Pathogenesis of ovine footrot disease: a complex picture

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FOOTROT (interdigital dermatitis [ID] and under-running footrot) is an infectious dermatitis of sheep feet that results in both poor welfare and production losses. Footrot is present in more than 90 per cent of flocks (Winter and others 2015) and accounts for approximately 70 per cent of lameness in sheep in England. The cost to the UK sheep industry is estimated between £24 million and £80 million per annum (Nieuwhof and Bishop 2005, Wassink and others 2010).

In 1941, Beveridge identified Dichelobacter nodosus as the causal agent of ovine footrot. Through experimental challenge he concluded that D nodosus alone was required for footrot to occur but the disease was more severe when other bacterial species including Fusobacterium necrophorum and spirochaetes were present. More recent research has confirmed that D nodosus has virulence factors (Type IV fimbriae and AprV2 protease) that are essential for the occurrence of footrot (Kennan and others 2001, 2010).

Later studies of disease pathogenesis reported that F necrophorum was the causal agent of ID, and that the presence of F necrophorum was required before colonisation with D nodosus could occur (Roberts and Egerton 1969). This led to the concept that ID, also known as scald, and under-running footrot were two separate disease conditions, with ID making sheep more susceptible to under-running footrot (Grogono-Thomas and Johnston 1997, Winter 2004). This concept was widely believed and stated in veterinary textbooks, expert articles and information provided to farmers (Winter 2004, Abbott and Lewis 2005).

The advent of molecular techniques has allowed further investigation of the roles of D nodosus and F necrophorum in the pathogenesis of ovine footrot. One such study is summarised on p 228 of this issue of Veterinary Record (Maboni and others 2016). In a cross-sectional study using postslaughter biopsy samples, Maboni and others (2016) demonstrate a significantly increased presence and load of D nodosus in feet with ID and under-running footrot.
D nodosus are likely to be the most infectious; therefore, rapid treatment of ID will minimise disease spread. Maboni and others also report increased prevalence and load of F necrophorum, but only in under-running footrot. This is similar to previous findings (Witcomb and others 2014, 2015, Frosth and others 2015). Results from these papers have slanted the evidence back towards Beveridge’s initial description of F necrophorum as an opportunistic pathogen rather than being required for disease initiation.

Most novel in the paper by Maboni and others is the two subspecies of F necrophorum – subspecies necrophorum and funduliforme. To our knowledge they present for the first time evidence that the subspecies necrophorum was more prevalent than the subspecies funduliforme in sheep in their study. The necrophorum subspecies is believed to be more pathogenic (Tan and others 1996) and this may therefore have relevance for disease pathogenesis or severity, although this is still unknown.

Maboni and others found no evidence to suggest involvement of Treponema species in the disease pathogenesis, although other bacteria including spirochaetes have been suggested to play a role in footrot (Beveridge 1941, Calvo-Bado and others 2011). The role of other species and the microbial community as a whole is not yet well understood, although the number of species detected in the microbiome of diseased feet is lower than in healthy feet (Calvo-Bado and others 2011).

Clinical importance for practitioners

- Under-running footrot and interdigital dermatitis (ID/scald) are presentations of one disease and these conditions must be managed together.
- Sheep with ID are probably the most infectious in a flock.
- This is a bacterial infectious disease, therefore rapid treatment, ideally with separation of sheep with ID or under-running footrot, reduces the spread of disease and is an essential tool for control.
- The most effective treatment is parenteral and topical antibacterials without foot trimming (Kaler and others 2010).

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New molecular techniques have allowed for further investigation of the roles of Dichelobacter nodosus and Fusobacterium necrophorum in the pathogenesis of ovine footrot.

Accounting for around 70 per cent of lameness in sheep in England, footrot is characterised by interdigital dermatitis and the separation of the skin and hoof horn (under-running footrot).

compared with healthy feet, similar to previous reports (Moore and others 2005, Calvo-Bado and others 2011, Witcomb and others 2014, 2015). These papers together provide evidence that ID and under-running footrot are two stages of the one disease process, namely footrot. This is clinically significant and highlights that both ID and under-running footrot should be treated and managed as one disease. Indeed, before this information was known, there was evidence for this in 2000 when farmers who treated both conditions promptly with parenteral and topical antibiotics had the lowest flock prevalence of lameness (Wassink and others 2003).

Maboni and others (2016) demonstrated in their study that the highest prevalence and load of D nodosus are on feet with ID, similar to the findings of Witcomb and others (2015). The authors highlight that the postslaughter, cross-sectional samples and the study by Witcomb and others (2015) do not provide evidence for disease progression. However, a longitudinal study (Witcomb and others 2014) had demonstrated that the load of D nodosus on feet increased significantly before the onset of ID and under-running footrot. Therefore, three studies indicate that D nodosus load is greatest on the skin of sheep with ID. Again this has relevance for disease management: sheep with ID with the highest load of D nodosus are likely to be the most infectious; therefore, rapid treatment of ID will minimise disease spread.

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Ovine footrot is a complex disease. A further challenge to our understanding of disease pathogenesis is variation in virulence within D nodosus. There are 10 serogroups of D nodosus that lead to 10 different host antigens with no cross protection; several serogroups can be present on a foot. D nodosus can be categorised as virulent or benign based on an amino acid sequence of

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an extracellular protease (Kennan and others 2014). Maboni and others (2016) demonstrate that the majority of strains in their study were virulent, similar to previous findings in Great Britain (Moore and others 2005) and Australia (Kennan and others 2014). The relationship between numbers of serogroups, genetic virulence and disease severity is not clear. Research into the interaction between D nodosus and F necrophorum, between different strains of D nodosus, and the microbial community as a whole is still needed to further our understanding of this complex disease and to inform flock-specific control programmes. These questions are being addressed through a grant currently funded by the Biotechnology and Biological Sciences Research Council’s Animal Health Research Club (ARC) (Animal Health Research Club 2012).

Maboni and others (2016) and Witcomb and others (2015) both report that D nodosus and F necrophorum are almost exclusively observed in the epidermis, at all stages of disease. This might partly explain the relatively low efficacy and duration of protection of existing vaccines, which use systemic antibody production rather than protection in the epidermis. A second ARC grant is investigating host inflammatory responses to D nodosus in the foot with the aim of rational vaccine design that would be more efficient at preventing footrot, both ID and under-running.

In conclusion, Maboni and others and other recent research using the latest laboratory techniques confirm that, as Beveridge concluded in 1941, D nodosus is the causative agent of ID and under-running footrot. The role of F necrophorum has been debated over the years but there is increasing evidence that it is an opportunistic secondary pathogen. Much else remains unknown and further research is necessary to understand the relationship between virulence of D nodosus and disease severity, and how this impacts on vaccine development and disease control.

References


Beveridge, W. I. B. (1941) Foot-rot in sheep: a transmissible disease due to infection with Fusiformis nodosus (n.sp.); studies on its cause, epidemiology and control. CSIRO Australian Bulletin 140, 1-56


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