

# Papillomatous Digital Dermatitis (Footwarts) of Cattle: Research Review and Update

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## Introduction

Because papillomatous digital dermatitis (footwarts) of cattle is an economically important, emerging and contagious disease of undefined cause, the University of California School of Veterinary Medicine established a multidisciplinary taskforce in 1993 to investigate the following:

- Response to various therapies (Treatment and Control Studies)
- Prevalence and predisposing risk factors (Epidemiologic Studies)
- Microbiologic causative factors (Etiologic Studies)
- Transmissibility, pathology and vaccine studies (Experimental Studies)

## Review and update

Over the past seven years, considerable progress has been made. In 1992, we found that footwart lesions were not true viral warts, but wart-like growths infected with bacteria. (1) This led to treatment and control studies, which found that lesions disappeared within a few days after antibiotic treatment. This was a significant breakthrough because it demonstrated for the first time that bacteria played a role in the genesis of the disease, and it led to inexpensive, effective treatments rather than culling affected cows. On-farm treatment trials followed from 1994 to 1997. (2, 3, 4, 5) These trials documented that antibiotics were effective and safe in the form of topical wraps or as in-parlor foot sprays, even in large dairies. However, the trials also found recurrence of lesions in about 60 percent of clinically cured cows. (2, 6) More research is needed in order to elucidate the reasons for recurrence.

In 1993, our epidemiologic studies revealed several important findings: 1) the prevalence of footwarts increased epidemically (8-fold) in California from 1991 to 1993, 2) herd prevalences in southern and central regions were very high (approximately 75 percent) and, 3) within-herd prevalences averaged 12 percent and ranged from 1–99 percent statewide. (7) In 1994, we demonstrated that the odds of having a high prevalence of disease was 20 times greater if the corrals had deep slurry and four times greater if replacement heifers were brought in. (8) (We later used this *slurry-factor* discovery to advantage by demonstrating that experimental transmission was highly repeatable only if the feet were exposed to constant moisture and reduced access to air.) This proved that footwarts develop as a result of multiple environmental and infectious factors, and that these factors need to be understood in order to develop

rational control programs. Building on this knowledge, we discovered that during a 15-month study on three affected dairies, 50 percent of cows never got the disease and 10 percent got it repeatedly. (9) More research on the influence of foot conformation on the prevalence of footwarts is needed because it will impact selection and culling practices.

In 1992, our etiologic studies found several types of unusual bacteria consistently within footwart lesions. This was significant because it meant that the infectious part of the disease was not simple, (as it would be if a single organism was involved) but complex, similar to other infectious foot diseases like footrot. We then found that spirochetes not only dominated the other bacteria, but also were truly invasive (1); that is, they invaded living tissue, not merely colonizing the outer layers of non-living skin. Therefore, we focused our efforts on the spirochetes and successfully cultured and characterized them into three distinct groups (1993–98). (10,11) We identified them all as *Treponema* spp. by cultural, biochemical and electron microscopic characteristics and by DNA *fingerprinting* techniques. Group 1 most closely resembled *Treponema denticola*, group 2 most closely resembled *Treponema phagedenis* and group 3 most closely resembled *Treponema medium/vincentii*. The isolation of the treponemes was another breakthrough because it allowed us to perform specific immunologic studies that confirmed that they were truly infectious, not mere barnyard contaminants. (12) We further showed that they are involved in the genesis of the disease by demonstrating a significant antibody response in affected cows compared to unaffected cows. In other studies, we have demonstrated the common presence of three additional bacteria. These bacteria are non-invasive, but their significance cannot be dismissed until further research rules in or out their co-pathogenic role.

In our early experimental studies, we found that footwarts were not transmissible, even to feet predisposed by trauma. (13) We also found that lesions may spontaneously shrink and disappear. Later experiments revealed that these observations are explained by drying of the skin and that transmission depends on exposing the feet to prolonged moisture and reduced access to air. (14) These findings prove again that development and persistence of footwarts depends on environmental as well as infectious factors. Because the experimental environmental factors simulated those found on high prevalence dairies (deep slurry), reducing the depth of slurry should result in decreased prevalence of the disease.

Further field research is needed to confirm this hypothesis.

The development of the calf transmission model has paved the way for experiments aimed at determining the precise cause (etiology) of footwarts as well as determining the protective effect of a vaccine. Recently, we demonstrated experimentally that *Treponema* spp. are primarily and predominantly involved in the development of footwarts. (15) Our pilot vaccine trial was recently completed. Results indicate that the vaccine did not protect against experimental challenge. Because footwarts is a complex disease and because it is difficult to develop effective vaccines against spirochetal diseases, we were not surprised. It must also be emphasized that before anyone can begin to be confident that a vaccine contains all causal bacteria, the disease needs to be recreated with pure cultures of these bacteria. This would be an appropriate future research objective.

## Conclusions

To summarize, we have established that footwarts is not a simple viral wart as previously thought, but a complex infectious disease caused by multiple bacterial agents working with and depending on environmental factors. We recommend that future research studies focus on: 1) factors influencing recurrence and spread; 2) significance of the non-spirochete bacteria involved; 3) virulence factors of the spirochetes; 4) recreation of the disease with isolated bacteria; and, 5) protective effect of vaccines.

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