production management in dairy practice. Wageningen Pers. Chapter 7 part 1.
CBS. 2003. Agricultural Census 2003. Statistics Netherlands, ed. Elsevier, Voorburg, The Netherlands.
Farm Animal welfare Council. 1997. Report on Welfare of Dairy Cattle. FAWC. Tolworth, United Kingdom.
Groen, A.F., Hellinga, I. and Oldebroek K. 1994. Genetic correlations of clinical mastitis and feet and leg problems with milk yield and type traits in Dutch Black and White dairy cattle. Neth. J. of Agric. Sci. 42-4; 371-378.
Manske, T., Hultgren J., Bergsten, C. 2002. Prevalence and interrelationships of hoof lesions and lameness in Swedisch dairy cows. Prev. Vet. Med. 54: 247-263.
Mill, J.M. and Ward, W.R. 1994 Lameness and the farmers' knowledge, training and awareness. Vet. Rec. 134, 162-164.
SAS User's Guide: Statistics, Version 8.e , 2000 .SAS Inst., INC., Cary, NC.
Somers J.G.J.C., Frankena K, Noordhuizen-Stassen E.N. en Metz J.H.M. 2003. Prevalence of claw disorders in Dutch dairy cows exposed to several floor systems. J. Dairy Sci. 86: 2082-2093.
Sprecher D.J., Hostetler D.E. and Kaneene, J.B. 1997. A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. Theriogenology 47, 1179-1187.
Weaver, A.D. 2000. Lameness. Pages 149-202 in The Health of Dairy Cattle. A.H. Andrews, ed. Blackwell Science, Oxford, United Kingdom.

THE EFFECTS OF SEASON AND STAGE OF LACTATION ON LAMENESS IN 900 DAIRY COWS

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Introduction

In 1997 a study investigating the impact of oral biotin supplementation on lameness in dairy cows was initiated.

The results of this study demonstrated that white line disease was almost halved in cattle supplemented with biotin (Hedges et al, 2001) and that this reduction had most impact in cattle above parity three which had been supplemented with oral biotin for at least six months (Poetzch et al., 2003). This intervention study also provided a unique opportunity to collect accurate information on the timing and causes of lameness in the 900 cows that were monitored for 18 months from five herds in Gloucestershire.

Materials and Methods

The farmers identified when cows were lame and all lame cows were then examined, at no cost to the farmer, by one of a team of veterinarians. The veterinarians were trained to use a standard nomenclature to identify lesions and to identify the lesion that they considered was the cause of lameness. The date of inspection, the cow and farm identification and the cause of lameness were recorded on a standard form. Lame feet were photographed to record the lesion causing lameness and this enabled checks to be made on the accuracy of recording and on the consistency of the terminology used when vets met at regular review meetings.. Also, all the herds were locomotion scored by their attending vet at their routine visit or at least four times per year to ensure that all lame cows were examined. The occurrence of the top four causes of lameness: sole ulcer, white line disease, foul and digital dermatitis were explored descriptively. The occurrence of each cause of lameness is presented as an incidence rate, that is, the number of occurrences per 100 cow years in the study. The proportion of cases of each type of lameness were presented by stage of lactation, time of year and occurrence in heifers versus cows.

Results

During the 18 month study 70% of the 900 cows became lame, a total of 700 cases of lameness. The overall incidence rate (IR) of lameness was 69 cases per 100 cow years. Approximately 50% of the lameness was caused by sole ulcer (SU), white line disease (WLD) and digital dermatitis (DD) and foul, the IR of the three former causes of lameness were approximately equal at around 11 - 13 cases per 100 cow years; the incidence of 'foul' was almost half at 7 cases per 100 cow years (Figure 1 and Table 1). The remaining causes of lameness were attributed to one or more of 15 other abnormalities (Table 1). Lameness occurred throughout lactation but peaked at three months after calving (Figure 3). There were interesting patterns of incidence of lameness by time of year. Digital dermatitis and foul were primarily a disease of winter (although these was a peak of foul in August) and sole ulcer occurred all year round. White line disease occurred in cycles of four- month waves.

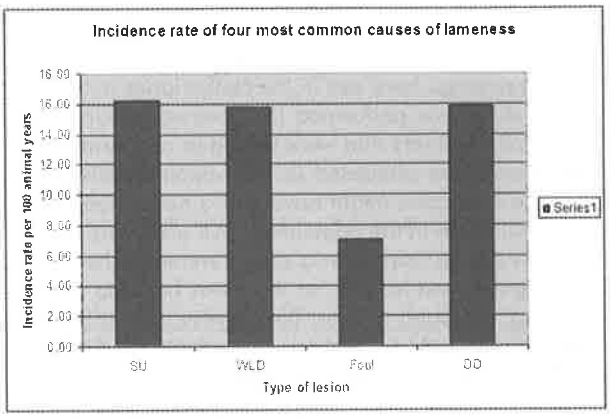

Figure 1. Incidence rate of the four most common lesions. SU = sole ulcer, WLD = white line disease, digital dermatitis

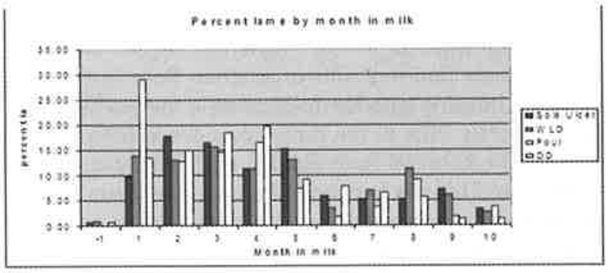

Figure 2. Incidence rate of four most common lesions by hether the animal joined the study as a pregnant heifer or DD = older cow

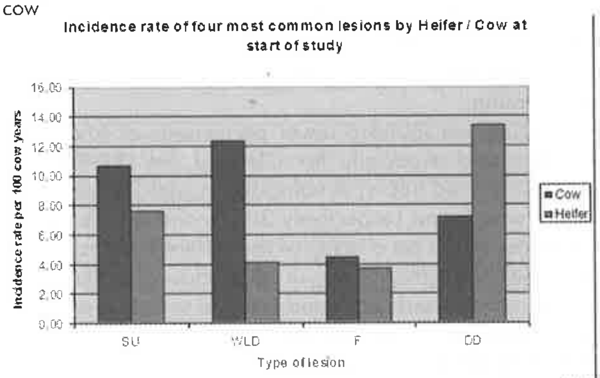

Figure 3. Occurrence of lameness by month in milk from within one month precalving (-1) to the tenth month post calving

Table 1. The incidence rate per 100 cow years of of all causes of lameness.

Cause of lameness	Incidence	Cause of lameness	Incidence
Sole Ulcer	13.84	Foot trauma	0.36
White line	12.68	Toe pus	0.36
Digital Dermatitis	11.96	Laminitis	0.27
Interdigital necrobacillosis	7.14	Leg oedema	0.27
Heel ulcer	5.80	Pedal bone necrosis	0.27
Foreign body	3.12	Soft sole	0.27
Sole ledge/Overgrowth	2.95	Nerve damage	0.27
Under run sole	2.14	Swollen knee	0.18
No diagnosis	1.78	Septic foot	0.18
Sole haemorrhage	2.14	Hock damage	0.18
Granulation tissue	1.25	Septic arthritis	0.18
Interdigital growth	1.16	Thin soles	0.09
Slurry heel	0.27	Pus and joint sepsis	0.09
Under run wall	0.18	Under run heel	0.09
Vertical wall fissure	0.53	Joint injury	0.09
Swollen hock	0.53	False toe	0.09
Coronary band trauma	0.53	Claw polyp	0.09
Horizontal wall fissure	0.53	Fetlock damage	0.09
Abscess	0.53	Arthritic changes	0.09
Block trauma	0.45	Heel bruising/trauma	0.09
Swollen fetlock	0.36	Hip injury	0.09
Sole penetration	0.36		

Discussion

This study is unusual in that all lame cows were examined by experienced veterinarians, that discussion meetings took place throughout the study to confirm standardisation of lesion description, and that it was based on lesions causing lameness
rather than lesions only. At 69 cases per 100 cow years this study identified a higher incidence of lameness than in a previous UK study at 54.6 cases per 100 (Clarkson et al., 1996). As expected, sole ulcers, white line disease, digital dermatitis and 'foul' were the main causes of lameness, although the incidence of heel ulcers was almost as high as foul (5.8 vs 7.46 cases per 100 cows), indicating the considerable importance of this recently described condition as a cause of lameness in dairy cows (Blowey et al., 2000).
The increase in the incidence of lameness during early lactation has been postulated as a defect in the corium in late dry period / early lactation and that this disrupts horn formation, which is later seen as hoof disorders such as sole ulcer and white line disease. At calving, horn growth is reduced, wear increases and a number of other management changes such as different housing and diet may lead to either an increased fragility of the corium and/or bruising from excess trauma. Assuming a sole 10 - 15mm thick, growing at 5mm per month. it would take two months for defective horn to grow down through the bearing surface of the There was also a marked increase in proportion of the infectious causes of lameness, digital dermatitis and foul, in the first month after calving, with digital dermatitis continuing to increase until the 4th month. Part of this increase is likely to be due to the housing of cattle after calving, and the fact that in this study the majority of cows were late summer and autumn calving, viz. from August to November .It is well known that there is a marked immune suppression in all periparturient cows, and this immune suppression is likely to predispose to an increase in digital dermatitis and foul. There was an increase in DD each year from a trough in August to a peak in March/April, i.e. from around the time of housing until the cows go out to graze pastures in the spring. It is not possible to separate the effects of stage of lactation from those of period of housing. Increased exposure to slurry is likely to increase the incidence of both DD and foul.
The incidence of hoof disorders, namely sole ulcers and white line disease, was higher in cows than in heifers. Poetzch and others (2003) reported that the incidence of white line disorders increased with increasing parity. The effect of parity on sole ulcers was less marked (Hedges, 2001); this might indicate that sole ulcers have a slightly different aetiology, also supported by the very different occurrence in these lesions by time of year. DD was much more prevalent in heifers than in cows (Figure 2). One might expect an environmental effect, e.g. heifers afraid to use cubicles and hence spend longer standing in the slurry, to affect both conditions. This did not appear to be the case. This could be because 'foul' is affected by the environment less than DD, or because some other factor, e.g. immune response, is more important for DD.

Conclusions

Detailed descriptive studies of this nature are an invaluable tool in our understanding of the nature and causes of lameness, although care needs to taken to ensure that a distinction is made between conjecture and fact.

References

Blowey, RW Watson C, Green LE, Hedges VJ, Packington AJ (2000) The incidence of heel ulcers in a study of lameness in five UK dairy herds Proc 11th Int Symposium on Disorders of the Ruminant Digit, Parma, Italy, September 2000
Hedges, V. J., R. W., Blowey, A. J. Packington, C. J. O'Callaghan and L. E. Green. 2001. A longitudinal field trial of the effect of biotin supplementation on lameness in dairy cows. J. Dairy Sci. 84:1969-1975.
Pötzsch C. J., Hedges, V. J., Blowey, R. W., Packington, A. J. and Green, L.E. (2003) The effect of biotin supplementation on white line disease in dairy cattle. Journal of Dairy Science 86 2577 - 258

OCCURRENCE OF HOOF DISEASES IN DAIRY CATTLE IN FINLAND

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Iintroduction

No national recording system for hoof diseases has existed in Finland. Some hoof trimmers keep regular records of their clients´, but national records of lameness have been made only by vets called for clinical cases.
In 2001 only 20-30 % of farms in Finland had their animals´ hooves trimmed regularly, and hoof problems with lameness were an increasing concern.
During this same year the Finnish Hoof Trimmers, Suomen Rehu Ltd. and Vetman Ltd. instigated a programme to improve the hoof health and welfare of cows on Finnish dairy farms. This programme included a national recording system and information on how to improve hoof health with regular professional hoof trimming, good, sensible feeding, a healthy environment and adequate care of growing heifers.
Hoof trimmers were first taught to keep similar records to standardize their recordings by the project group before actually using the system. The Data system for results was developed by Suomen Rehu Ltd.

Information on farming management (tie stalls/loose housing, cold loose housing), bedding, flooring and feeding systems was collected by the hoof trimmers from those farms who had agreed to participate. Data were analysed in spring 2003, and the programme was named Healthy Hooves (Terveet Sorkat). One aim of this national programme was to determine prevalence and severity of corkscrew claw. Hoof trimmers in Finland have been very concerned about corkscrew claws because even a 90° screw in the sole creates numerous problems.

Matherial and methods

The form for recording hoof findings and diseases was sent to all participating hoof trimmers. Diseases were divided into the following 10 groups: Sole hemorrhages, chronic laminitis, white line disease, sole ulcer, interdigital dermatitis, heel-horn erosion, digital dermatitis, 90° screw claw, other hoof diseases, and preventive hoof care (no findings in this category).
Acute laminitis and interdigital flegmon were excluded, because these are usually diagnosed by veterinarians, not by hoof trimmers, and they are recorded separately.
Data collection began at the end of February 2002. After one and a half years, almost 1200 (>10%) farms from different areas in Finland were involved in the programme and were visited regularly by hoof trimmers.

We included only those farms where at least 90 % of cows were trimmed at least once during 2002 and for which we had all the farm information requested. Hoof trimming for the same farm was included in the analysis only once. Thus, 425 farms were left for more accurate statistical analysis. The diseases were categorized into the groups of 1. non-infectious diseases (= hemorrhages, chronic laminitis, white line disease, sole ulcer), 2. infectious diseases (= interdigital dermatitis, heel-horn erosion, digital dermatitis) and 3. other diseases (= Screw claw and others).
Corkcrew claw refers to claws with 180° screwing; screw claw in our data refers to a clow which is screwed 90°. Only screw soles of hind leg claws were considered.

All models were tested in a logistic regression mixed model (SAS:GLIMMIX), and contained all factors in the hypotheses, including the random hoof trimmer id and the random farm id nested in the hoof trimmer id.

Results

By the end of the year 2002, a total of 29 038 cows had been trimmed.
The Healthy Hooves Programme received recordings from 50 of the 80 hoof trimmers from March 2002 to December 2002 (15-2300 cows per hoof trimmer)
Over half (51,3 %) of all cows were trimmed without any findings, the remainder (48.7 %) having at least one problem in their hooves; 33 % of the cows had more than one problem.
Prevalences of hoof diseases on farms participating in the Healthy Hooves Programme in 2002. are presented in Table 1. The uncorrected data ignore the hoof trimming percentage; corrected data include only data from those farms with complete information and where almost 90% of cows were trimmed in 2002.

Table 1: Prevalence of hoof diseases on farm participating in the Healthy Hooves Programme in Finland in 2002.

Prevalences of hoof diseases in	Uncorrected data (%)	Corrected data (%)
Hemorrhages and chronic laminitis	28,70	25,89
White line disease	10,30	8,60
Non-infectious diseases	39,40	38,26
Sole ulcer	4,30	3,77
Infectious diseases on all farms	10,90	8,52
Infectious diseases in loose houses		16,39
90° Screw claw	11,10	9,83

The corrected data included 87 farms and 4723 cows (average 54.3 cows per farm) in loose housing systems, and 338 farms and 8539 cows (average 25.3 cows per farm, which is more than the average number of cows per farm in Finland) in tie stalls.

Cows in loose housing systems had twice the odds ratio for screw claws of cows in tie stalls. Recording differences between hoof trimmers were small. In 25 % of the farms, no screw claws were observed. The incidence of hemorrhages and chronic laminitis was not correlated to feeding, number of hoof trimmings, flooring or housing in this data.
Cows in loose housing systems had 13 times higher odds ratios for infectious diseases. Large differences were present between hoof trimmers in recording infectious diseases.

Discussion

The Healthy Hooves project, which was implemented for practical reasons and needs has some limitations. Participation was voluntary, and thus, the farms taking part were likely to be "better farms". Another drawback is that over half of the farms did not use hoof trimming for all cows, and when these farms were excluded from analysis, the number of the farms in the study was drastically reduced..
Prevalences of hoof diseases were higher in all data than in corrected data, indicating that farms that did not use hoof trimming for all cows had probably trimmed more cows with symptoms. The corrected data are therefore more accurate.
The prevalence of hoof diseases indicates the risk for laminitis-like lesions. The prevalence of non-infectious diseases (38 %) was rather high. The incidence of subclinical and chronic laminitis was not correlated to feeding,

number of hoof trimmings, flooring or housing in this data. This suggests that data collection should have been planned more carefully. Farm samples were collected on rubber mat flooring (i.e. a soft area) and the type of mat was not taken into consideration. Differences between mats could be significant. The same applies to feeding. Further, more accurate investigations on feeding and laminitis in Finland are needed; these have already been started this autumn.

The prevalence of infectious diseases was low. This is probably because of the large number of tie stalls in Finland; no infectious diseases are usually found in tie stalls. However, over the last few years, farmers have built more loose housing systems so the prevalence of infectious diseases is expected to increase in the future. Inside loose houses the prevalence was also relatively low. The reason for this could be the large amount of new loose housing systems in Finland. Some of the farms also use outside walking areas with snow for cows, which probably helps. Differences between hoof trimmers may be another explanatory factor.
Our infectious diseases are mainly heel-horn erosion. Digital dermatitis so far is very rare in Finland.

The occurrence of screw claw was interesting. While no differences were observed between slatted floors and scraper floorings, loose housing systems have more risk for screw claws. Whether this is because of more walking or because heifers are more often raised on slatted hard floors in Finland, or for some other reason, remains obscure.

Acknowldgements

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PREVALENCE OF LAMENESS IN DAIRY AND BEEF CATTLE OF MARVDASHT AREA DURING THE WINTER

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Introduction

Lameness is considered to be a major welfare and economic problem on dairy farms (6, 7). Lameness economically rank third or fourth after mastitis, infertility and possibly metabolic diseases (1,2,5). Since no report was available about the incidence of lameness in Marvdasht area, this investigation was performed.

Materials and Methods

During winter of 1997, dairy and beef cattle of 68 farms of Marvdasht area were examined for lameness. Marvdasht is located in the north of Fars province in south Iran. These farms represent about 60 percent of total farms in the area. At first, the farmers were interviewed by questionaire to record the information. Information collected from herds included: breed, production purpose(dairy or beef), management, number of affected animals and their age. After that, by observation and clinical examination, the affected limbs, and finally type of lesions or diseases were recognised.

Results

Generally 2.39 percent of examined animals were lame. Distribution of disorders was as follow: dairy cows (56.92 %), beef cattle (43.07%). Pure breed of Freisian (54.61%), mixed breed of Freisian (20.76%) and native breed (24.61%). Most lesions were located in the foot (90.76%). The hindlimbs showed the most lesions(70.76%).The medial digits of forlimbs were least affected (4.54%) and the lateral digits of the hindlimbs were the most affected (18.18%). Among the different types of lameness, the most common were interdigital necrobacillosis or footrot, septic pododermatitis, sole ulcer and overgrown hoof. The details of foot lesions are shown in table 1.

Table 1. Distribution of foot lesions in lame cattle

	Front foot(%)	Hind foot(%)	Total (%)
Septic pododermatitis	3.80	10.47	14.27
Pododermatitis traumatica	1.90	3.80	5.70
Heel erosion	1.90	2.85	4.75
Sole ulcer	2.85	8.57	11.42
White line disease	–	1.90	1.90
Vertical hoof crack	–	0.95	0.95
Overgrown hoof	6.66	14.28	20.94
Congenital claw abnormality	2.85	–	2.85
Footrot	8.57	22.85	31.42
Interdigital dermatitis	–	5.71	5.71
Total (%)	28.53	71.38	100

Discussion

2.3 percent locomotor disorders of farms in Marvdasht area is not high, but it can and reduce the level of production. In most parts of Iran, rainfall is mainly in winter. Therefore the humidity of environment and wetting of bed could have an important role for occuring lameness in this area. The results of this survey are rather similar to others (3, 4, 7), although in some European countries the incidence of lameness is high due to more rain, and cold weather.

Foot and leg problems are a major health concern for many dairy farms. Most studies show a greater incidence(90%) of lameness in the foot, and of these, 90 % involve the rear feet. In this study about 70 % of lesions involved the hindlimbs. Some congenital lesions just involved forelimbs.

Some diseases such as interdigital necrobacillosis, footrot and hoof overgrowth are related to poor management. Lack of a regular schedule of hoof trimming was the major management problem of these farms.

Bed condition, rainfall, and management were the determining factors in the occurrence of lameness in this survey.

Acknowledgement

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References

1. Collick, DW ; Ward, WR and Dobson, H (1989). Association between types of lameness and fertility. Vet. Rec. 125: 103-106.

2. Dewes, HF (1978). Some aspects of lameness in dairy herds. New Zealand Vet. J. 26: 147-159.

3. Eddy, RG and Scott, CP (1980). Some observations on incidence of lameness in dairy cattle in Somerset. Vet. Rec. 106:140-144.

4. Rowlands, GJ ; Russell, AM and Williams, LA (1983). Effects of season, herd size, management system and veterinary practice on the lameness incidence in dairy cattle. Vet. Rec. 113: 441-445.

5. Weaver, AD (1994). Economic importance of digital disease in cattle. Bovine Prac. 14: 222-224.

6. Wells, S ; Trent, AM ; March, WE and Robinson, RA (1993). Prevalence and severity of lameness in lactating dairy cows in a sample of Minnesota and Wisconsin herds. JAVMA 202:78-81.

7. Whitaker, DA ; Kelly, JM and Smith EJ (1983). Incidence of lameness in dairy cows. Vet. Rec. 113: 60-62.

IDENTIFYING THE CAUSES OF LAMENESS: ON FARM DATA COLLECTION METHODS FOR A COHORT STUDY

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Introduction

Lameness encompasses a whole range of diseases and conditions that impair walking. As such identifying the causes of 'lameness' is inherently difficult. Some risk factors for specific feet and leg conditions have been identified in previous studies. For example, floor type, cubicle dimension, stage of lactation and milk production have been demonstrated to be associated with lameness in cattle (Faull et al., 1996; Leach et al, 1997; Green et al, 2002; Webster, 2002). As yet the relative importance of such factors has not been investigated and the associations between specific causes of lameness and management, environment and individual cow characteristics has not been fully elucidated.

This study aims to identify environmental and management conditions that influence the level and type of lameness on 53 dairy farms in England and Wales. The study uses data on occurrence of lameness collected by the farmers together with a range of recording techniques collected at visits to the farms. The data collection techniques and data collected at each visit to the farms are described below.

Materials and Methods

Fifty-three dairy farms in England and Wales were enrolled into the study in February 2003. The farms vary both in size (27-450 cows) and geographical location

(Cornwall, Devon, Somerset, Dorset, Gloucestershire, Wiltshire, Oxfordshire, Warwickshire, Staffordshire, Derbyshire, Yorkshire, Cheshire, Lancashire and Wales). One, or more, of three recorders; Zoe Barker (ZB), Rebecca Brassey (RB) and Jonathan Amory (JA) are visiting each farm 3-4 times between February 2003 and March 2004 (Table 1).

Recording lameness

The lesion recording form (Figure 1) has been provided to farmers/herdsmen and is been completed and returned by post each month. This is used to record the type and position of lesions. The age and production of each cow will be collected from national milk records.

Cow number identity	Site of lesion	Foot affected	Lame	Lesions seen	Date today	Time in milk (weeks)	Who trimmed foot
	(place a cross)	(circle one only)	(circle one)	(circle all appropriate, star * cause)			(circle all appropriate)
			Sound	Sole ulcer White line			
		LF RF	Not sound	Digital dermatitis Foul			Farmer
			Definitely	Other (please state)			
			lame	———			Foot Trimmer
		LH RH	Hobbling				
							Vet

Figure 1. Lesion recording form completed by farmer/herdsmen during foot trimming, one row per foot

Definitions of observations made at the visits

All milking and dry cows present on the farm are locomotion scored, scored for cleanliness and hock damage; these scores may vary with time, and so are made at each visit (Table 2). The method for locomotion scoring is based on that described by Sprecher et al., (1996) which assesses the posture of the animal through the presence or absence of an arched back when walking and standing. The method was validated on farm to ensure good agreement between recorders. Faecal consistency scores (Hughes , 2001) (Table 3) are also made at each visit.

Permanent structures on the farm such as buildings, yards, tracks and walkways are not expected to vary between visits so have been assessed once either at summer or winter visits. The observations are comprised of visual assessments and measurements of dimensions of e.g. cubicles, tracks and buildings. Methods to determine the suitability of the cubicles in terms of size and comfort are currently being developed and will be used during visit 4.

A farmer interview was done at visit 2. This requested data on the management of all aspects of the dairy system, including housing, breeding, nutrition and herd health. A second questionnaire will be completed at the final visit and is designed to gauge farmers' attitudes to their system and lameness in their herds.

Table 1 - Timing of farm visits and observations made

	Visit 1	Visit 2	Visit 3	Visit 4
Time	Feb-May 03	June-Aug 03	Nov- Dec 03	Jan - Mar 04
Number of farms visited	53	52	<51	51
Location of herd	Housing/ early turnout	Summer Grazing	Early winter housing	Late winter housing
Recordings Taken				
From each identified cow				
Locomotion score	x	x	x	x
Cleanliness of hind quarters	x	x	x	x
Evidence of hock damage	x	x	x	x
From the environment				
Assessment of bedding quality and quantity	x		x	x
Consistency of faecal pats	x	x	x	•
Observations of summer environment		x		
Observations of winter environment			x	x
Observations of cubicle use				x
From the farmer				
Records of all foot trimming, posted each month				
Farmer interview Questionnaire		x		
Final interview Questionnaire				x

Table 2 - Scores used to assess cow condition and environment

	Score 1	Score 2	Score 3
Locomotion scores (all cows)	Flat back when standing Flat back when walking	Flat back when standing Arched back walking	Arched back when standing Arched back when walking
Cow cleanliness (all cows)	Completely clean hind quarters	Dirty feet and legs	Dirty feet, legs and flank
Hock damage (all cows)	No Lesions on hock	Lesion(s) visible on hock	Severe lesion, bleeding or swelling
Bedding cleanliness (all houses)	Bedding clean and dry	Bedding lightly soiled but dry	Bedding heavily soiled and wet
Bedding depth (all houses)	Bedding deep and even	Bedding uneven and patchy	Lack of Bedding

Table 3 - Faecal scoring system (Hughes 2001)

DESCRIPTION	SCORE
Dehydrated - Dry and stiff	1
Normal - Circular pat with depression in centre	2
Loose - Slightly liquid, and thinly spread	3
Liquid - Thin watery faeces, may be abnormal colour or contain undigested forage	

Results

The scores used to assess the cows condition and their environment have been a successful method to collect data on the farms, with minimal disruption to the farmers/herdsmen. The main problem remains the identification of cows with dirty or faded brand numbers during locomotion scoring if either the farmer/herdsman is not available or the cows are exiting the parlour in large groups. The cow cleanliness score may not be as sensitive as hoped as the feet of very few cows remain completely clean so over 99 per cent of cows in visits 1 and 2 were score 2 or 3.

There was 100% compliance for the farmer interview questionnaire. The data collected may be prone to recall bias because of differing opinions among the farmers/herdsmen as to its importance (Martin et al, 1987). However, our direct observations will assist in assessing the internal validity of the study.

Visit 3 is currently under way and observations of the winter housing are being made. Farmers/herdsmen remain enthusiastic and continue to return lesion recording forms regularly. To date, two farms have withdrawn from the study. One farmer has sold her herd and another reduced herd size and extra responsibilities.

Conclusions

The data on potential risks will be compared with locomotion score and lesion records and the significance of these risk factors determined. The final aim is to produce a clear list of recommendations specific to farms, which farmers may use as a tool to reduce lameness in their herd.

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References

FAULL, W.B., HUGHES, J.W., CLARKSON, M.J., DOWNHAM, D.Y., MANSON, F.J., MERRIT, J.B., MURRAY, R.D., RUSSELL, W.B., SUTHERST, J.E., AND WARD, W.R. (1996). Epidemiology of lameness in dairy cattle: the influence of cubicles and indoor and outdoors walking surfaces. Vet Rec. 139:130-136

GREEN, L.E., HEDGES, V.J., SCHUKKEN, Y.H., BLOWEY, R.W. AND PACKINGTON, A.J. (2002). The impact of clinical lameness on the milk yield of dairy cows. J. Dairy Sci. 85:2250-2256

HUGHES J. (2001). A system for assessing cow cleanliness. In Practice 23 517-524/

LEACH, K.A., LOGUE, D.N., KEMPSOM, S.A., OFFER, J.E., TERNENT, H.E., AND RANDELLS, J.M. (1997) Claw lesions in dairy cattle: development of sole ulcer and white line haemorrhages during the first lactation. The Vet. Journal. 154: 215-225

MARTIN, S.W., MEEK, A.H. AND WILLEBERG, P. (1987) Veterinary Epidemiology, principles and methods. Iowa State University Press, Ames, Iowa.

SPRECHER, D.J., HOSTLER, D.E., AND KANEENE, J.B. (1996). A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. Theriolgenology. 47:1179-1187.

WEBSTER, A.J.F. (2002) Effects of housing practices on the development of foot lesions in dairy heifer in early lactation. Vet Rec. 151:9-12

LAMENESS TREATMENT RATES IN WISCONSIN DAIRY HERDS

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Introduction

Few studies have documented lameness treatment rates in North American dairy herds. Case definition, ability to detect lame individuals and frequency of delivery of the hoof trimming program significantly affect the incidence of new and recurrent cases.

Materials and Methods

Treatment records for lame cows were kept on 10 Wisconsin dairy herds for 12 months using records obtained from the farmer and hoof-trimmer. Six herds were free stall housed and four herds were tie stall housed. Half used sand bedded stalls and half used a rubber mat or mattress. Stall base types were equally distributed by housing type. Each herd was visited twice during the recording period and lameness prevalence assessed using a four-point locomotion scoring system described by Cook (2003). A new lameness treatment was defined as a limb-case treated within one 28 day period. A recurrent treatment occurred in the same limb, greater than 28 days after the first treatment for the same condition.

Results

Mean (SE) herd size was 145 (28.2) cows. Mean (SE) herd lameness prevalence, determined by averaging the results of locomotion scoring at two visits during the recording period, was 22.2% (2.8). A total of 1155 lameness treatment events were recorded in the 10 herds. Lameness affected 39.2% (7.3) of the herd on average. Herd mean (SE) lameness treatment rate was 69.1 (15.6) limb-cases per 100 cows per year, with a mean recurrence rate of 12.2%. Mean annual lameness treatment rate was higher in free stall herds at 91.1 limb-cases per 100 cows than in tie stall herds at 40.2 limb-cases per 100 cows.

The mean ratio of incidence to prevalence for all herds was 3.2:1, with a range from 0.7:1 to 5.4:1. The lowest ratios were from herds where the farmer was the sole lameness recorder, and the highest ratios were found in herds with the most frequent hoof-trimming visits.

Papillomatous digital dermatitis (PDD) was the most common lesion found, accounting for 56.8% of all treatments. Other infectious lesions, such as foot rot, were uncommon. Sole ulcer (18.4%), white line disease (10.43%) and sole hemorrhage (6.4%) were the most common claw horn lesions identified.

Examination of lameness rates by lesion type in herds utilizing different stall surfaces indicated some interesting trends. Mean (SE) treatment rate for claw horn lesions was 37.4% (14.7) in mat and mattress stall herds and 16.8% (3.7) in sand stall herds (P = 0.16). Mean (SE) treatment rate for infectious lesions was 58.5% (19.3) in sand stall herds and 23.3% (17.2) in mat and mattress stall herds (P=0.7). Table 1 shows mean rate and proportion for each lesion by stall base.

Table 1. Comparison of mean annual lameness treatment rates per 100 cows (and proportion of total) by cause, between herds using sand stalls (n=5) and herds using mat or mattress stalls (n=5).

Mean rate per 100 cows (Proportion of total)	Sand Stall Herds	Mat and Mattress Stall Herds
	Infectious and Other Lesions	
Papillomatous Digital Dermatitis (PDD)	55.7 (74.1)	20.5 (33.9)
PDD with Interdigital Hyperplasia (IDH)	1.2 (1.6)	0.5 (0.8)
IDH	0 (0)	0.2 (0.3)
Foot Rot	0.6 (0.8)	1.0 (1.6)
Heel Horn Erosion	1.0 (1.3)	1.1 (1.9)
Rate of Infectious Lesions	58.5	23.3
	Claw Horn Lesions	
Sole Hemorrhage	2.5 (3.3)	7.9 (13.0)
Sole Ulcer	11.2 (14.9)	18.2 (30.1)
Toe Ulcer	0.5 (0.6)	0.6 (1.0)
White Line Disease	2.2 (2.9)	10.4 (17.1)
Septic	0.4 (0.5)	0 (0)
Claw Horn Lesion with PDD	0 (0)	0.3 (0.4)
Rate of Claw Horn Lesions	16.8	37.4

The highest rates of lameness treatment by month occurred in September and during the period January - February. The proportion of limb treatments due to claw horn lesions compared to infectious lesions peaked in September at 59.4% of all treatments. For all other months, infectious lesions exceeded 50% of all treatments (Figure 1).

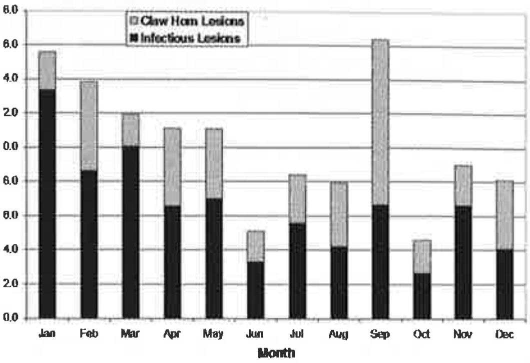

Figure 1. Monthly lameness treatment rate for 10 herds, presented by lesion type.

Discussion

PDD is by far the most common cause of lameness in North American dairy herds in both free stall and tie stall housing. Cold weather during the late winter may lead to manure handling problems in the alleys and reduced frequency of foot-bathing, triggering an increased rate of new PDD infections. Sole ulcer was the most common claw horn lesion identified, with white line disease also common. Claw horn lesion treatment rate was 55% less in sand stall herds than in mattress herds, suggesting some benefit to using sand bedding in the prevention of these lesions. This benefit did not extend to the control of PDD. Changes in cow behavior combined with an increased risk of sub-acute ruminal acidosis during the period of summer heat stress may be responsible for an increased rate of claw horn lesions observed around 2 months later.

References

Cook, N. B. 2003. Prevalence of lameness among dairy cattle in Wisconsin as a function of housing type and stall surface. JAVMA 223:1324-1328.

THE USE OF MATHEMATICAL MODELS AND OBSERVATIONAL EPIDEMIOLOGICAL STUDIES TO INVESTIGATE THE TRANSMISSION OF BOVINE DIGITAL DERMATITIS

Vink W.D., Murray R.D., Demirkan I., Carter S.D., French N.P.

Abstract:

A multigroup mathematical model of a dairy herd was formulated to represent the transmission of bovine digital dermatitis within and between five management groups (unweaned calves, weaned calves, heifers, lactating cows and dry cows). This poster describes the initial formulation of the mathematical model, which has informed the design of both cross-sectional and longitudinal field studies. A pilot study was performed on four farms, which were classified as high prevalence, medium prevalence, low prevalence and free of digital dermatitis. A questionnaire and housing inspection were performed on each farm prior to testing. Serum samples were taken from all dairy cattle on the farms at the time of testing (a total of 1270). Body hygiene scoring and foot condition scoring were carried out. The feet of 286 animals were inspected for clinical lesions. Data on age and management group, serological status, presence or absence of clinical lesions and hygiene scores were used to revise and parameterise the model, to refine case definition and infection status of animals within a herd, and to inform the design of the longitudinal study.

UTILIZATION OF A WEBSITE ON CATTLE LAMENESS TO PROMOTE TRAINING, DISCUSSION AND THE DISSEMINATION OF INFORMATION

Vink W.D., Logue D.N., French N.P.

Abstract:

A collaborative project was undertaken by the University of Liverpool and the Scottish Agricultural College, the primary objective of which was to recommend effective and appropriate intervention strategies for reducing lameness in dairy cattle. A website was developed to make the results of the project universally accessible. Longer-term objectives are to inform and involve a broader audience (including dairy farmers, foot trimmers and researchers), by providing a platform for disseminating information and promoting discussion on currently relevant topics. This website was fundamentally updated and redesigned in 2003; it is accessible at http://www.cattle-lameness.com/. This poster presents the design and functionality of this website, including its potential as an information tool and decision support system for farmers and professionals. It enables access to be made to databases containing literature references to scientific papers published on cattle lameness. It provides a flexible learning programme on cattle lameness and footcare, and presents the results of current and past research. Discussion fora on three relevant topics have been included. Updates can be received via news articles. It is hoped that a facility such as this will increase active discussion and collaboration between all stakeholders involved in the field of cattle lameness.

Some reflections on research on Bovine Laminitis Aspects of clinical and fundamental research

Christer Bergsten and Christoph Mülling

The idea of our presentation is to describe laminitis research from a clinicians perspective and from a lab researchers view embedded in the historical background of this area of research. Looking back at the real amazing multidisciplinary nature of our symposia where we have a remarkable mix of very practical and very fundamental papers and everything in between. We think time has come to get together a glance at our activities and methodology. A glance from the clinicians practical oriented perspective as well as from the rather theoretical point of view of a lab researcher. Christoph will provide an insight into a morphologists work and Christer will illustrate some clinical research.

How old is specific research on laminitis in cattle? And who was the first laminitis researcher?

Recognition of bovine lameness has been documented for hundred of years and then the reason was most commonly footrot (interdigital phlegmon, necrobacillosis etc). Today, footrot still is a problem for some herds but other infectious diseases and claw horn disorders are less explored and of more research interest. Research on laminitis as an independent area of interest started in the eighteen-forties. The first reference reporting on laminitis was that of Bedel in 1839 who described a case with laminitis after parturition. Interestingly enough nowadays a focus of research on pathogenesis of laminitis is now on the molecular effects of parturition on the claw tissues. The Swedish Veterinary Journal in 1896 reported, "Inflammation of the claw corium is the most common cause of lameness and the often misshapen claw can be secondarily affected by a purulent process and/or the claw capsule could fall off. The affected animal is useless for work after it has been affected". This showed that clinical experiences from laminitis like symptoms were the same at that time, and that the prognosis was made from a practical point of view. Obviously the ox was also affected by laminitis. Today, economical and animal well-being considerations are more relevant. In the animal protection legislation we have to make a decision based on knowledge and experience on whether a specific claw disease is meaningful to treat or not, from an animal welfare point of view. If not successful to treat, it is stated that the animal should immediately be euthanized.
Since the nineteenth century there were several periods of higher interest for research interrupted by times were there was little interest and consequently little progress. In contrast the interest in laminitis in horses is much older. It goes back to antiquity. First descriptions on equine laminitis were given by Xenophon, Aristoteles, Apsyrtus and Hiercoles. The Greek name of the disease, Kritiasis, relates to overfeeding of barley. Xenofon noticed the characteristic symptoms, the hooves started to bleed and the horse became recumbent, which were due to pain in the hooves. These are the same symptoms observed in cattle today as was also described thoroughly by Nilsson (1963), who was aware of this relationship; "Laminitis and the diseases of the bovine extremities on the whole have long been overshadowed by the corresponding disease of the horse". It is natural that research on the aetiology of equine laminitis is used to explain bovine laminitis. But we should remember that bovine claws also were used in the research of equine laminitis. Obel (1948) as the first looking after a histopathological explanation on laminitis and he used claws and dew claws in his experiments because of the easiness to get samples and similarities with equine hooves. However, transferring existing knowledge from equine laminitis to the bovine claw without considering differences in biology, i.e. in morphology and function may have hindered progress in research for periods.

The early describers of laminitis were mainly clinicians and practitioners. Rusterholz presented a detailed description of the specific traumatic sole ulcer in 1920. His explanation of the disease already included several essential aspects of the modern understanding on aetiology and pathogenesis of this disease although it was not related to any metabolic disturbance at that time. Later, toxic, chemical agents through the feedstuff or metastatic spread from infections have also been proposed in the aetiology of laminitis and related claw horn lesions.

The first real interdisciplinary study including a thorough morphological description of laminitis as well as secondary affections such as sole ulcer was that of the Swedish practitioner Sture Nilsson published in 1963. He employed clinical morphological and experimental methodology for a broad interdisciplinary approach to the aetiology and pathogenesis of laminitis in cattle. His monograph added a lot to the knowledge about laminitis and created the basis for more than 30 years. Important was that he carried out specific claw research and was well aware of the fact that one needs specific claw research to increase understanding of bovine laminitis. In this way Nilsson was the pioneer of the modern multidisciplinary research carried out nowadays. And one has to admit that some of his ideas and hypotheses have at least in part been reinvented once in a while.

At present we are in a very active period of specific research on the aetiology and particularly the pathogenesis of the diseases of the bovine hoof. A lot of interest focuses on lameness as an economic and welfare issue in dairy farming world-wide. In Europe there is a strong political and growing public interest that welfare concerns are addressed in farm animal husbandry. A lot of funding was successfully raised in many countries for clinical as well as for fundamental research on national level or international in networks. The

LAMECOW project which we are going to hear more about during the symposium is an example for multidisciplinary international research. New methods are used to attack the questions from particular clinical observations till today's advanced epidemiology. One could say that we are living in good times for lameness researchers and their work.

What is the nature of laminitis research?

Claw diseases are caused by numerous causative factors. Due to the multifactorial nature of laminitis, ruminants research activities have become more and more interdisciplinary. Lameness research employs more and more modern methods of epidemiology, cellular biology and molecular biology. However, the same time the very practical problems and work continue.

Starting in 1992 at Rebild with 2 or 3 anatomical papers the last three symposia were characterised by a rapidly increase in number of papers presenting work that originates from fundamental research. At Lucerne Parma and Orlando whole sessions were coined by fundamental research papers sometimes very complex and difficult to follow because of the methodology. On the other hand with an increasing number of hoof trimmers participating we have very practical oriented very applied contributions as well.

Our symposia are now characterised by a mixture of very practical orientated and working people and those who are doing research in very fundamental and theoretical areas. Our work has become inter/multidisciplinary and we have now a very active exchange of ideas and results between the different disciplines.

My (Christoph´s) personal impression when I attended my first lameness symposium at Rebild in 1992 was that this group and their meetings provide an excellent interdisciplinary platform for research, research with the overall aim to reduce the incidence of lameness and number of animals suffering from pain and dysfunction. In a number of colleagues and in our personal perception the interdisciplinarity and the exchange of practical and theoretical ideas between practical and theoretical working individuals has continuously developed and is now the outstanding attribute of our symposia. Something that we should maintain and cultivate as a precious good.

Dissemination of knowledge - education

Parallel to the research activities there is growing awareness/sensitivity for the urgent need of improved education of all people involved in dairy farming.

At Orlando Paul Greenough clearly emphasised the deplorable discrepancy between the increasing accumulation of knowledge about biology and diseases of the bovine hoof on one side and the actual level of knowledge of veterinarians on the other side (Greenough, 2002). This is distressing because a good standard of knowledge is a prerequisite for successful prevention of lameness.

We certainly have a strong need for improvements in education and training of Anatomy, Physiology and Pathology of the bovine hoof. The prerequisite is that we consequently incorporate the acquired knowledge into the curricula of veterinary education but also at an adapted level in the training and education of everybody working in the dairy industry.

Anatomy - Morphology - Pathomorphology

As mentioned earlier the first systematic pathohistological examination of claw tissue was done by Nilsson (1963). He presented pictures from different forms and stages of laminitis in cattle. He was also the first to associate clinical laminitis with specific symptoms of the claws like sole haemorrhages and sole ulcer.

Morphological examination

Morphological examination comprises light and electron microscopy (LM and EM) as different ways using different technologies of looking at tissue sections. For both LM and EM a broad variety of methods is available such as histological staining methods immunostaining enzyme histochemistry, in situ hybridisation and others.

Preservation of the tissue cells and cellular components as early post mortem and as fresh as possible is of outstanding importance. Basically two ways are available. Cryofixation in liquid nitrogen or in a freezer and chemical fixation. Common fixatives for light microscopy are formaldehyde in a variety of preparations, mixtures of formalin and alcohol or mild acids. The fixation can be carried out either by immersion, i.e. by simply putting the tissue in the fixative or by perfusion, i.e. injection of the fixative in the vascular system of the organ. Subsequent to fixation tissue is embedded in wax or resins to allow cutting of very thin section son a microtome. Sections are mounted onto glass slides. After removal of the embedding medium staining, immunostaining or in situ hybridisation is carried out. The stained sections are covered with cover slides and ready for microscopic examination.

Electron microscopy

Transmission electron microscopy

An accelerated electron beam is transmitted through a tissue section of 90 nm in thickness in an evacuated steel column. The picture is observed on a fluorescent screen and documented by a camera. Conventional Transmission Electron microscopy enables primary magnifications of a few hundred thousand times revealing subcellular structures such as cell organelles and even large molecules. First electron-micrographs of normal claw tissue were included in the thesis of Wilkens in 1963. More than 20 years later systematic electron microscopic examination of healthy claws was done by Dirks in 1985 and by Mülling (1993) and Warzecha (1993). Electron microscopy provided insights in the ultrastructure of claw tissue and the cellular events during epidermal differentiation. It was also used to study the ultrastructural alterations in dyskeratotic epidermis in laminitis.

Scanning electron microscopy

An electron beam is scanning the surface of a dehydrated and metal coated specimen. The surface formations are depicted and the information is transformed into a monitor picture or digital image. This technique is ideal for examination of surfaces and their three-dimensional aspects. In combination with micro-maceration an microseparation techniques SEM investigations have revealed the architecture of the dermal papillary body in the different regions of the claw. SEM investigation of corrosion casts of the dermal vascular system has gathered detailed knowledge on the angioarchitecture of the dermal vascular system included the dense capillary network and special structure for regulation of the blood flow (Hirschberg, 1998, 2001, Vermunt and Leach, 1992

Method of first choice for studies on the microcirculation within organs is the scanning electron microscopic investigation of corrosion casts of the blood vessels. An low-viscosity resin is injected in the vascular system after complete removal of the blood. Via a cannula in the main digital artery. Once the vessels are complete filled the resin rest for hardening. The tissue is completely removed by heat maceration in hot sodium hydroxide for about 2 weeks. The remaining cast is cleaned, dissected and mounted for SEM observation.

In vitro models

For studying the cellular and molecular mechanisms of laminitis there are now a number of in vitro models available: tissue explants two and three dimensional cell culture systems and additionally in near future an isolated limb perfusion model. This allows in vitro studies in highly standardises cell clines, standardised cell culture and testing of the results in more complex organotypic cultures and finally testing in the isolated limb perfusion model under conditions as close as possible in a model to the in vivo situation. Tissue explant studies have already provided important insights into regulation off differentiation in healthy and diseased claw tissue (Hendry et al, 1999, 2001,, 2003). Cell lines and simple three-dimensional cell cultures are available already (Nebel et al., 2002). A novel three-dimensional organotypic culture system will be introduced during this symposium (Nebel et al., 2004). And the isolated limb perfusion model developed under the LAME COW project provides a very promising platform for future studies on the pathomechanisms of laminitis (Wüstenberg et al., 2004).

A number of historical candidate factors and substances will be tested in these systems and evaluated for their influence on claw cell and tissue integrity. The demonstration of the still missing link between metabolic disorders and laminitic alterations in claw tissue is an outstanding aim of this area of research. The weakest link of the attachment of the pedal bone at the inside of the claw capsule the locus minoris resistentiae has yet to be demonstrated. Disturbances in the microcirculation by vasoactive agents and the insult to the laminar region of the dermis has never convincingly been demonstrated in cattle.

Three dimensional cell culture provides a powerful tool to study the complex paracrine regulation of cellular differentiation and dermo-epidermal signalling. The accumulation of knowledge in human skin research is breathtaking. We can learn a lot from the studies on pathomechanisms of skin diseases and transfer methodology to laminitis research.

Claw biology and laminitis

Laminitis in cattle is an specific disease of the bovine species. Its aetiology, pathogenesis and nature are characteristic and different from the equine hoof because of the structural and biomechanical differences. Consequently diagnostic procedures, strategies for treatment and prophylactics have to consider the specific aspects of claw biology.

Laminitis is a systemic disease with a primary manifestation in the claw because of its unique morphology. Anatomical studies have shown that there are three critical structures in the claw. These are: 1. the dermal vascular system, 2. the differentiating epidermal cells; and 3. the connective tissue system in the suspensory and the supportive apparatus.

1. Dermal vascular system of the claw

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 of microcirculation (Hirschberg et al 1998, 2001). 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. It has frequently been hypothesised that AVAs play an important role in initial stages of bovine laminitis (Vermunt and Leach 1992). More recent thorough micro-corrosion cast studies done by Hirschberg and others (1999, 2001) demonstrated that the vascular system of the bovine claw is different in many aspects from that described for the horse hoof. Interestingly enough these authors show that there are almost no AVAs in the vascular system of healthy claws. Although present and needed in skin for thermoregulation, they are not functionally required and do not develop in the modified skin of the claw. Consequently the hypotheses favouring closure of AVAs as a major initial event in the pathogenesis of laminitis do not apply. From these studies we have some evidence that an increased number of AVAs detectable in diseased claws occurs as an adaptive structural change. The results of Christmann and others (2002) provide initial evidence that the alterations of microcirculation related to initial laminitic like events are different in the claw. They demonstrated in grain overload steers an increase in capillary pressure and post-capillary resistance. This facilitates transvascular movement and an increase in tissue pressure. Digital venous constriction is thought to be the initial step in these events. However, in contrast to horses no significant changes in pre-capillary resistance and digital blood flow was found. The differences in haemodynamic changes observed in the bovine digit compared to the equine may contribute to the differences in clinical presentation of laminitis (Christmann et al 2002).

Nilsson described in 1963 formation of "Neocapillaries" in the dermis of claws of animals suffering from subacute laminitis. Recently Hirschberg and others (2003) investigated the formation of new blood vessels in diseased claws. Basing on their morphological studies on the microvascularisation and angioadaptation of the claw they postulate a central role of pododermal angiogenesis for development and function of the claw and for the pathogenesis of laminitis. It well may be that studies on the angiogenesis, i.e. the de novo formation of blood vessels will significantly contribute to the understanding of laminitis and open new doors for the prevention of laminitis.

2. Differentiating epidermal cells

Formation of claw horn (i.e. cornification of the epidermis) is the result of proliferation, cellular differentiation, i.e. keratinization and programmed cell death (Mülling and Budras 1998). This process is controlled by a variety of bioactive molecules such as growth factors and neuropeptides provided by the dermis and/or the vascular system.

There is growing evidence from morphological and in vitro studies that disruption of the differentiation of keratinocytes in the differentiating hoof epidermis is the major reactive event during pathogenesis of laminitis occurring secondary to dermal alterations resulting in disruption of appropriate epidermal supply. This was the theory suggested in equine laminitis by Obel (1948) and has been explored also by Ekfalck (1991) and Wattle (2001). Most of what we can see in subacute or chronic laminitis on the claw is related to or a result of the reactive changes in the epidermis.

3. Weakness of the connective tissue in the suspensory apparatus

Basing on recent studies two major hypothesis have been developed and established in current discussion about the etiology of the postulated weakness of the connective tissue part of the suspensory apparatus. One hypothesis favours the central role of matrix metalloproteinsases and its activation through a novel gelatinolytic protease "hoofase" (Webster and Tarlton 2002) the other the effects of hormones particularly relaxin present in the peripartal period (Jönsson, 1969).

Pathophysiology - missing link behind the nutritional and metabolic influences

Epidermal differentiation is essentially dependent on supply with nutrients and oxygen from the capillaries in the underlying dermis and is very sensitive to any disruption of supply. According to this whatever changes occur in permeability or perfusion of the dermal blood vessels necessarily has consequences for epidermal differentiation and horn formation. However, the question as to what causes vascular alterations still need to be answered. A number of candidate factors mediators are listed and discussed in literature to be responsible for initial changes in dermal vessels. Ranking among the most frequently listed are endotoxins, histamine and lactate. Effects of these on the vascular system have been demonstrated in several studies. The link between these factors and disturbances of microcirculation has not yet been proven in vivo.

When Nilsson first described bovine laminitis in the 1960s (1963), the release of histamine from protein sources in the diet was found to be a reasonable explanation to laminitis. This was also the theory of the cause of laminitis from the first PhD thesis on equine laminitis some decades earlier. □kerblom (1934; □kerblom, 1977) demonstrated experimentally that E. coli bacteria enzymes could decarboxylate histidin to histamine in protein-rich grain. It has not, however, been possible to provoke laminitis symptoms by injecting histamine alone in the blood in equine or bovine experiments. However, when steers were first overfed with grain followed by a histamine injection, they showed severe laminitis symptoms that lasted for one to four days (Takahashi, 1981). Russell (2001) showed that feed lot steers on a high starch diet developed acid resistant E coli in a significant higher number than did steers with excess to hay in the diet. This gave the idea that histamine producing E coli could be more of a risk for laminitis if they were acid resistant and produced their histamine in the intestines. In search for histamine producing bacteria a new bacterium was detected that has the ability to produce more histamine than any earlier known bacterium Garner (2002). Histamine producing bacteria was found in faeces from grain fed cattle and in cecum from a horse but not in ruminal fluid from cattle fed hay. These findings add some more knowledge that could get us a little closer to the role of histamine in the aetiology if laminitis. Another often-adopted explanation of the etiology is that large quantities of endotoxins (toxin released by gram negative bacteria such as E. coli) are produced as when the gastro-intestinal metabolism is disturbed in an acid environment. The mucous membranes of the gastro-intestinal system normally protect it from absorbing toxins into the blood stream. The mucosa of the bovine fore stomachs are especially fairly resistant while the intestines may be more susceptible. (That may explain the higher susceptibility for laminitis in horses than in cattle). But, if the natural barrier is weakened or damaged and if absorbed to the circulatory system, endotoxins are extremely potent and trigger a prostaglandin cascade (chain reaction). An imbalance of the prostaglandins tromoxane and prostacycline is evident, and thrombi are produced, which obstruct the small blood vessels (capillaries) of the laminar corium. The blood circulation is locally deteriorated and the result can be compared to a "heart attack" of the feet. The decreased oxygen and nutrient supply damages the corium's horn-producing cells, as earlier been explained. Elevated levels of endotoxins have been observed in laminitis and thrombosis was evident in the corium (Andersson and Bergman, 1980). Researchers have also tried to provoke laminitis by injecting endotoxins into the circulatory system in cattle with results hard to interpret (Mortensen et al.1986).

A more recent hypothesis of equine laminitis has found that laminar enzymes, possibly metalloproteinases, have altered the basal membrane of the laminar corium. Once the junction is affected the corium and horn could be stretched apart from each other, relative to the severity of the lesion (Pollitt, 1996). Streptococcus bovis in the equine gut has been suggested as a potentional metalloproteinase activator in acute laminitis (Pollitt, 1999). This is interesting as background for bovine laminitis, as lactic-acid producing bacteria are associated with the disease. So far, this hypothesis has not been reproduced in cattle but studies are carried on. Recent preliminary results from studies of Belknap and others (2002) demonstrating the expression of proinflammatory mediators suggest that a systemic and digital inflammatory process is involved in laminitis in cattle.

Traumatic effects of laminitis

Once the claw bone attachment in the horn capsule has been disrupted the claw is at risk for further damage. Due to the severity of the disruption, loading and biomechanics, the claw bone sinks and/or rotates more or less permanently inside the horn capsule and the prominent parts of the bone contuse the adjacent sole corium (Ossent and Lisher, 1998). The contusion or pinching of the corium depends on the counter pressure and causes a secondary inflammation of the sole corium with oedema and haemorrhaging. The haemorrhages from the corium will be incorporated into the growing horn and will be visible at trimming. The lesions on the sole can be compared to a print of the bone where it hits the sole. The most commonly affected regions are the rear part of the claw bone, recognized as the "typical" sole-ulcer site, the junction of the sole and white line of the posterior outer wall, and the toe area. These lesions have been used extensively in research to find triggering factors. Smedegaard (1964) made a scoring for sole haemorrhages and sole ulcers and related the lesions to trauma alone. In late seventies Peterse (1979) made a similar scoring and invented the vocabulary sub clinical laminitis for lesions detected without clinical symptoms at that time. The time lapse from the initial insult until the haemorrhage can be detected depends on the growth rate of the sole and the sole thickness and with mild corium lesions.

As it takes two to three months until the hoof lesions are visible, the close relationship between laminitis and hoof lesions has not always been well understood. Sometimes sole haemorrhages are misinterpreted as stone bruises. Changes of the bone position inside the claw capsule also affect the grooving wall. You can see a horizontal break point of the wall, also known as a hardship groove or laminitic ring, after each disruption of horn growth (Greenough, 1985). The newly produced wall horn of the upper part follows the new position of the bone while the lower part of the wall, beneath the groove, reflects the position of the bone before the laminitis period. Thus, the rotated bone inside the capsule is reflected as a concavity of the wall outside. In claws from slaughtered cows with chronic recurrent laminitis, a concavity of the dorsal wall was associated with a permanent rotation of the claw bone and protrusion of the corium (Kehler and Sohrt, 2000). Whether there is a separation evident between laminar corium and horn lamellae is disputed. Lischer did not reveal any separation in cases where there was a significant sinking of the claw bone. An own (Bergsten) investigation of feed lot steers with severe laminitis problems found, however, all characteristic laminitis related claw horn lesions macroscopically and histologically; sole and white line haemorrhages, P3 rotation, toe necrosis, double soles, contusion of corium at typical sole ulcer site, histological separation of lamellar horn with cap horn formation etc. Thus, pathological changes from clinical cases can surely vary from one case to the other and for the future we sould need more sophisticated methods to study the development of biomechanical changes in a living animal, maybe with the use of magnetic resonance imaging.

Claw conformation and weight distribution

The weight distribution between the inner and outer claws, of the sole and wall area within each claw, and the shock absorbing mechanisms in the foot are related to the trauma of the sole corium towards the floor. At normal gait the heel bulbs and the outer wall will make the first contact with the ground and the weight will be distributed equally between the outer and inner claw. While the soft bulbs reduce the shock of the rear part of the claws the weight on the wall will successfully be transferred on the sole due to slight splaying of the claws. Inside the capsule, the suspensory apparatus made of collagen tissue and the digital cushions made of fat pads protect the sole corium.

Sole lesions are rarely seen in free-ranged cattle on a soft foundation and their soles are concave from the outer wall to the central part. Tranter and Morris (1992) found that when animals were moved from pasture to hard floors during lactation, the rear outer claw's natural sole concavity disappeared while the inner claw's concavity remained. This is a common observation from claws that have been on concrete for a while. A vicious cycle starts, which increases sole growth and wear in the overloaded area and causes asymmetric claws. Asymmetric rear claws, where the outer claw is larger than the inner claw and the sole is flat, is the most common site for sole lesions. Consequently, if the natural concave shape of the claws has disappeared, and the sole is flat or convex, the sole will take more weight than the wall initially, and shock absorption will rely more on the suspensory apparatus and the digital cushion. Lischer et al. (2002) compared normal feet and feet affected with sole ulcers from slaughtered cows. Claws with sole ulcers had a more sunken claw bone, and more compressed soft tissues and less fat in the cushions.

Claw trimming

Claw trimming has proved to reduce lameness associated with laminitis when correctly performed. Manson and Leaver (1988) showed that trimming the feet before calving resulted in fewer lame cows than when the feet were not trimmed. Preliminary results from a Swedish study Manske et al. (2002) showed almost twice as many sole ulcers in animals trimmed only once each year compared to those trimmed twice. One function of claw of trimming is to detect lesions at an early stage before clinical symptoms or severe lesions develop. The other part is to prevent lesions by correcting the loading. Distl and Mair (1990) showed that when the distribution of weight between the claws is unequal and the sole bears too much weight, there is greater risk for sole lesions. When the feet are trimmed, the weight is more equally distributed between and within the claws. However, it is more difficult to equalize the weight distribution in cows with excessive wear, i.e. cows in free stalls on abrasive concrete floors. Today research is under progress in several countries to evaluate weight distribution between and within claws in dairy cows under different conditions.

Preventing laminitis, claw lesions and lameness

Laminitis with secondary claw lesions is frequently a herd problem. As with other production diseases, laminitis is multifactorial and management decisions are critical to reduce most laminitis risk factors. Thus, the possibility to prevent laminitis increases if farmers or managers understand and are aware of the problem and its consequences. There will be larger herds, and breeding for higher milk yield will continue. Laminitis-related lesions have shown to have a high heritability (Manske 2001, personal communication). It would be beneficial to include lameness and claw lesion records in breeding programs in the future. Such records can also help dairy producers monitor herd problems and make decisions to improve hoof health, and to evaluate the effects of a preventative protocol (Bergsten 2000)

Perspectives of laminitis research

The future of lameness research is multidisciplinary. The adaptation and application of modern methods of cell and molecular biology will present an increasing number of results enabling a better and more comprehensive understanding of the pathomechanisms of laminitis. Of central importance will be the elucidation of the initial events and the early stages during pathogenesis of laminitis. The effects of bioactive molecules on the dermal vascular system and of molecules responsible for the alterations of the connective tissue in the suspensory apparatus have to be studied using all in vitro models available. And the findings have to be related to ruminant metabolism to identify links between metabolic stress and laminitis. Another focus will be on the reactive changes in the epidermis particularly the mechanisms involved in disruption of epidermal differentiation.

Good and active research on one side and vivid communication between practical and fundamental research are the prerequisites for meeting the challenges.

Towards the end of our observations we would like to emphasise an issue of outstanding importance for the future: This is the dissemination of the acquired knowledge and its incorporation into education and training at different levels and in different languages. This we are convinced, will turn out at least as challenging as research.

We have to make use of all the acquired knowledge generated by so many active and enthusiastic researchers to strengthen the prevention of lameness in accordance to the motto of this symposium " prevention is better than cure".

References

Andersson L, Bergman A, (1980), Pathology of bovine laminitis especially as regards vascular lesions. Acta vet. scand. 21, 559-566.

Bedel, (1839): Bulletin de la Société centrale de med. vet. Cited according to Nilsson (1963).

Belknap EB, Christmann U, Cochran A, Belknap J (2002) Expression of proinflammatory mediators and vasoactive substances in laminitic cattle. In: Shearer J (ed.), Proceedings of the 12th International symposium on lameness in Ruminants, January 9-13, Orlando, 387.

Bergsten C, (2000). Work shop report about the documentation of claw diseases; Part 2, Descriptive nomenclature and scoring of foot lesions at hoof trimming,. In: International Symposium on Disorders of the Ruminant Digit & International Conference on Bovine Lameness, Parma, Sept 3-7, pp. 226-229.

Boosman R, Németh F and others (1991) Bovine laminitis: clinical aspects, pathology and pathogenesis with reference to acute equine laminitis. The Veterinary Quarterly 13, 163-171.

Budras K-D, Mülling Ch, Horowitz A (1996) The rate of keratinization of the wall segment of the cattle hoof and its relationship to width and structure of the zona alba (white line) with respect to claw disease. Am J Vet Res 57,444-555.

Christmann U, Belknap EB, Lin HC, Belknap JK (2002) Evaluation of hemodynamics in the normal and laminitic bovine digit. In: Shearer J (ed.), Proceedings of the 12th International symposium on lameness in Ruminants, January 9-13, Orlando, 165-166.

Dirks, C. (1985): Makroskopische, licht- und elektronenmikroskopische Untersuchungen über den Rückenteil der Rinderklaue, Diss. med. vet., Berlin.

Distl O, Mair A, (1990). Pedobarometric forces at the sole/floor interface. In: Update in Cattle Lameness, VI Int Symp Disorders Ruminant Digit, Liverpool, pp. 143-162.

Ebeid M (1993) Bovine Laminitis: A Review. Veterinary Bulletin 63, 205-213.

Hirschberg, R, Mülling Ch, Bragulla H (1999) Microvasculature of the Bovine Claw Demonstrated by Improved Micro-Corrosion- Casting Technique. Microscopy Research and Technique 45, 184-197.

Ekfalck A, (1991). Studies on the morphology and biochemistry of the epidermal tissue of the equine and the bovine hoof with special reference to laminitis. PhD Thesis. Swed. Univ. Agric. Sci., Dept. Med. Surg., Uppsala, Sweden.

Garner MR, Flint JF, Russell JB, (2002), Allisonella histaminiformans gen. nov., sp. nov. A novel bacterium that produces histamine, utilizes histidine as its sole energy source, and could play a role in bovine and equine laminitis. Syst Appl Microbiol 25, 498-506.

Greenough PR, (1985), The subclinical laminitis syndrome. Bov. Pract. 20, 144-149.

Greenough PR, Vermunt JJ (1991) Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. The Veterinary Record 128, 11-17.

Greenough, PR (2002) The State of our Art. Proceedings of the 12th International Symposium on Lameness in Ruminants, January 9 - 13, 2002, Orlando/Florida, pp 1-4.

Hendry K.A.K., MacCallum, et al.(1999): Effect of endocrine and paracrine factors on protein synthesis and cell proliferation in bovine hoof tissue culture. J Dairy Res 66: 23-33.

Hendry K.A.K., MacCallum, et al. (2001): Keratinocyte proliferentiation and differentiation in healthy and ulcerated bovine hoof epidermis. J Dairy Res 68, 525-537.

Hendry KA, Knight CH, Galbraith H, Wilde CJ. (2003) Basement membrane integrity and keratinisation in healthy and ulcerated bovine hoof tissue. J Dairy Res 70:19-27

Hirschberg R, Mülling Ch, Budras K-D (2001) Pododermal angioarchitecture of the bovine claw in relation to form and function of the papillary body. A scanning electron microscopic study. Microscopy Research and Technique 54, 375-385.

Hirschberg, R.M., Budras K.-D. and J. Plendel (2003): Pododermale Angiogenese-Neue Ansätze zur Therapie von Klauenerkrankungen. 25. Kongress der Deutschen Veterinärmedizinischen Gesellschaft, 3.-4. April 2003, Berlin, 91-94.

Jönsson G, Pehrson B, (1969), Studies of the downer syndrome in dairy cows. Zbl. Vet. Med. A 16, 757-784.

Kehler W, Sohrt JT, (2000). Special considerations of laminitis lesions for claw trimming (hind claw of Holstein-Friesian cows). In: International Symposium on Disorders of the Ruminant Digit & International Conference on Bovine Lameness, Parma, Sept 3-7, pp. 258-259.

Lischer, C. and P. Ossent (1994). Laminitis in cattle: a literature review. Tierarztl Prax 22 , 424-432.

Lischer C, Ossent P, Raber M, Geyer H, (2002), Suspensory structures and supporting tissues of the third phalanx of cows and their relevance to the development of typical sole ulcers (Rusterholz ulcers). Vet Rec 151, 694-698.

Manske T, Hultgren J, Bergsten C, (2002), The effect of claw trimming on the hoof health of Swedish dairy cattle. Prev Vet Med 54, 113-129.

Manson FJ, Leaver JD, (1987), Effect of Concentrate to Silage Ratio and Hoof Trimming on Lameness in Dairy-Cows. Anim. Prod. 44, 469-469.

Mortensen K, Hesselholt M, Basse A, (1986) Pathogenesis of bovine laminitis (diffuse aseptic pododermatitis).Experimental models. In: 4th World Congress on Diseases of Cattle, Dublin, Ireland, pp. 1025-1030.

Mülling, Ch. (1993): Struktur, Verhornung und Hornqualität in Ballen, Sohle und Weisser Linie der Rinderklaue und ihre Bedeutung für Klauenerkrankungen, Diss. med. vet., Berlin.

Mülling Ch (1998): Anatomy and Structure of hoof horn. In: Lischer CJ (ed.),Proceedings 10th International Symposium on Lameness in Ruminants, September 7-10, 1998, Lucerne, 176-188.

Mülling Ch, Budras K-D (1998): Influence of environmental factors on horn quality of the bovine hoof. In: Lischer CJ (ed.), Proceedings 10th International Symposium on Lameness in Ruminants, September 7-10, 1998, Lucerne, 214-215.

Nebel U, Ch K W Mülling: Organotypic cell culture of bovine hoof keratinocytes.
The 12th International Symposium on Lameness in Ruminants, January 9-13, 2002

Nebel, U., Hoffmann, D., Mülling, Ch.K.W., Budras, K.-D. (2004): Co-culture of epidermal and dermal bovine hoof cells in a perfusion chamber - -Development and first experimental experiences. Paper to be presented on this symposium.

Nilsson, S-A. (1963): Clinical Morphological and Experimental Studies of laminitis in cattle. Acta Vet. Scand. 4 (Suppl.1).

Ossent P (1999) Subclinical bovine laminitis. Cattle Practice 7, 193-195.

Ossent P, Lischer CJ (1998) Bovine laminitis: the lesions and their pathogenesis. In Practice 20, 415-427.

Peterse DJ, (1980). Judgement of bovine claws by the occurrence of sole lesions (De beoordeling van de runderklauw op basis van het optreden van zoollaesies). PhD Thesis. Rijksuniversiteit, Utrecht, the Netherlands.

Pollit CC (1990) The Pathophysiology of equine laminitis. In: Vet. Cont. Educ. (ed.), Foot lameness in horses, Palmerston, North Australia, 65-71.

Pollitt CC, (1996), Basement membrane pathology: a feature of acute equine laminitis. Equine Vet J 28, 38-46.

Pollitt CC, (1999) Equine laminitis: A revised pathophysiology. In: American Association of Equine Practitioners, pp. 188-192.

Russell JB, Rychlik JL, (2001), Factors that alter rumen microbial ecology. Science 292, 1119-1122.

Rusterholz, A. (1920): Das spezifisch traumatische Klauen-Sohlengeschwür des Rindes. Schweizer Archiv für Tierheilkunde 62, 421-446.

Smilie RH, Hoblet KH and others (1999) Subclinical laminitis in dairy cows: use of severity of hoof lesions to rank and evaluate herds. Veterinary Record 144, 17-21.

Smedegaard HH, (1964), Contusion of the sole in cattle. The Veterinarian 2, 119-139.

Takahashi K, Young BA, (1981), Effects of grain overfeeding and histamine injection on physiological responses related to acute bovine laminitis. Jap. J. vet. Sci. 43, 375-385.

Tarlton JF, Webster AJ (2000) Biomechanical and biochemical analyses of changes in the supportive structure of the bovine hoof in maiden heifers and around the time of first calving. In: Mortellaro CM, de Vecchis L, Brizzi A (eds.), Proceedings of the 11th International Symposium on lameness in Ruminants, Parma Italy, 113-118.

Tarlton JF, Webster AJ (2002) A biochemical and biomechanical basis for the pathogenesis of claw horn lesions. In: Shearer J (ed.), Proceedings of the 12th International symposium on lameness in Ruminants, January 9-13, Orlando, 395-398.

Tranter WP, Morris RS, (1992), Hoof Growth and Wear in Pasture-Fed Dairy-Cattle. N.Z. vet. J. 40, 89-96.

Wattle OS, (2001), Cytokeratins of the matrices of the chestnut (torus carpeus) and periople in horses with acute laminitis. Am J Vet Res 62, 425-432.

Vermunt JJ, Leach DH (1992) A scanning electron microscopic study of the vascular system of the bovine hind limb claw. N Z Vet J 40, 146-154.

Vermunt, JJ, Greenough PR (1994) Predisposing factors of laminitis in cattle. Br Vet J 150, 151-64.

Warzecha, Ch. (1993): The Ruminant Hoof. Diss. med. vet., Zürich.

Westerfeld I, Mülling Ch, Budras K-D(2000)Suspensory apparatus of the distal phalanx (Ph III) in the bovine hoof. In: Mortellaro CM, de Vecchis L, Brizzi A (eds.), Proceedings of the 11th International Symposium on lameness in Ruminants, Parma Italy, 103-105.

Wilkens H. (1963): Zur makroskopischen und mikroskopischen Morphologi ede rRinderklaue mit einem Vergelich vder Architektur von Klauen- und Hufröhrchen. Hannover, Tierärztl. Hochschule, Habilitationsschrift, 1963.

Wüstenberg, R.-Y., Mülling, Ch. K. W., Budras, K.-D. (2004): The isolated haemoperfused cow limb as a model for studying the pathogenesis of bovine laminitis. Paper to be presented on this symposium.

□kerblom E, (1934). Über die Ätiologie und Pathogenese der Futterrehe beim Pferde. PhD Thesis. Stockholm, Sweden.

□kerblom E, (1977), F□ng - histamine - rheumatic symptoms. Svensk Vet.tidn. 29, 5-10.

MECHANICAL PROPERTIES OF THE SOLE HOOF HORN OF HEIFERS BEFORE AND DURING THE FIRST LACTATION - A PREDICTION OF LAMENESS SUSCEPTIBILITY

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Introduction

The beginning of the lactation period has been related to an increase in the number and severity of claw horn lesions of dairy cows and heifers (Offer et al., 2000). The rearing of heifers, their growth rate, feeding and the occurrence of foot lesions have been found to affect the occurrence of lameness in later life (Thomas et al., 1999). The aim of this experiment was to study the incidence of lameness and the pattern of lesion formation in the sole horn during the pre- and postpartum period of first lactation heifers and to compare the results to mechanical tests of the hoof horn and the Holstein UK Index classification for conformation to predict heifers susceptibility to lameness.

Materials and Methods - Mechanical tests were performed on samples of hoof horn of the sole taken from 20 heifers at 40 days before calving and at 50, 100 and 150 days after calving. At the same time all claws were scored for the level of haemorrhage and ulceration of the sole horn. Locomotion score was assessed weekly and heifers were scored for the Holstein UK Index (HUKI) once during the first lactation. The heifers were kept at pasture and joined the lactating cow group one month before calving. After calving the heifers were kept in a straw yard in a separate group. Samples for the mechanical tests were collected of all claws and analysed on a texture analyser for punch resistance using a P/2N needle probe and for elastic modulus through a tension test. Five punch tests and 2 tension tests were completed on the sole and white line areas of each claw. The data was analysed by ANOVA - GLM (Minitab 12.0), using period and claw as fixed effects and thickness of the samples as a covariant when punch tests were analysed. The comparison of means was done using the Tukey test (95% confidence interval). Regression analysis was performed to test the effect of days in the postpartum period on the locomotion score. Correlation analysis between variables was done using Pearsons correlation.

Results

Effect of parturition on locomotion and lesion score and on mechanical tests. The locomotion score was found to increase significantly (P<0.001) from day 7 up to day 120 of the postpartum period and subsequently decreased after day 150 postpartum (Locomotion score = -8 x 10-5 days2 + 0.0212 days + 0.9759, R2adj. = 0.795). Hoof lesion score was calculated separately for the sole and white line areas of each claw of each hoof. The mean total lesion score of the sole and white line areas was found to increase significantly (P<0.001) from the prepartum to the postpartum period and during the postpartum period (-40 days - 764.1 and 584.5; 50 days - 1295.0 and 1180.2; 100 days - 1623.4 and 1636.9 for sole and white line respectively). The days of the peripartum period had no significant effect on mean elastic modulus of the tension test of the white line horn (day -40 - 91.32 N/mm2, day 50 - 77.78 N/mm2, day 100 - 78.62 N/mm2 and day150 - 104.12 N/mm2). Punch resistance of the white line horn decreased significantly between day 40 prepartum and days 50 and 100 of the postpartum period (day -40 - 6.92N a, day 50 - 5.92N c, day 100 - 5.88N bc and day 150 - 6.24N ab). The punch resistance and elastic modulus of the tension test of the sole horn increased significantly at day 100 postpartum (day -40 - 8.68N b and 88.76N/mm2b, day 50 - 8.60N b and 71.49N/mm2b, day 100 - 10.57Na and

97.96N/mm2b, day 150 - 10.74N a and 151.60N/mm2a). This increase in lesion score and in locomotion score after parturition has been reported in heifers and cows (Offer et al., 2000) and is related to physiological and management changes around parturition.

Lesion score, punch resistance and elastic modulus of different claws. The mean values for punch force, elastic modulus and lesion score of the sole and white line areas of the claw horn of the front and hind claws in different measurement periods are presented in Table 1. No difference was found between the elastic modulus of front and hind claws in any measurement period. No difference in the punch resistance and lesion score of claws was found in the prepartum period. The punch resistance of the sole and white line areas of the claw horn were significantly greater in the front claws compared with the hind claws in the postpartum period ($P<0.01$), but no significant difference was found between the inner and outer claws of front and hind feet. At 100 days postpartum the lesion score of the sole and white line areas was significantly greater in the hind claws compared to the front claws ($P<0.001$) and the increase in the lesion score was significantly greater in the hind claws compared with the front claws ($P<0.001$). In the hind feet, the outer claws had a significantly ($P<0.05$) greater lesion score compared to the inner claws during the postpartum period. In the front feet the lesion score was significantly higher ($P<0.05$) in the outer claws before parturition and in the inner claws ($P<0.01$) after parturition. The greater resistance to puncture in front claws compared to hind claws may be related to the different pressure distribution in these claws, predisposing the hind claw to suffer lesions through contusion.

Table 1: Punch resistance and lesion score of the sole and white line (wl) areas of the claw horn and elastic modulus of the claw horn of front and hind claws -40 and 100 days peripartum in first lactation heifers

Claws	front	hind	sem	P
Lesion score –40 days	71.09	75.05	jun.21	NS
Lesion score 100 days	149.32	223.72	9.00	0.001
Increase in lesion score –40 to 100	78.23	148.67	okt.59	0.001
Punch resistance sole –40 days (N)	avg.24	jul.41	0.46	0.01
Punch resistance sole100 days (N)	11.jun	okt.32	0.24	0.05
Punch resistance wl –40 days (N)	jun.25	maj.95	0.48	NS
Punch resistance wl 100 days (N)	jun.43	maj.69	0.24	0.01
Elastic modulus –40 days (N/mm2)	92.78	86.80	17.0	NS
Elastic modulus 100 days (N/mm2)	96.17	101.26	dec.73	NS

NS - not significant

Correlations between locomotion and lesion score, punch resistance and HUKI conformation score.

The elastic modulus of the tension test of the sole at day 100 postpartum was significantly ($P<0.01$) negatively correlated to locomotion score at day 154 postpartum ($R2 = - 0.61$). The number of days the heifers were lame throughout the lactation, corresponding to heifers with a locomotion score > 3, and the number of days animals were severely lame, corresponding to heifers with a locomotion score > 4, throughout the lactation were significantly ($P <0.01$) negatively correlated to the punch resistance of the sole horn at 100 days postpartum ($R2 = - 0.50$).

The HUKI score for rear legs was significantly ($P<0.05$) and positively correlated to the punch resistance of the sole and white line horn at 40 days prepartum ($R2 = 0.55$ and 0.50) and to the elastic modulus of the sole area at 50 days postpartum ($R2 = 0.53$). The HUKI score for feet was significantly ($P<0.01$) and positively correlated to the punch resistance and negatively correlated to lesion score of the white line area at 40 days prepartum ($R2 = - 0.50$) and to the number of days the animals were severely lame throughout the lactation, corresponding with a locomotion score > 4 ($R2 = - 0.50$). The HUKI locomotion score was significantly ($P<0.05$) and positively correlated to the punch resistance of the white line horn at 100 days postpartum ($R2 = 0.50$). The HUKI total score for legs and feet was significantly ($P<0.05$ to 0.01) negatively correlated to the punch resistance of the white line horn at 150 days postpartum ($R2 = - 0.50$) and the number of days the animals were severely lame throughout the lactation, corresponding with a locomotion score > 4 ($R2 = - 0.48$). The HUKI total final score was significantly ($P<0.05$ to 0.01) negatively correlated to the punch resistance of the white line horn at days 50 and 150 postpartum ($r = - 0.50$) and to the punch resistance of the sole horn at day 150 postpartum ($R2 = - 0.60$).

Discussion

Mechanical tests reflected the changes in housing and in haemorrhage levels that occurred between the prepartum and postpartum period. The correlation between the HUKI conformation traits and punch resistance and elastic modulus of the horn indicate that certain types of conformation may predispose the heifers to have a weaker hoof horn structure. Sole horn conformation is related to the pressure exerted by the weight distribution in the claws that is related to the conformation of feet and legs (Toussaint-Raven, 1985). Lower punch resistance and elastic modulus was found in heifers with straighter rear legs, lower foot angle, poor HUKI locomotion score, lower scores for the composite trait legs and feet and a higher final total score. Using a combination of scoring for conformation traits, mechanical tests and lesion scoring there is a potential for the selection of increased hoof horn quality at the beginning of the first lactation.

References

Offer, J.E.; McNulty, D.N. and Logue, D.N. (2000). Observations of lameness, hoof conformation and development of lesions in dairy cattle over four lactations. The Veterinary Record 147: 105-109.

Thomas,C; Leach, KA; Logue,DN; Ferris,C and Phipps,RH Management options to reduce load.

Metabolic stress in dairy cows. Occasional publication No 24, British Society of Animal Science, 1999.
Toussaint Raven, E. (1985). Cattle footcare and claw trimming. Farming Press Ltd., Suffolk, UK, pp.126.

the claw at the contact area is subjected and trimming promotes a more even distribution of the vertical force over both claws of one limb while walking.

THE FORCE AND PRESSURE DISTRIBUTION ON THE CLAWS OF CATTLE AND THE BIOMECHANICAL EFFECT OF PREVENTIVE TRIMMING.

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Introduction

Increased use of artificial housing in dairy cattle husbandry has led to higher prevalence of claw disorders. Claw disorders affect cattle behavior and reduce animal welfare. The body weight of the cow is applied to the four feet and distributed over the contact area of the claws with the floor. This result in pressure on the soles; while standing on a hard substrate contact areas can be reduced and pressure concentrations can be induced (van der Tol et al., 2002). These pressures reach relatively high values during locomotion on a flat hard surface; resulting horn damage may be one of the primary causes of claw disorders (van der Tol et al., 2003).
To prevent claw disorders, preventive trimming is often applied as a management routine, two or three times annually. In Western Europe the method used most is the so-called "Dutch method" described by Toussaint-Raven et al. (1985). In short, the goal of trimming is to promote natural loading of the claws by reshaping the claw capsule in three steps; (1) the toe length of the medial claw is cut to 7.5 cm and the sole is made flat, whereby the wall and sole are pared to the same level, (2) the contact area of the lateral claw is adjusted to the same height as the medial claw and (3) the axial walls are concavely reshaped. The current opinion in the modern dairy industry is that trimming two to three times a year to provide the cow with good balanced feet and a large contact area is the best option to prevent claw disorders. It has been shown empirically that claw disorders occur less frequently when the feet are trimmed and that the lameness-cases are less severe.
At the claw-floor interface forces due to standing and locomotion are applied to the floor and vice versa. The vertical ground reaction force is distributed over the contact area (pressures). In two experiments the forces and pressures at contact area were determined, (1) while standing before and after trimming and (2) while walking after trimming. The hypotheses were tested that trimming reduces the average and maximum pressures to which

Material and Method

Animals: The hind feet of the Holstein-Friesian cows were trimmed by an experienced trimmer according to the Dutch method.
Measuring apparatus: This system consisted of a pressure distribution plate and a force plate, sampled simultaneously (250 Hz). The pressure distribution plate (Footscan Scientific Version®, RSscan International, Olen, Belgium) has a 976 mm x 325 mm measuring surface, containing 8192 conductive pressure-sensitive polymer sensors. These sensors measure vertical force only. Since the surface of the sensors is known (0.39 cm2), the pressures can be determined automatically. This plate was embedded in an aluminum plate, which was solidly assembled on the force plate (Type Z4852/c, 600 mm x 900 mm, Kistler Corp, Winterthur, Switzerland). The assembly was placed level in a concrete pathway. The pathway and measuring apparatus were covered with a 6 mm thick rubber mat to provide enough frictional force to allow normal locomotion. The force platform measured the total vertical, longitudinal and transverse components of the ground reaction force (GRF). By means of the pressure plate a distinction between the vertical GRF applied to the lateral and medial claw could be made and the pressures exerted to the foot and contact area of the foot with the floor could be determined.
First experiment: The force and pressure distribution under the feet of five cows were measured before and after trimming. Second experiment: Five recently trimmed cows were repeatedly walked down the path, led by an experienced handler to measure a single step of each limb five times. To allow normal posture and unrestrained locomotion the path was covered with rubber mats (6 mm).
Before and after trimming, the following parameters were determined:

- The total vertical GRF (GRF-v) exerted on the lateral (GRF-lat) and medial claw (GRF-med).
- The contact area (CA) of the foot with the floor (cm2).
- The average pressure (P-av) by GRF-v/CA (N/cm2)
- The maximum pressure (P-max) exerted on the foot (N/cm2) During walking, the GRF was analysed:
- The total vertical GRF (GRF-v) exerted on the lateral (GRF-lat) and medial claw (GRF-med).

The pressure data was corrected for the amount of loading (weight) exerted on the limb and expressed in Newton per square centimeter per kg weight exerted on the limb (N/cm2/kg). To be able to compare GRF data between cows the stance time, during which the foot contacts the floor, was standardized to 100% and corrected for body weight. The GRF was expressed in Newton per kilogram bodyweight for each percent point of the stance time.

Results and discussion

First experiment: The vertical loading of the hind limb while standing was exerted for 20% to the medial and 80% to the lateral claw, after trimming the figures changed to 30% and 70% respectively. This indicates that the hind claws remained unbalanced after trimming. As postulated by Toussaint-Raven (1985) trimming increased the total contact area. Although the average pressure (corrected for the weight exerted by the limb) was significantly decreased, the maximum pressure was not significantly affected.

Second experiment: In the forelimbs the vertical GRF was more or less equally divided between the lateral and medial claw (Figure 2a). However, a remarkably different loading pattern occurred in the hind limbs. The major part (75%) of the vertical GRF is exerted on the lateral claw (Figure 2b); especially during the first 7% of the stance phase (heel strike) only the lateral claw is loaded. Consequently the lateral hind claw, which is completely subjected to the impact at heel strike, might be prone to damage during walking.

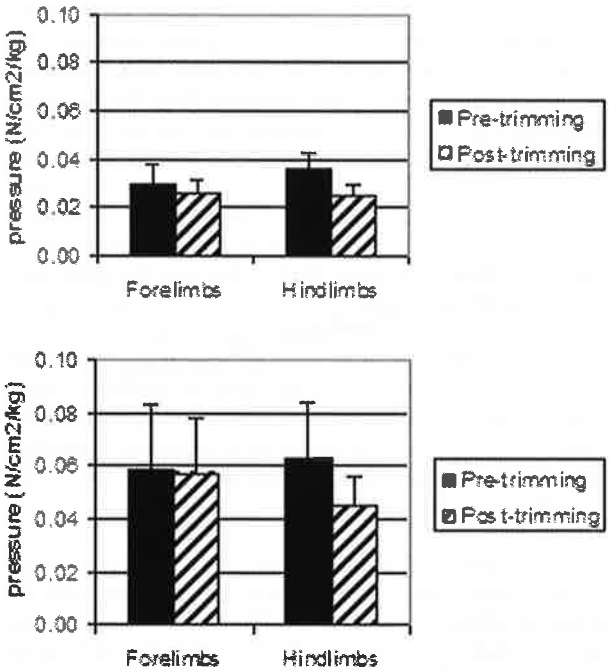

Figure 1: The average (a) and maximum (b) pressure during standing still, before and after trimming.
* Indicates significance at $P < 0.05$

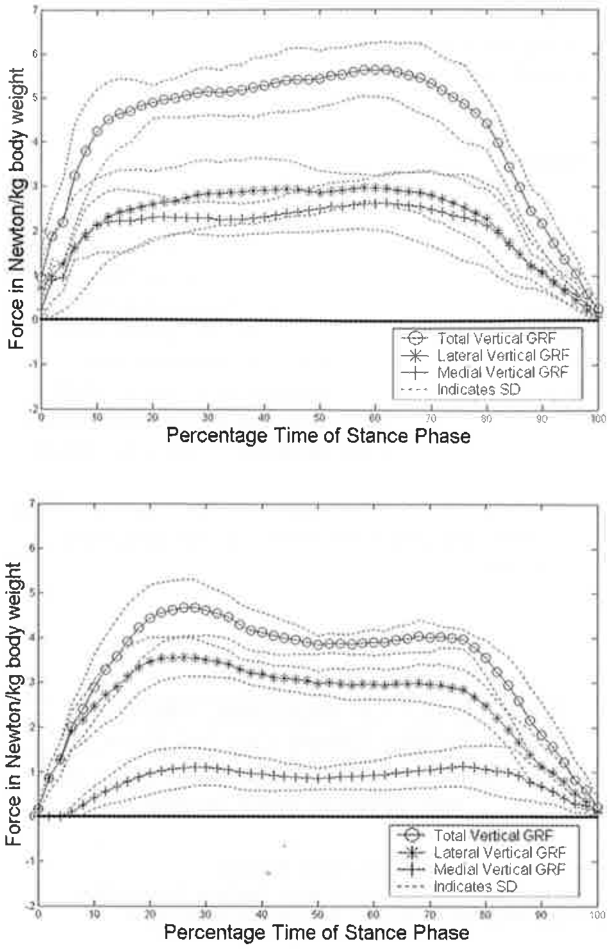

Figure 2: The distribution of the vertical force over the medial and lateral claws during the standing phase of walking for the right forelimb (A) and the right hind limb (B); values are expressed in Newton per kg bodyweight.

For standing as well as for walking, the results after trimming clearly indicate that the vertical loading of the hind limbs were still unbalanced. Trimming did not significantly affect the maximum pressures. Previous work has shown that after trimming these maximum pressures are located on the softer parts of the claw capsule (van der Tol et al., 2002). This might lead to haemorrhages, sole ulcers or overloading of the digital cushion. Moreover, the high strain rate at heel strike occurring on concrete floors might induce the horn fractures of the claws. On the other hand, due to this phenomenon the corium could be triggered to increase proliferation of horn-cells at the bulbs to provide better damping-characteristics, which might explain horn-overgrowth at the bulb of lateral hind claw. To prevent local pressure maxima an additional goal of preventive trimming is proposed. Force balance should not be the main focus of claw trimming. When a reduction of the maximum pressures can be achieved in such a way that the strongest parts of the claw capsule (i.e. the wall) will be subjected to the highest pressures, the claws might be less prone to claw disorders of mechanical origin.

To improve floor-systems where cattle are kept, it might be wise to better distribute the pressures on the claw-floor interface and to prevent the occurrence of too high strain rates by better damping-characteristics of the floor; rubber might be a good alternative.

References

Van der Tol, P.P.J., J.H.M. Metz, E.N. Noordhuizen-Stassen, W. Back, C.R. Braam and W.A. Weijs. 2003. The Vertical Ground Reaction Force and the Pressure Distribution on the Claws of Dairy Cows while Walking on a Flat Substrate. J. Dairy Sci. 86 (9): 2875-2883.
Van der Tol, P.P.J., J.H.M. Metz, E.N. Noordhuizen-Stassen, W. Back, C.R. Braam and W.A. Weijs. 2002. The pressure distribution under the bovine claw during square standing on a flat substrate. J. Dairy Sci. 85(6): 1476-1481.
Toussaint-Raven, E., R.T. Halstra, and D.J. Peterse. 1985. Cattle Foot care and Claw trimming. Farming Press. Ipswich. UK.

THE EFFECT OF MOISTURE, FREEZING AND SAMPLE SHAPE ON THE PUNCH RESISTANCE AND ELASTIC MODULUS OF THE BOVINE SOLE HORN

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Introduction

The morphology and mechanical properties of the equine and bovine hoof horn such as hardness, elastic modulus, bending stiffness and fracture toughness were described in detail in several studies (Kasapi and Gosline, 1996; Kasapi and Gosline, 1997; Collins et al., 1998; Hinterhofer et al, 1998; Baillie et al.; 2000). Hoof keratin is modelled as a fibre reinforced composite material consisting of microfibrils embedded in an amorphous, non-fibrous matrix, formed by globular proteins. The keratinised cells are further organised into either tubular structures or intertubular material, forming a macro scale composite (Kasapi and Gosline, 1997). Most tests however were performed on morbid samples. The aim of the experiment was to set up a punch test to be used on hoof slivers collected from live dairy cows. Factors that could have an influence over the test results, such as moisture of the samples, sample dimensions and storing conditions were tested. The moisture content of the hoof horn has been found to influence the mechanical properties of the hoof horn (Collins et al., 1998; Hinterhofer et al, 1998; Baillie et al.; 2000).

Materials and Methods

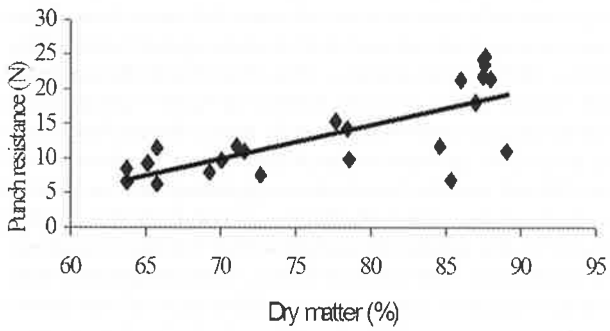
The hooves of six beef cattle, aged between 24 and 28 months, were obtained from an abattoir. On the same day of collection samples were taken from areas 5 and 2, corresponding to sole and white line horn areas, of the sole of each claw, according to the International Foot Map. They were kept in sealed plastic bags at room temperature until conditioning or analysis at the same day. The first batch of samples of all claws was kept in environments containing relative humidities (RH) of 11, 33, 58, 75 and 97 % for 7 days. Samples from each hoof were tested thereafter 12 times for punch resistance (PR) and elastic modulus (EM) on a TA.XT2i and TA.HDi Texture Analysers with a 25 and 100 kg load cells (Stable Micro-Systems). Hoof horn samples were analysed for punch resistance using a P2N needle probe and were hold in place between 2 metal plates that had a hole in their centre and were screwed together and to the base of the Texture Analyser. Metal plates had a thickness of 15 mm and the hole a diameter of 10 mm. The test-mode was to measure force in compression. Before and during the test the probe travelled at a speed of 1.0 mm/sec (Aranwela et al., 1999). When the probe reached the sample and the trigger force of 5 g was obtained, the force-displacement curve was recorded. Value of maximum force at punch (N) was obtained from the force-displacement curve. For the tension test the samples were shaped in a "dog bone shape" and had the following measurements in the centre area: 2 mm x 20 mm x thickness (0.05-0.3 mm). Specimens were held in place with self-tightening roller grips. The test-mode used was to measure force in tension. During the test the grips were moved apart at a speed of 1mm/sec until reaching the distance of 30 mm, when they returned to the start position. At that distance tests were conducted to failure, the grips were moved apart until the samples were split in two and the force displacement curve was recorded. Data from sole and white line areas were recorded separately. After the tests were completed the dry matter (DM) of the samples was determined.

A second batch of samples, with physiological moisture content, was tested at the same day to determine the effect of sample thickness on PR. The thickness varied between 0.05 and 0.3 mm. Further samples were tested for the effect of storage duration in plastic bags and of freezing on DM and mechanical properties. For the testing of the effect of storage 6 samples of each claw were placed in self-sealed plastic bags and stored at a temperature of 2 C. Samples were tested for punch resistance and elastic modulus at 0, 48, 96, 144 and 192 hours. For the testing of the effect of freezing samples were tested on the same day of collection (not frozen) followed by which the samples were stored in a freezer (-20 0C) in 3 separate plastic bags. The first bag was taken out of the freezer on the following day and left to defrost at room temperature for 4 hrs. Mechanical tests were completed. After being stored for one week and for one month, the second and third bags were taken out of the freezer and mechanical tests were completed. Effects of RH, DM and sample thickness were analysed by regression analysis.

The data from the time stored in plastic bags and freezing tests was analysed in ANOVA - GLM (Minitab 13.0) for the effect of treatment and means were compared using the Tukey test (95 % confidence interval).

Results

Increase in DM (%) resulted in a significant (P<0.01) linear increase in the PR (N) of the sole (PR = 0.4901 DM - 24.394, Rsq.= 0.54) and the white line horn (PR= 0.4301 DM - 24.874, Rsq.= 0.64) (Figure 1 and 2). With the increase in the dry matter the samples changed their behaviour from elastic to brittle, resulting in greater variation in punch resistance data. The EM (N/mm2) of the sole horn was significantly (P<0.01) positively and exponentially related to the DM (EM= 0.0602e0.1012x, Rsq.= 0.81). DM varied from 63.7 to 89.1%, PR of the sole horn from 6.24 to 24.66N, PR of the white line horn from 2.17 to 18.60N and EM from 85.5 to 751.9N/mm2. The days (1 to 8) taken to analyse the samples (Table 1) and freezing for up to 30 days (Table 2) had no significant effect on the DM and PR of the sole and white line horn. There was a significant (P<0.01) increase in the EM of the sole horn when samples were frozen for 30 days (Table 2). PR increased in a positive significant (P<0.001) linear way in relation to the thickness (mm) of the area tested (PR= 6.679 + 34.531 thickness, Rsq.= 0.66).



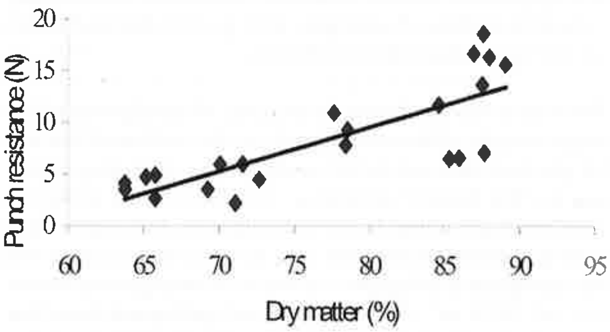


Table 1 Effect of days to analysis on the dry matter (DM), punch resistance of the sole horn (PR sole) and the white line horn (PR wl) and elastic modulus of the sole horn (Emod sole) and white line horn (Emod wl)

Days to analysis	0	2	4	6	8	sem	sig.
DM (%)	72.54	72.05	72.88	71.25	71.11	0.568	NS
PR sole (N)	avg.78	okt.40	okt.13	sep.81	sep.61	0.762	NS
PR wl (N)	apr.95	jun.40	6.avg	jun.51	jun.90	0.662	NS
E mod sole (N/mm2)	158.2	148.1	226.3	125.8	175.5	43.36	NS
E mod wl (N/mm2)	91.8	159.5	218.9	143.3	194.1	37.27	NS

NS - not significant

Table 2 Effect of days frozen on the dry matter (DM) and punch resistance (PR) of the sole and white line (wl) horn and elastic modulus of the sole (Emod sole) and white line horn (Emod wl)

Days	0	1	7	30	sem	P
DM (%)	72.34	-	-	74.22	0.779	NS
PR sole (N)	avg.34	avg.72	sep.86	9.okt	0.660	NS
Pres wl (N)	jun.21	jun.63	jul.44	7.okt	0.701	NS
Emod sole (N/mm2)	144.4 a	268.3 ab	251.1 ab	403.1 b	42.53	***
Emod wl (N/mm2)	116.6	136.5	292.7	330.7	67.21	NS

NS - not significant, a,b - different letters indicate values that are statistically different

Discussion

Punch resistance and elastic modulus were affected by the moisture content of the hoof horn in a similar way as hardness, bending stiffness and fracture toughness (Collins et al., 1998; Hinterhofer et al., 1998; Baillie et al.; 2000). Hinterhofer et al. (1998) pointed out the importance of testing the samples at physiological moisture levels to represent the in vivo situation. In the short term, moisture loss is the factor that is likely to have the greatest effect on the mechanical properties of hoof horn samples. The prevention of moisture loss was thoroughly described by Collins et al. (1998) and Hinterhofer et al. (1998) and methods of prevention described in their methodology. Collins et al. (1998) wrapped the samples in 3 layers of Para film and stored them at 40C and Hinterhofer et al. (1998) kept the samples in resealable plastic bags at 40C. In this study, keeping the samples in plastic bags and at 40C for up to 8 days did not result in significant changes of the moisture content of the samples. As a consequence no significant alteration of the punch resistance and elastic modulus of the samples was found. Thickness should be included as a covariant in the analysis of PR tests.

References

Aranwela, N.; Sanson, G. and Read, J. (1999). Methods of assessing leaf-fracture properties. New Phytologist 144: 369-393.

Baillie, C.; Southam, C.; Buxton, A. and Pavan, P. (2000). Structure and properties of the bovine hoof horn. Advanced Composite Letters 9:101-113.

Budras, K.D. and Mulling, Ch. (1998). Structure and function of the bovine claw. Proceedings of the Tenth International Symposium on Lameness in Ruminants, 7 - 10 Sept. 1998, Casino Lucerne, Switzerland, pp.189-191.

Collins, S.N., Cope, B.C., Hopegood, L., Latham, R.J., Linford, R.G. and Reilly, J.D. (1998). Stiffness as a function of moisture content in natural materials: Characterisation of hoof horn samples. Journal of Materials Science 33: 5185-5191.

Hinterhofer, C., Stanek, Ch. And Binder, K. (1998). Elastic modulus of equine hoof horn, tested in wall samples, sole samples and frog samples at varying levels of moisture. Berliner und Munchener Tierarztliche Wochenschrift 111 : 217-221.

Kasapi, M.A. and Gosline, J.M. (1996). Strain-rate dependent mechanical properties of the equine hoof wall. The Journal of Experimental Biology 199: 1133-1146.

Kasapi, M.A. and Gosline, J.M. (1997). Design complexity an fracture control in the equine hoof wall. The Journal of Experimental Biology 200: 1639-1659.

THE WEIGHT-BEARING APPARATUS OF THE BOVINE CLAW

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Introduction

Under modern production conditions, claw diseases have a in high incidence and are a severe problem in cattle practice. Therefore, the investigation of structure, form and function of the bovine claw as a prerequisite for understanding the pathogenesis of common claw diseases has gained great importance, but recent publications focus mainly on the cornifying epidermal layers of the claw. Nevertheless, the dermo-subcutaneous system also plays an important - but often neglected - key role in the normal and pathologically altered function of the claw.

The aim of this study was a detailed investigation of the dermal and subcutaneous structures of the claw in order to detect structural modifications or functional adaptations. Special emphasis was laid on demonstrating the three-dimensional architecture of the connective tissue fibre system of the claw.

Material and methods

The suspensory apparatus of the distal phalanx in the entire circumference of the bovine claw was investigated employing light and electron microscopy as well as histochemical and morphometrical techniques. Therefore, 26 sound claws of adult bovine fore and hind limbs were obtained from a local abattoir. Samples were taken from proximal, middle and the distal regions of the wall segment in the circumference of the claw, as well as from the solear and bulbar segment. Additionally, whole hind claws were inserted into a special loading device, and the resulting deformities of the different regions of the claw were measured and documented.

Results and discussion

The suspensory apparatus is a complex system of different functional regions, reaching from the surface of the distal phalanx to the outer coronary epidermal horn layers. It can be subdivided into a four-zonal chondro-apophyseal fibre insertion area at the phalangeal surface, a specifically orientated dermal system of directional tense fibres, a fibre- insertion area at the dermo-epidermal border line and last but not least a locking of forces with the specific epidermal structures.

The four-zonal insertion area represents the connective tissue complex of the suspensory apparatus, which is mainly dermal and consists of a lamellar bone zone, a zone of calcified and uncalcified fibrous cartilage and a zone of parallel-orientated dense fibres. The four zones of the chondro-apophyseal insertion area facilitate an elastic-resilient coupling to the distal phalanx and thus allow a smooth transition between fibrous connective tissue and osseous material properties. The surface of the distal phalanx features proximo-distally orientated ridges that serve as an anchorage area for the chondro-apophyseal insertion zone; whereas between these ridges periosteum is developed that enables growth and adaptation of the distal phalanx. The zone of parallel-orientated dense fibres consists mainly of type I collagenous fibres, elastic and reticular fibres and is flanked by loosely arranged connective tissue containing numerous cells and blood vessels. It acts as a supporting and anchoring system of the distal phalanx and conducts the tension forces to the epidermal component of the claw suspensory apparatus. The zone of loose, well-vascularised connective tissue is an equally modified part of the dermis and provides a nutritional supply to both the claw suspensory apparatus and the avascular epidermis. Additionally, it permits a close messenger- and growth-factor-mediated dermo-epidermal co-ordination.

The tension forces borne by bundles of collagenous fibre originating in different angles from the surface of the distal phalanx are indirectly transferred to the horny lamellae via the basal membrane, and from there on to the coronary horn of the horny claw capsule. The mechanical load is distributed onto the very large surface area of the dermo-epidermal borderline which is enlarged by formation of 1800 to 1200 dermal and epidermal lamellae, respectively. The latter applies to in the hind limb, whereas in the fore limb only about 1600 to 1800 lamellae are formed in the distal part of the wall segment. The lamellary, cap and terminal epidermis thus represent the epidermal compound of the claw suspensory apparatus which stabilises the anchorage via specialised intra- and inter-cellular elements and permits an elastic-resilient transfer of tension forces.

At the tip of the claw, the diverging dense fibre system originating from the surface of the distal phalanx inserts not only on the dermo-epidermal interface of the distal cap and terminal papillae of the wall segment, and the papillae of the solear segment, but is also aligned with the three-dimensional interconnecting dermal fibre system of the solear segment. This creakes a complex and highly derived functional junction between the parietal and the ground surface-contacting components of the claw.

Functional implications:

Considering all specified modifications of the wall segment the functional conclusion is that the claw suspensory apparatus is a highly specialised anchoring system that accomplishes the main part of force transformation in the dorsal and in the apical third of the abaxial parietal area of the claw. The distal phalanx is lowered under load, and the traction caused by the claw suspensory apparatus in the inner coronary horn thus induces the claw mechanism. The claw mechanism permits a the reversible alteration in shape of the claw capsule and the structures encased within, caused by loading and unloading of the claw. A proximo-distal increased, and dorso-plantarl decreased, force gradient is detectable.

The body weight-generated pressure forces are transferred to the distal phalanx via the skeletal system. In the first phase of weight-bearing, the pressure force is transduced into motion by elements of the bulbar and solear segment, while it is transformed into a tension force by the claw suspensory apparatus in the second phase. The different load capacities and the resulting structural and functional modifications in the respective segments of the claw, can be considered together as the weight-bearing apparatus of the claw. The weight-bearing apparatus consists of a shock-absorbing apparatus on the one hand, i.e., the anti-shock pad and pressure buffer of the bulbar segment (Räber, 2000) and the respective elements of the solear segment, and the suspensory apparatus of the distal phalanx on the other hand, i.e., the tension force transforming areas. The claw suspensory apparatus in a narrower sense consists of the wall segment and the sustaining elements of the solear segment. For suspension of the distal phalanx, the extensor tendon, the deep flexor tendon and the whole ligamentousapparatus of the ungular joint are equally essential, therefore these form the claw suspensory apparatus in a broader sense. The solear segment comprises both tension- transforming as well as pressure-absorbing elements and is therefore defined as the transfer or transitional region. The suspensory and the shock-absorbing apparatus of the distal phalanx are highly specialised epidermal and dermo-subcutaneous systems, both accomodating the weight-bearing induced strains.

Clinical implications:

Additional measurements of the loaded and unloaded claw revealed the natural loading circumstances and the relative load capacity of the claw, resulting in the detection of particular loci minores resistentiae. Regions with fibro-cartilaginous insertions such as the wall and solear segment, and insertion areas of ligaments and tendons, are exposed to multidirectional forces while they are not protected by a subcutaneous cushion. Therefore, these regions deserve major attention in improperly loaded claws. Regions displaying a subcutaneous cushion, like the coronary and bulbar segment, participate in the load-induced strain but are accordingly protected.

The claw suspensory apparatus is a highly derived anchoring system in which the dermis plays the most important role and is therefore most sensitive. The avascular epidermis is completely dependant on nutritional supply via the dermal blood vessels. Therefore, any microcirculatory alterations within the dermis must affect the living epidermal cells. Thus, even latent malnutrition of the basal living epidermal cells causes pathological alterations in epidermal horn production and horn quality. This interdependance and close regulation of the dermo-epidermal system explains why any alteration within the highly derived pododerma will lead to appropriat changes within the remaining components of the system.

References

Räber, ME (2000): Das Ballenpolster beim Rind. Ein Beitrag zur funktionellen Anatomie der Klaue. Doctoral dissertation. Universität Zürich, Veterinär-Chirurgische Klinik

PODODERMAL ANGIOGENESIS - NEW ASPECTS OF DEVELOPMENT AND FUNCTION OF THE BOVINE CLAW

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Introduction

The pododermal microvascularisation has been postulated to play a key role in both the physiological function of the bovine claw and in the pathogenesis of claw diseases, particularly of the laminitis-associated claw diseases. However, studies dealing with the pododermal vasculature on cellular or molecular level are scarce, and results from research on the pathogenesis of equine laminitis or structure and function of the equine hoof, respectively, were often transferred uncritically to the bovine claw without taking into account the differences in stance and weight-bearing between both species. Systematic studies on the weight-bearing system of the claw (Räber, 2000; Westerfeld, 2003) and on the bovine pododermal angioarchitecture (Vermunt and Leach, 1992; Hirschberg