# Clinical Perspectives of Digital Dermatitis in Dairy and Beef Cattle

Paul J. Plummer, DVM, PhD*, Adam Krull, DVM, PhD

**KEYWORDS**

• Digital dermatitis • Treponema • Bovine • Lameness

**KEY POINTS**

- Digital dermatitis (DD) is a common disease process of the skin of both dairy and beef cattle.
- Advanced lesions are associated with clinical lameness, whereas early lesions cause local skin disease with minimal lameness.
- Topical treatment with oxytetracycline is the common therapy for advanced lesions but has a high rate of recrudescence.
- An integrated management plan that relies on a combination of topical treatment of advanced lesions coupled with footbathing to control progression of earlier lesions is the most effective strategy.

## INTRODUCTION

### *Description of Digital Dermatitis*

The first article to describe the macroscopic appearance of a large number of DD lesions was done on 10 California dairies by Read and Walker in 1998.[1] A majority of the lesions were circumscribed, erosive to papillomatous, and surrounded by a ridge of hyperkeratotic skin bearing hypertrophied hairs. These lesions were typically circular to oval, raised above the surrounding skin, and 2 cm to 6 cm in diameter. Lesions were more likely to involve the rear legs (82%) and a majority (83%) were located on the proximal border of the interdigital space. The macroscopic differences in DD lesion morphology have been described with several novel scoring systems primarily used in research settings.[2–4] The "M" scoring system and the Iowa DD scoring system both describe the macroscopic changes that take place between a normal bovine foot and an end-stage DD lesion. Although each system describes lesions slightly differently, both describe lesions in preclinical and clinical states, with lameness

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Veterinary Diagnostic and Production Animal Medicine, Iowa State University College of Veterinary Medicine, Ames, IA USA
* Corresponding author.
*E-mail address:* pplummer@iastate.edu

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only associated with certain stages. It has also been shown that there can be dynamic macroscopic changes between these stages in as few as 7 days.[5,6]

The histopathologic changes associated with DD have been described in numerous publications,[4,7–17] with several of these studies summarizing the histopathologic changes associated with a large set of DD lesions.[4,11] DD lesions were described as having a highly proliferative epidermis, pronounced rete ridge formation, hyperplastic stratum corneum, and acanthotic stratum spinosum. Additional descriptions include lesions having zones of acute degeneration, necrosis, and focal thinning of the stratum corneum with inflammatory cell infiltration. A consistent finding is the microscopic observation of spirochetes within the lesions through the use of silver staining.

### *Pathophysiology and Etiology*

Bovine DD was first morphologically described in 1974 at the 8th International Meeting on Diseases of Cattle in Milan, Italy,[18] but despite more than 40 years of research, the fulfillment of Koch's postulates[19] in identification of an etiologic agent has yet to be achieved. The first report of a spirochete-like, filamentous organism within DD lesions was described by Blowey and Sharp in 1988.[16] It was soon found that these organisms belong to the species *Treponema* and that became the first bacterial species cultivated and implicated in the etiology of bovine DD.[20] Even from the original report, which described 2 unique bacterial morphologies that belonged to the *Treponema* spp, the identification of multiple *Treponema* spp through visual, biochemical, immunologic, and molecular techniques has been a consistent finding.

*Treponema* spp have been implicated as the causative agent in DD due to their identification in DD lesions by cultivation,[21–23] fluorescence in situ hybridization (FISH),[8,22,24–30] polymerase chain reaction (PCR),[21,31–33] and metagenomics.[3,25,34,35] The nomenclature for the different types of *Treponema* spp has been constantly undergoing changes based on many of the phylotypes having yet to be cultivated. At this point, there are 4 clusters—cluster 1 (*T denticola*/*T pedis*–like), cluster 2 (*T phagedenis*–like), cluster 3 (*T refringens*–like), and cluster 4 (*T medium*/*T vincentii*–like) — that have been reported in the majority of the literature as having clinical relevance to DD.[26] Studies of DD-associated *Treponema* spp have also identified them as having the ability to cause disease by impairing the innate immune and wound repair functions of bovine macrophages.[36] Multiple immunologic studies have also found an increase in antibodies to *Treponema* spp in herds and individual cows with DD.[37,38] Despite all the evidence for *Treponema* spp as the causative agent for DD, Koch's postulates have yet to be fulfilled. Attempts to induce DD lesions with pure cultures of *Treponema* spp have largely failed to consistently induce disease with the characteristic size and severity of naturally occurring DD lesions.[39,40] Additionally, vaccinations against DD-associated *Treponema* spp have failed to decrease the incidence or severity of disease.[41] There is not enough evidence currently available to differentiate *Treponema* spp from a causative organism or merely an organism associated with clinical DD lesions.

For these reasons, numerous other organisms have been studied to determine each one's significance in causing disease. Various *Campylobacter* species as well as *Dichelobacter nodosus* have been cultured from DD lesions and from normal bovine skin.[24] Several researchers[26] have used FISH to determine the level of tissue invasion of various potential pathogens. *D nodosus* was found in 27% and 51% of DD lesions and *Fusobacterium necrophorum* was identified in DD lesions but was found to have minimal invasion in any of the DD tissues evaluated. PCR detection using species-specific primers found *D nodosus* in 100% of DD lesions but also in 60% of normal