

Digital dermatitis - research and control

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Lameness in dairy cattle is an important issue and research has indicated that digital dermatitis is one of its most important causes. Fortunately, this is an infectious condition that can be controlled.

Introduction

Digital dermatitis (DD) is a bacterial infection of the epidermal skin of the heel. The condition was first reported in 1972 by Carlos Mortellaro in Italy. Since then, the disease has spread across Europe and into the Netherlands, where it caused a good deal of problems in the late '70s and early '80s. The first case was reported in the UK in 1985, and since then it has become widespread throughout the country. In an intervention study (Hedges *et al.*, 2001) involving five herds and a total of 1,109 cow years, and where each one of over 700 cases of lameness was examined and recorded by a vet, the incidence of DD was around 12 cases per 100 cows per annum and ranked equal to sole ulcers and white line defects as a cause of lameness (Figure 1). 'Foul' was in fourth place at seven cases per 100 cows per annum.

An infectious disease

An interesting feature of DD is that it can be controlled. DD is an infectious disease with many similarities to mastitis, and control would be considerably improved if we kept this in mind. With both conditions, it is important to remember that:

- animals with lesions are infectious and act as a reservoir of infection for others;

- the subclinical carriers need to be recognised and dealt with;
- attention to environment is important;
- regular disinfection of the affected body part (twice daily for mastitis) is required for control.

The prevalence and severity of DD is significantly affected by the immunosuppression associated with calving and by factors that lead to increased standing and increased exposure to slurry in the post partum period. This is why lameness from DD increases after calving, with peak levels occurring one to four months post-calving. Heifers are often especially badly affected because they spend a long time standing, waiting to be milked and waiting to feed.

In the past, too much effort has been aimed at treatment and we need to focus much more on prevention. Treatment alone will not control the disease just as treatment of mastitis alone will not effectively control mastitis. The best form of digital dermatitis control is therefore environmental management plus a proactive footbath beginning in early lactation. Foot bathing needs to be regularly repeated to be effective, with the frequency depending on the level of environmental challenge. Cows' teats are disinfected twice daily. We should be doing the

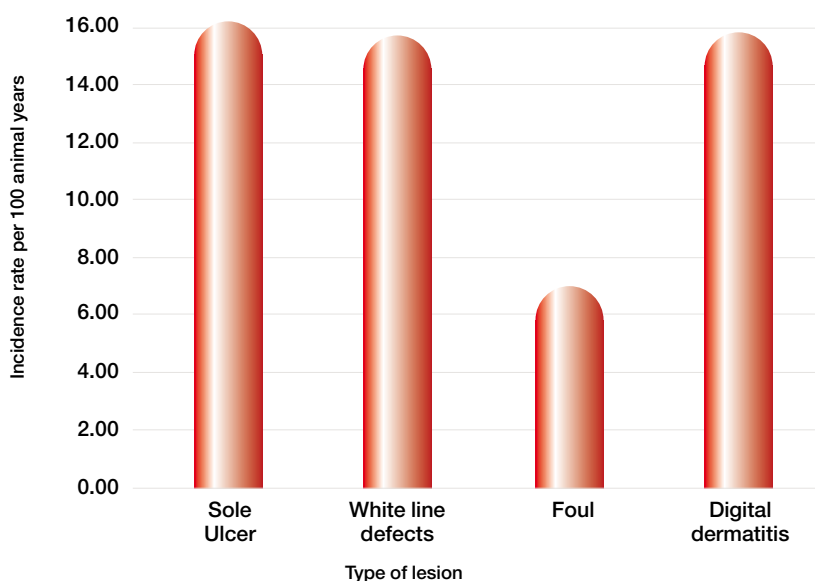


Figure 1: Incidence rate of four most common causes of lameness (Hedges *et al.*, 2001).



Figure 2: A typical digital dermatitis lesion.



Figure 3: A hairy wart is the chronic proliferative form of digital dermatitis.

same for cows' feet if we are serious about the control of infectious foot disease. We would not contemplate attempting to control mastitis by teat dipping once a month, so it is surely illogical to think that we can control DD by foot bathing once a month.

DD lesions

Typically, DD is seen as a red, raw area of epidermitis, surprisingly painful to the touch (Figure 2) and with a characteristic foul odour. Lesions are most commonly sited on the skin just between the bulbs of the heels and the reservoir of infection is thought to persist in the interdigital pouch, which is the space between the claws towards the heel. Lesions may also be seen at other sites such as the skin of the interdigital cleft, at the front of the interdigital cleft, under-running the sole or even on a sole ulcer. If lesions occur on the anterior coronary band they can totally disrupt horn formation and lead to a vertical fissure. Many cows with vertical fissures never recover because an osteitis develops on the underlying pedal bone, and the excess new bone formed forces the fissure apart. The North American 'hairy wart' form of DD (Figure 3) is becoming increasingly common in the UK. This occurs in chronic, longstanding infections because epidermal proliferation is a common response to the causative organism. Using an immunoperoxidase procedure and monoclonal antibodies, UK work has recently identified the presence of the specific DD organism in necrotic anterior udder lesions (Blowey

and Read, in press) and in lesions of interdigital dermatitis (Evans *et al.*, in press), i.e. there is increasing evidence that DD and interdigital dermatitis are the same condition.

Aetiology

First reported in the UK in 1988 (Blowey and Sharp, 1988), DD is a bacterial infection caused by a spirochaete, most probably a treponeme (Read *et al.*, 1992; Demirkan *et al.*, 1998). Initial studies suggested that *Borrelia burgdorferi*, the cause of Lyme disease, might be implicated, and although calves seroconvert at the same time as lesions appear, the correlation is by no means specific (Demirkan *et al.*, 1999). The two spirochaetes most likely to be implicated are the treponemes designated 2-1498 and 1-9185MED by Walker *et al.*, (1995). The Liverpool group has found a close serological association with UK DD lesions and these two organisms.

A strong association exists between *Fusobacterium necrophorum* and treponemes in human oral infections, and it may well be that a synergy exists in the pathogenesis of the disease. Damp conditions are important for the proliferation of the organism. Experimental transmission of DD has only been achieved by soaking the skin of a calf with water daily for eight days and then applying fresh DD exudate onto scarified skin using gauze (Read, unpublished data). Lesions of DD developed within 14 days, whereas no DD developed on dry skin even after 90 days. This shows the extreme importance of wet environmental conditions in the development of the disease.

Epidemiology

Work by Laven (1999) and others at ADAS, Bridgetts Research Centre (Winchester, UK), where 1,810 examinations were performed on 328 cows in different housing systems and at different stages of lactation, showed that approximately 41% of cows had lesions of varying severity and, of these cows, 66% had lesions in both hind feet. As considerably more than half of the cows with DD had lesions in both hind feet, it suggests that immunity is of limited importance in the pathogenesis of the disease, otherwise infection in one foot might prevent infection from becoming established in the second foot. An alternative hypothesis is that cows that spend longer standing (e.g., lower ranking animals waiting to feed) have a greater exposure to slurry and are more likely to develop DD. Invariably, there is an increase in lesions in the first four to six weeks after calving. This may be due to the marked immune suppression that occurs around the time of calving, and animals exposed to DD before calving were much more likely to develop the disease after calving. Conversely, a proportion of cows never develop DD, even in the automatic scraper group which shows higher disease prevalence (see below). This suggests that either some cows have an innate immunity, or that the conformation of their foot and/or their behavioural pattern is such that they were not exposed to the same level of contamination.



Figure 4. Use enough bedding such that some falls into the cubicle passage.

In a comparison of housing systems (Laven 1999), cows in cubicles had 1.6 times more lesions than cows housed in straw yards and, even when lesions did occur, straw yard cows had much less severe lesions. In addition, cows in cubicles with automatic scrapers had 1.2 times more lesions than cows in cubicles of the same design but without automatic scrapers. It has been suggested that this occurs because:

- the scrapers pull a 'wave' of slurry through the building and this increases the contamination of cows' legs;
- the corrosive effect of the mixture of urine and faeces has a particularly important role in the pathogenesis of the disease;
- automatic scrapers remove the layer of bedding that falls off the cubicle step that would otherwise reduce the level of moisture in the passage.

It may, therefore, be better to either scrape by tractor, or to run the automatic scraper when the cows are out of the building, e.g. for milking. As there is a lower incidence of DD in straw yards, an additional preventive measure would be to house freshly-calved heifers in straw yards for the critical first two to four weeks after calving. When

bedding cubicles, there should be enough bedding such that some falls off the cubicle and into the passage (Figure 4). This then acts as a 'cushion' as the cow reverses out of the cubicle, and for those cows that stand with their front feet in and their hind feet in the passage, the bedding acts as a shock absorber, it keeps the feet drier, and it reduces the level of exposure to slurry. Levels of bedding around 2kg/cow per day of straw or sawdust are ideal.

The influence of calving

Laven (1999) showed that the prevalence of DD lesions pre-calving had a big influence on the incidence of lameness due to DD in early lactation. Heifers were reared in either cubicles or straw yards up to just before calving and, as one would expect, it was the cubicle-reared heifers with the greatest exposure to slurry that had the highest prevalence of DD lesions. Most of these were very mild skin lesions, and few of the pre-calving heifers showed any significant lameness. However, after calving, these very mild lesions deteriorated considerably and many of the first lactation heifers became lame and needed treatment.

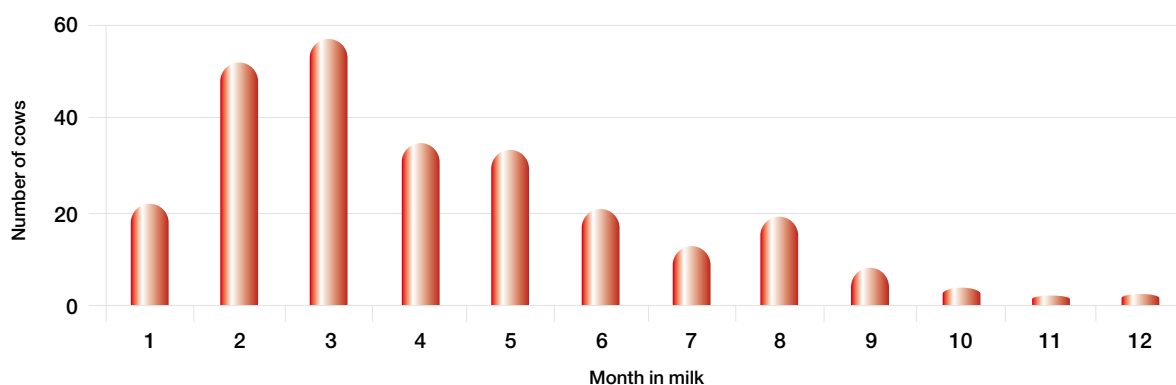


Figure 5: Number of cattle lame by month of lactation (Blowey *et al.*, 2004).



Figure 6: Foot baths with ridges are uncomfortable for cows to walk on.

Similarly, Blowey *et al.*, (2004), in a biotin intervention study involving 1,109 cow years on trial in which over 700 cases of lameness were recorded and identified, showed a marked increase in the proportion of the infectious causes of lameness, DD and foul, in the first month after calving (Figure 5). This increase in the infectious causes of lameness is likely to be associated with two factors, namely immune suppression and excess standing (and therefore exposure to slurry).

Treatment and control

As with mastitis, we must not get confused between treatment and control, although clearly the treatment of individual lesions is an important part of control, because it reduces the rate of shedding of the organism into the environment. Individual cases are best treated by thorough cleaning of the lesion followed by topical application of antibiotic or disinfectant held in place by a dressing. Use of a mastitis tube on a well cleaned lesion works well, possibly because intramammary antibiotics are specifically formulated to penetrate tissue. Parenteral therapy may be indicated for advanced anterior lesions.

Prevention

Prevention is based on keeping the housing and general environment as clean and dry as possible and by whole herd footbaths through solutions of disinfectant or antibiotic. As discussed above, dry cows can have mild lesions of DD and, with the marked immune suppression associated with calving, these proliferate to produce lameness four to eight weeks into lactation. The best form of DD control is therefore a proactive footbath, preferably starting with cows in the transition group or at least in early lactation, to prevent these mild lesions from developing. The frequency of bathing will vary with the severity of the challenge (e.g., the environmental conditions) but increasing numbers of farms now footbath the whole herd once daily for seven days a week. Alternative routines include seven days on then seven days off, or five days a week every week, using a freshly prepared footbath once daily.

The previous reactive approach, namely only foot bathing when lesions had developed sufficiently severely to produce lameness, was clearly illogical, because this allowed lesions to develop to such an extent that treatment was almost impossible. We would not use this approach with mastitis, so why do we suggest it for DD? A significant prevalence of raw 'open' DD lesions in a herd must act as a source of infection for other cows in the herd in the same way that a *Staphylococcus aureus* carrier is a major risk to other cows. Cows with raw open lesions of DD should be treated as a matter of urgency to reduce the spread of infection. This is often not done.

Common disinfectants include 4% formalin, copper salts, quaternary ammonium, organic acids, 'Virkon S' (Antec Ltd) and proprietary products such as Delaval 'Double Action', although it is likely that any disinfectant applied regularly would be effective. If the challenge is mild, then use of circulation cleaner (which usually runs to waste at the end of the wash cycle) works well, and several farmers now have this piped direct into their foot baths. It may be better to use a pre-wash with circulation cleaner followed by the disinfectant. If formalin baths are used

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they should not be too deep (otherwise the skin of the lower leg may blister), they should not be used near to the milking parlour, and they should not be used where there are raw open lesions of DD present, as this will be too painful. However, if cows are bathed once daily for two to four weeks in succession, even the most chronic dermatitis lesions regress, and the disease will be kept well under control. Frequent use of formalin will also control interdigital skin hyperplasia, foul and slurry heel. There is an opinion that repeated use of formalin makes the hooves too hard. I do not believe this. If formalin hardens the hoof then this can only be to the benefit of the cow, and we now have several herds in the practice that have foot bathed, like this, daily for one or two years with no adverse effect.

Practical control measures.

The following is a summary of the main points of control:

Foot bathing

- Ensure the bath is at least one 'parlour row' of cows away from the parlour, so that foot bathing does not disrupt milking and no formalin smell is detectable in the parlour.
- Do not use baths with ridged floors (Figure 6), designed to open the claws – cows hate standing on them!
- When you start foot bathing, put straw in the bath for the first week or two as this encourages cows to pass.
- Foot bath regularly with disinfectants – a single passage per week will not work. Once daily is ideal and, if carried out effectively in the majority of herds, will totally suppress DD and almost totally eliminate 'foul' and interdigital skin hyperplasia lesions.
- Double foot baths help with cow flow, because if one cow is reluctant to enter but another cow passes her, this encourages the reluctant cow to pass.
- Ideally start bathing the transition cows and continue regularly into lactation.
- Fully automated baths are now available (Delaval Ltd) that empty and fill with the correct strength of solution each day.

Environmental hygiene

- Scrape passages regularly, but avoid the use of automatic scrapers.
- Provide enough cubicle bedding so that some falls into the gutter to keep feet clean and dry.
- Ensure buildings are well drained and ventilated to remove water.
- Repair pitted concrete where slurry pools.
- Minimise standing times, as this predisposes to increased exposure to slurry.
- Avoid high stocking densities. Aim for a maximum of 90% cubicle occupancy and ideally cubicle passages should be 10ft wide and cows should have 2ft per cow of feed space. Less than this could lead to excess standing, predisposing to DD.

Remember, cows' teats are disinfected regularly. We should be doing the same for cows' feet if we are serious about control of infectious disease.

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