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Review

Ovine footrot: New approaches to an old disease

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ABSTRACT

Footrot is a bacterial disease that has substantial economic and welfare impacts in sheep and can be difficult to manage. Research is focussed on reducing the impact that footrot has on farmers and their flocks and better understanding the aetiology of the disease. Key areas of current research include, developing better vaccines, deploying tailored vaccines in a specific and targeted fashion on individual farms, analysing and developing better farm management practices to suit specific sheep farming environments, elucidating the virulence genes and bacterial population dynamics that drive footrot and using genetic testing in combination with selective breeding to produce stock that are more resilient to disease.

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Footrot is a contagious hoof disease of sheep and other ungulates and begins as an interdigital dermatitis, which is followed by formation of lesions on the interdigital wall of the hoof and subsequent separation of the hard horn from the foot (called under-running). The essential transmissible agent of the disease is the bacterium *Dichelobacter nodosus* (*D. nodosus*), although the role of other infective agents in the onset of disease is not fully understood.

Historically, footrot was reported to have been prevalent within English sheep in the 18th century and in France it was recognised as a contagious disease by the 19th century (Stewart, 1989). It was also identified on sheep farms in the United States of America, Italy, Germany and Australia by the early 19th century. On some Australian sheep farms, the impact of footrot was severe, with many deaths being recorded (Stewart, 1989). We believe the seminal work "Footrot and Foot Abscess of Ruminants" (Egerton et al., 1989) remains the definitive history of footrot.

Footrot can result in poor feed intake, losses in production, a reduction in wool strength and in the worst

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cases; death from a combination of starvation, thirst and other systemic bacterial infections that occur in sheep that spend prolonged periods recumbent (Stewart, 1989). The cost of the disease can be substantial with estimates of £24 million per annum in the United Kingdom (Nieuwhof and Bishop, 2005) and Aus $42.6 million in New South Wales before a state-level footrot eradication plan was undertaken (Egerton et al., 2004).

Despite having been known about and researched for over 200 years, and with a comprehensive synopsis (Egerton et al., 1989) written about the disease, footrot remains a problem world-wide. However, there are several promising areas of footrot research and development that may provide new tools and approaches for better management or eradication of the disease. These include the development of specific vaccines, a markedly increased knowledge of the genetics of *D. nodosus*, the development and evaluation of new footrot management strategies customised to particular environments and the development of new genetic testing and selective breeding tools, which would create stock that are less likely to be infected and are less affected once they have footrot.

1. Footrot aetiology: an update

D. nodosus, the infectious agent that causes footrot, is a gram-negative, anaerobic bacterium (Beveridge, 1941). A second gram-negative anaerobic bacterium *Fusobacterium necrophorum* (*F. necrophorum*) is also required for *D. nodosus* to successfully initiate an infection in pen trials (Roberts and Egerton, 1969). These two bacteria are highly associated ($p < 0.001$) with footrot in the field (Bennett et al., 2009) and while *D. nodosus* may form multi-strain infections, Hill et al. (2010) have recently reported that the median number of serogroups per affected hoof is one, although it ranged from one to four. This observation is supported by the findings of Buller et al. (2010), although Zhou and Hickford (2000) report up to seven different *D. nodosus* strains on a single hoof. Zhou et al. (2009a) have reported that *F. necrophorum* tends to be found as a monoclonal infection, although only a small number of hooves were studied.

The complexity of the bacteriology of footrot lesions is further complicated by the genetics of the virulence of *D. nodosus*, which are intricate and potentially involve mobile genetic factors, including extra-chromosomally derived virulence islands with phage and plasmid-like forms (Cheetham et al., 2008). The bacterial complexity of the disease is further complicated by the effect of variation in temperature and rainfall on disease presentation (Graham and Egerton, 1968), by variation in host genetics (Emery et al., 1984), by variation in stocking rate (Stewart, 1989) and by the use of different farming practices such as hoof-trimming or paring (Wassink et al., 2003a).

Temperatures above 10 °C appear to be required for footrot transmission to occur, while consistent rainfall over several weeks seems to be required for a footrot outbreak, rather than a single heavy rainfall event that only lasts a short period of time (Graham and Egerton, 1968). It has been proposed that wet weather affects footrot susceptibility, either by inducing physical changes in the hoof that make it more vulnerable to attack (Graham and Egerton, 1968), or by changing the biology of the pathogens that cause footrot.

Despite being anaerobic, *D. nodosus* is able to survive on plates exposed to air for up to 10 days (Myers et al., 2007) and both footrot and specifically *D. nodosus* can be transmitted between stock via soil contact (Stewart, 1989). As a result of being able to be transmitted via soil contact, stocking rates are likely to affect how quickly *D. nodosus* is transmitted through a mob (Stewart, 1989; Wassink et al., 2003a). Footrot is also difficult to manage, since as well as *D. nodosus* being able to survive on and be transmitted via soil, it can also persist for months as a sub-clinical infection within the inter-digital skin, or in small cryptic lesions within the hoof (Stewart, 1989).

Footrot displays a wide range of virulence and the disease has been categorised as virulent, benign or intermediate (Stewart et al., 1986). The virulence of a specific outbreak is driven by how a specific population or populations of *D. nodosus* interact with the host and the various factors that affect those infections. Virulent footrot is characterised by destruction of the horn and typically involves erosion of the skin-horn junction that penetrates the hoof, causing de-lamination because of under-running. In contrast, benign footrot causes inflammation of the inter-digital skin with inter-digital dermatitis, but no under-running or de-lamination is observed (Stewart et al., 1986). While intermediate footrot can cause under-running in some cases, it is observed to be much less widespread and transmittable within a flock, even in favourable environmental conditions. It is diagnosed by the isolation and identification of *D. nodosus* strains with intermediate virulence factors (Stewart et al., 1986). While the environment affects the transmission of footrot, one study has proposed that there is no basis for suggesting that intermediate footrot diagnosed in an unfavourable environment will cause virulent footrot if the disease spreads to sheep in a more favourable environment (Abbott and Egerton, 2003a), and this suggests that virulence is still determined primarily by *D. nodosus* and/or any other transmissible agent that moves with it between sheep.

1.1. Towards a better understanding of D. nodosus virulence

The genome of *D. nodosus* has been sequenced recently (Myers et al., 2007) and this is a major step forward in understanding footrot biology, as well as providing a platform that should allow a better understanding of the behaviour and virulence of the bacterium. *D. nodosus* has a wide range of virulence with strains classified as virulent, benign, or intermediate (Stewart et al., 1986). The virulence of *D. nodosus* isolates can be tested in vitro by testing for the presence, activity and stability of key virulence factors such as proteases or fimbriae-mediated motility (Stewart et al., 1986). Other virulence tests have also been developed that detect genetic elements associated with virulence, such as *intA* (previously known as *vap*) and *vrl* (Cheetham et al., 2006).

Despite both *intA* and *vrl* being associated with virulence in *D. nodosus*, these elements do not encode