

Lameness in small ruminants

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Summary

The main causes of lameness in sheep include foot rot (FR), interdigital dermatitis (ID), and contagious ovine digital dermatitis (CODD). FR is a bacterial disease caused by *Dichelobacter nodosus*. An infection by faecal bacterium *Fusobacterium necrophorum* may develop as a result of injuries to the interdigital skin occurring over a prolonged period in a wet environment. FR is highly contagious and can be transmitted from sheep to sheep via pasture, bedding or handling pens; however, this disease can also be spread by sheep that do not show any clinical indications of the disease. In the case of ID infection, only the presence of *Dichelobacter nodosus* causes lameness. In most cases of CODD infection, *Treponema vincentii* has been isolated. The mean prevalence of lameness was around 8-10%, varied between years and depended on the climate and the standard of hygienic conditions. The main causes of lameness in goats were cracks and erosion on the horn of the bulbs of the heel that extended along the internal side of the axial hoof wall. Similarly as in sheep, *Dichelobacter nodosus* and *Fusobacterium necrophorum* were isolated from foot lesions. The clinical diagnosis was ID. The mean prevalence of lameness in goats ranged from 9% to 15%. The common detection of *Fusobacterium necrophorum* together with *Dichelobacter nodosus* supports the hypothesis that FR results from a synergistic interaction between these two organisms. Risk factors associated with infection and lameness in small ruminants are as follows: the wet season and moisture, smudge of dirt with mud of dens for animals, concentration of animals, virulence of the bacteria present, and the frequency of routine foot trimming. Particularly in sheep, an increased frequency of foot trimming is associated with an increased prevalence of FR. Lameness in small ruminants may also be related to an abnormal conformation of limbs or to lesions of the skin and udder. A highly reliable method for the evaluation of locomotion in small ruminants is the scoring scale using scores from 0 to 6. The treatment of infected animals consists primarily of their separation from the flock and the application of an antibacterial therapy, in which almost any topical antibiotic and foot spray can be effective. During transmission periods it is advisable to bathe animals' feet in zinc sulphate (10 or 15%) or formalin (3%) every five days. Supplemental dietary biotin at 5.25 mg/day healed hoof lesions within 7 months. An improved locomotion of sheep was visible within 4 months. Vaccination plays a valuable role, but it is not fully effective, and immunity is of relatively short duration. Vaccination should be repeated at six-month intervals. Prevention and control of the two most common causes of lameness in small ruminants (foot rot and digital dermatitis) that eliminate *Dichelobacter nodosus* and *Fusobacterium necrophorum* are more feasible given the climate and environment can lead to minimization of lameness, improvement of animal welfare and increased productivity.

Keywords: small ruminants, lameness, foot rot, interdigital dermatitis, contagious ovine digital dermatitis, treatment

Foot rot (FR) and interdigital dermatitis (ID) are the two most common causes of lameness in sheep, with foot lesions causing approximately 80% of lameness cases (26, 27, 42). Many authors are of the opinion that contagious ovine digital dermatitis (CODD) also causes sheep lameness (42, 44, 49). The clinical presentation of FR is the separation of the hoof horn from the sensitive tissue of the claw with a fetid grey pus (42, 47). The clinical image of ID is an inflammation of the interdigital space with red, moist interdigital skin and a characteristic white or grey pasty exudate (47).

First named „severe virulent footrot,” CODD refers to a disease in which the spirochaetes isolated are closely related to those isolated in bovine digital dermatitis, except for the absence of *Dichelobacter nodosus* (47).

The mean prevalence of FR and ID ranged from 3.1% to 9.4%, and from 6.6% to 8.2%, respectively (25, 42). The mean daily prevalence of ID in lambs was 15.6% with a large peak in its prevalence in late spring and early summer (42). Over 90% of sheep farmers in the United Kingdom report lameness in their flocks at a rate of 8-10% (25). The prevalence rate of

lameness in sheep in Nigeria was 17.89% (7). Transitory lameness in Makui sheep, amounting to 17% among lambs of less than 4 months of age and 3.5% among adult sheep, was associated with the only ticks (*Hyalomma* spp.) that had been detected on all animals examined (2). This disease causes a considerable deterioration in the welfare of animals and has economic consequences (17, 18, 36, 37, 49).

Clinical lameness in goats is a result of the presence of ulcers with or without granulomatous tissues in the interdigital skin and sole regions. Granulomas appear as vascular nodules within or protruding from the sole. The nodules are haemorrhagic and up to 1 cm in diameter. These lesions are also associated with necrotic soles and with a distinct and characteristic odor (14). A digital disease in goats results in their poor productivity and subsequent economic losses to the farm. The prevalence of lameness in successive years varies, depending on the climate and hygienic conditions. In the control of this disease it is important to improve management systems on farms (38). Christodouloupoulos (11) reported that 15% of goats showed lameness with foot lesions, and 24% of goats showed lameness without foot lesions. The foot lesions were cracks and erosion on the horn of the bulbs of the heel. The clinical diagnosis was ID. In British dairy goats the mean prevalence of lameness was 9.1% (22).

Clinical lameness of small ruminants causes adverse changes in the welfare of individual animals or the flock, and health is an integral part of the welfare of animals (5, 9, 11, 31, 39, 49). An extensive environment is more congenial to sheep and more conducive to their welfare compared with intensive systems (15). Lameness also has economic consequences for the productivity of lame animals (11, 18). For example, the most prevalent cause of lameness in sheep in Great Britain led to economic losses of £ 24 million annually (£1 = approx. €1.19, as of January 2011) (37). The cost of each individual case of lameness was estimated at £1.32 per ewe and £0.15 per lamb (37, 45).

This study presents the pathogenesis and prevalence of foot diseases, risk factors associated with infection, the assessment of locomotion, and treatment methods in small ruminants.

Pathogenesis, clinical signs and prevalence of foot diseases

Foot rot is a bacterial disease caused by *Dichelobacter nodosus*. It is a common cause of lameness in lambs and mature sheep (4, 5, 10). The accepted pathogenesis for the disease is as follows: when the interdigital skin of the foot is damaged or wet for a prolonged period, it may be infected by a faecal bacterium *Fusobacterium necrophorum* (4, 23). In isolation, *Fusobacterium necrophorum* may cause inflammation of the interdigital skin and produce toxins that cause necrosis of the surface of the interdigital skin, facilitating entry for other bacteria, including

Dichelobacter nodosus (4). Foot rot is highly contagious and can be transmitted from sheep to sheep via pasture, bedding or handling pens; however, this disease may also be spread by sheep that do not show clinical signs of disease (5). *Fusobacterium necrophorum* can be merely an opportunistic infection that colonizes sheep's feet infected by FR. There are suggestions that *Fusobacterium necrophorum* may be transmitted to and from the mouth of sheep to the paddock, although the manner of the transmission has not been thoroughly clarified to date (4). Zhou et al. (50) describe the detection of the *Fusobacterium necrophorum* lktA gene on hoof samples taken from FR infected sheep, goats and cattle, and the identification of four lktA sequences. This suggests that *Fusobacterium necrophorum* is frequently, but not always, present on the hooves of lame animals and that it is genetically diverse. The widespread detection of *Fusobacterium necrophorum* together with *Dichelobacter nodosus* supports the hypothesis that FR results from a synergistic interaction between these two organisms (4, 23). In goats, especially those exposed to wet bedding, *Fusobacterium necrophorum* causes erosion starting in the bulbs of the heel and expanding along the internal side of the axial hoof wall (11). Although *Dichelobacter nodosus* has been isolated from only one foot with lesions, it may be involved in horn detachment and, ultimately, in the development of foot lesions. Lesions, as well as horn separation, abscesses of the sole and FR, are significantly associated with lameness, and they are related to environmental rather than genetic or nutritional factors (22).

Approximately 80% of lameness cases in sheep in the United Kingdom are attributed to *Dichelobacter nodosus* and *Fusobacterium necrophorum* (23), and in Great Britain up to 86% of farms are affected by FR (42). A high prevalence of CODD was also observed in infected flocks (about 25% in ewes and 15% in lambs), and in some cases *Dichelobacter nodosus* was also isolated (23). In sheep suffering from CODD, treponemas were found in 70% of cases, and in many cases *Dichelobacter nodosus* was also present (35). This bacterium was isolated from all ten flocks in which FR was suspected, from all six flocks in which only clinical signs of CODD were observed, and from three out of four flocks in which both diseases were observed. A spirochaete isolated from CODD is *Treponema vincentii* (42). *Dichelobacter nodosus* was isolated from affected sheep on 124 farms in Australia, between the years 2000 and 2005. The FR type on each farm was classified on the basis of gelatin gel tests and a field clinical diagnosis as *stable benign*, *virulent* or *unstable benign*, and 37%, 41% and 22% of farms, respectively, were classified into the above mentioned types (10).

There are 19 serotypes of *Dichelobacter nodosus*, which are classified into 10 major serogroups (A-I, M), and several of these serotypes are usually present in

sheep on farms in Great Britain (34). Not all serotypes are found in individual countries. In Bhutan only one serogroup (B) has been identified so far in sheep with FR; only this serogroup of *Dichelobacter nodosus* was identified in 40 isolates cultured from affected sheep (20). The use of new cultural techniques, the polymerase chain reaction (PCR), protease zymogram and serogruping have shown a much more ambiguous picture, since *Dichelobacter nodosus* has been isolated from sheep with ID, FR, and CODD, as well as from apparently healthy sheep in the same flocks (34). The PCR methodology in evaluated sampling strategies identified the two most common serogroups of *Dichelobacter nodosus* in the flock, and it was used to eliminate those serogroups from infected flocks through vaccination targeted at up to two specific serogroups (21). However, none of the evaluated sampling strategies provided expected results. The virulence of *Dichelobacter nodosus* varies between and within serotypes. Virulence is positively correlated with the number of fimbriae on *Dichelobacter nodosus*, and these are associated with the filmA gene, which encodes a fimbrial subunit protein (30). The results presented by these authors indicate that the fimbrial subunit gene is essential for the virulence of *Dichelobacter nodosus* in sheep. *Dichelobacter nodosus* strains have been classified as virulent, moderate and benign. Protease production enables penetration into deeper tissues of the foot. The greater the quantity or strength of the protease, the deeper the penetration into the tissue and the faster the occurrence of clinical signs (30).

Originally, sheep infected with ID were classified as non-lame, but the current definition of interdigital dermatitis includes severe lameness in sheep (47, 49). In the case of ID infection, only the presence of *Dichelobacter nodosus* causes lameness. This bacterium is certainly present in many feet with signs of ID (34). Interdigital lesions were the most common causes of locomotor disorders (73.1%), followed by disorders of the hoof (13.5%), and painful disorders of joints (6.2%) (12). Some studies indicate a knowledge gap between sheep advisors and sheep farmers with regard to the naming of the six most common foot lesions in sheep. About 20% of farmers named all the six lesions correctly, but a majority recognized only ID or FR, while approximately 80% of advisors recognized all the lesions. Often FR was the name given to the other common hoof lesions (26).

Risk factors associated with infection and lameness

Most causes of lameness are observed during the wet season (especially in sheep). Reducing the level of moisture in the environment in which animals live may reduce the incidence of lameness (16). The rapidity with which other animals are infected depends on moisture, muddiness of the ground, concentration of animals, and virulence of the bacteria present (42). Often seasonality is associated with FR outbreaks,

usually during times of higher rainfall and warm weather. In a questionnaire survey, 47% of farmers reported that infection was attributable to the weather, 40% claimed that the manner of foot infection was „ground-to-sheep transmission,” 26% stated that this infection depended on the susceptibility of sheep, 26% reported that the manner of infection was „sheep-to-sheep transmission,” and 13% attributed infection to „interdigital dermatitis”. (45). Similarly, the number of lame goats shows a significant seasonal variation (11). The first case of lameness was recorded in December, and the maximum occurrence was found in April, whereas no cases were observed between July and November. Other factors influencing the spread of FR include management that is the housing system, reported by 12% of farmers, „stocking density,” reported by 11%, and the lack of proper control, reported by 7% (42, 45).

It is very interesting that the prevalence of FR in ewe flocks increased with an increased frequency of routine foot trimming. The number of cases per 100 ewes per day was 6.7 in flocks that trimmed ewes „never or once” per year, 11.2 in flocks that trimmed „twice” per year, and 15.6 in flocks that trimmed „more than twice” per year (42). In another study (41) the same team of authors reported that the relationship between a high prevalence of FR and routine foot trimming may have occurred either because routine foot trimming increased the occurrence of FR, or because farmers trimmed feet more often when the prevalence of FR was high. Similarly, other authors report that foot trimming in sheep is associated with an increased risk of FR/ID; however, the use of parenteral antibacterials is associated with a reduced risk (19, 25). Risk factors associated with the occurrence of lameness in Saanen-cross goats include wet bedding and overgrown hooves (11). The frequency of routine foot trimming in adult British dairy goats on four farms showed an inverse relationship between the frequency of foot trimming and the prevalence of lameness (22). Overgrown horn on at least one foot was observed in 83.1-95.5% of the goats.

Factors affecting sheep lameness included lesions of the skin and udder, abnormal gait of all limbs, weakness of both hind limbs, weakness of both fore limbs, and an abnormal conformation of limbs (12). A poor foot conformation is a risk factor for an increased incidence of FR and further lameness. A good or poor foot conformation is a result of environmental conditions (6, 7, 28). Management factors, as well as the frequency of foot bathing, separation of lame sheep at pasture, and stocking density also affect the prevalence of ID and FR (25).

Differences between breed types (Dorset, 1/2 Dorper, 3/4 or greater Dorper, Gulf Coast Native, Katahdin, and St. Croix) in their response to the treatment of virulent FR are minimal. Animals exposed to FR for a shorter period of time are less likely to be culled than

animals exposed to FR for a longer time. Lambs respond to treatment better than mature sheep (8). Bishop and Morris (5) showed that phenotypic assessment combined with genetic markers for FR resistance would be advisable in the control of this disease. An estimation of the prevalence of shelly hoof in Blackface and Texel sheep showed that shelly hoof has a high prevalence in over 9,000 sheep of these breeds (47% for Blackface and 24% for Texel ewes) and is under moderate genetic control ($h^2 = 0.3$). The results showed a genetic basis for a poor horn structure leading to the expression of shelly hoof. This problem can be solved in the long term through genetic selection. It seems possible to improve the hoof horn quality with nutritional supplements. Productivity benefits are likely to occur in a synergistic way together with selection (13).

The assessment of locomotion

The development of a numerical rating scale (NRS) to assess locomotion in sheep started in 1989, when categories from 0 to 4 were used (0 = normal movement, 1 = occasional limping, 2 = lifting foot when standing, not lame when moving, 3 = carrying foot, but lame on movement, and 4 = carrying foot at all times), but observer agreement was not assessed (25). Another NRS with inter- and intra-observer agreement was drawn up by Welsh et al. (46), who also used a scale from 0 to 4 (0 = clinically sound, 1 = barely detectable lameness, 2 = obvious lameness, 3 = severe head nod and possibly resting the affected foot when standing, and 4 = carrying foot at the trot). This scale, however, contains subjective phrases, e.g. „obvious” lameness. The scoring scale presented by Kaler et al. (29), which uses categories from 0 to 6 (0 = bears weight evenly on all four feet, 6 = will not stand or move), is objective and based on a group of visual observations. It is a highly reliable method for the evaluation of locomotion in sheep. Genetic studies on the resistance to foot rot have resulted in the development and evaluation of a five point scoring system (49). This system comprises the following scores: „0” (normal hoof, no lesion), „1” (mild ID), „2” (more extensive ID and necrotising inflammation of interdigital skin), „3” (severe ID and under-running of the horn of the heel and sole), „4” (severe ID and under running of the horn of the heel, sole and walls of the hoof). Clinical examination of sheep requires regularity and no detail can be omitted. Many different scoring systems can be used in clinical practice for the assessment of different degrees of lameness in sheep (33).

Treatment and control methods

Treatment of FR cases should be aimed at eliminating the infection from individual animals and preventing its spread to other sheep in the flock (24, 25, 42). The initial step in the treatment is to open the infected area and to correct the foot trim; however, trimming should only be used for diagnostic purposes and to

remove obviously loose horn (48). Although routine foot trimming more than once a year has been associated with a significantly higher prevalence of infection (42), Abbott et al. (1) reported that the higher prevalence could have been a reason for more frequent trimming, rather than a causal factor. Hence, it needs to be recognized that dirty, badly maintained permanent pens with muddy approaches are likely to create more problems than they solve. Once the area is open for drainage, almost any topical antibiotic or foot spray can be effective (28, 42, 43). Antibacterial therapy leads to recovery from lameness and reduces the risk of poor foot conformation (28). For goats it is recommended to add dry straw bedding, disinfect the bedding material with limestone powder, perform conservative foot trimming more frequently, and disinfect tools in 10% formalin after each foot is trimmed (11). In cases of severe foot lesions, a topical application of an ointment preparation (40 g vaseline, 40 g lanoline, and 10 g balsam of Peru) and a bandage is recommended. Minor foot lesions may be treated with oxytetracycline/gentian violet aerosol spray. This treatment leads to a reduction of lameness within 1 day and full recovery in 2-5 days (11).

In order to minimize lameness in sheep, farmers should isolate mildly lame sheep in a group within 1-3 days of their first being lame (27). The proposed management of lame animals limits the mean prevalence of lameness to < 5% (40). The researchers propose regular footbathing every five days with zinc sulphate (10 or 15%) or formalin (3%) during transmission periods (24, 48). However, they state that foot bathing is effective only if the handling equipment is of high quality, and the sheep can stand on a hard surface (concrete or stones). Zinc supplementation for sheep affected by FR and control sheep at approximately 9 mg zinc/kg live weight (2 g $SO_4Zn \cdot 7H_2O$) per animal twice weekly did not bring expected benefits (32). There was no difference in the foot health over time (from October to February) between zinc-supplemented and control groups. A beneficial effect of zinc administration for the control of FR could be related to a low zinc status in animals (between 12.95 and 14.84 $\mu\text{mol/l}$). Vaccination plays a valuable role, especially when the main transmission period is not known and may vary between years (24). Vaccination of sheep in Australia and in the United Kingdom has had beneficial effects; however, the preventive is not fully effective and the immunity is of relatively short duration (48). Vaccination needs to be repeated at six-month intervals. Hoof disorders, as well as sole ulcers, sole hemorrhage and heel erosion, in sheep are responsive to biotin supplementation (3). Supplemental dietary biotin at 5.25 mg/day healed hoof lesions within 7 months. An improved locomotion of sheep was visible within 4 months. Sheep and lambs affected by transitory lameness caused by ticks underwent treatment with a tick disinfectant by the external applica-

tion of 2 ml/l of amitraz in water (Mactak[®], Keshavarz Chemical Company, Iran) (2). Sheep were completely recovered after 2 days of acaricide treatment.

In conclusion, prevention and control of the two most common causes of lameness in small ruminants (foot rot and digital dermatitis) rather than the elimination of *Dichelobacter nodosus* and *Fusobacterium necrophorum* are more feasible given the climate and environment can lead to a minimization of lameness, improvement of animal welfare and an increase in productivity.

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