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## BRIEF COMMUNICATION: An investigation of mastitis in a hill-country sheep flock

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### Introduction

The prevalence of mastitis in New Zealand ewes has not been studied for over 40 years. Quinlivan (1968a,b) examined 19427 Romney ewes in the lower North Island and detected mastitis in 0.6-1.7%. Clarke (1972) surveyed 118249 ewes in the lower South Island and found the incidence of mastitis was 0.6-0.7%. Skyrme (1970) reported the occurrence of “hard udders” and implicated them in lamb deaths. Perhaps because of such reports of low incidence, little research into sheep mastitis has been done in New Zealand.

During 30 years of milking sheep, about 5% of ewes (n~700) presented after lambing with a hard udder (Peterson unpublished observations), the cause of which was unknown until Bruce et al. (2013) studied 13 ewes with clinical signs of hard udder and found occluded teats and ducts due to chronic progressive inflammation in response to bacterial infection (agent unidentified) together with obliteration of lactiferous sinuses (sometimes including the teat canal) and scar tissue formation.

Ekdahl (1972) reported that bacteria implicated in mastitis in New Zealand ewes were *Staphylococcus aureus*, *Pasteurella (Mannheimia) haemolytica*, *Escherichia coli*, *Corynebacterium (Trueperella) pyogenes*, and various unidentified streptococci, bacilli and mycoplasmas, but that the primary casual organism was undoubtedly *S. aureus*.

Lack of knowledge regarding mastitis in New Zealand sheep led to this preliminary study of hill-country ewes. The objectives were to determine the prevalence of mastitis and whether different forms of mastitis could be detected by palpation after drying off. Subsequent post-mortem analyses were performed to determine the bacteria involved.

### Materials and methods

Udders of the Romney flock (n=1824) at Tuapaka farm, Palmerston North, were each examined once, beginning three weeks after weaning during the period 13 Jan-10 Feb 2016. Ewes ranging in age from 2.5-8.5 years (mean 4.8 years) were inverted in a sheep-handling device (Hecton Products, Invercargill) whilst the udder was palpated. The presence of a hard core in the teat canal, possibly suggestive of “hard udder”, or pus in the teat canal, the number and position of any lumps in the glands, externally visible abscesses, and other lesions, were recorded. Where possible, secretion was expressed from the gland and examined visually for lumps or pus.

The diagnosis of mastitis or abscessation of the udder was based upon any of the above signs and ewes diagnosed were marked for culling.

Four months after weaning (2-3 months after diagnosis) 57 ewes, selected at random, were slaughtered, udders removed and packed in ice. Gross examination and sampling of the udders was carried out the same day.

#### *Post-mortem examinations*

Cranial and caudal borders and left and right udder halves were palpated for consistency and presence of lumps. Teats were palpated for presence of a hard core within the teat sinus. Each teat was cut transversely approximately 10 mm from the tip; the teat sinuses on the cut surface were examined and the nature of any content was recorded. A stab incision using a sterile scalpel blade was made into each half of the udder. Lumps were incised and swabbed for microbial sampling. The nature of any secretion or exudate was recorded.

#### *Microbiological studies*

Swabs were immersed in sterile saline to dilute the bacteria and 10 µL spread on a 5% blood agar plate to obtain isolated colonies. Plates were incubated aerobically at 37°C for 48 h. Plates were observed for bacterial growth and single colonies picked and Gram-stained. Gram-positive cocci that were catalase positive were further processed using a *S. aureus* species-specific latex agglutination test. Other bacteria were not further analysed.

### Results

Of the 1824 ewes, 91 (5%) had clinical mastitis or hard udder, and 11 (0.6%) of the remaining ewes had other udder defects ranging from enlarged udders to missing teats (Table 1). Eighteen ewes had hard glands and a hard core within the teat canal preventing milk removal. The average age of the 102 ewes with udder defects was 6.15 years (median 7 years).

Of the 57 udders examined post-mortem, 23 had one or more abscesses in one mamma, while three had abscesses in both. Unaffected mammae appeared to be involuting normally; secretions varied from watery pale brown to dark brown and sometimes slightly opaque and flocculent. In three udders, healed scars from ruptured abscesses were present. One or both teats of 17 ewes had palpably thickened cores, and in nine of these, the teat cisterns contained pus. The gland cisterns and major

**Table 1** Numbers and proportions of 1824 weaned hill-county ewes at Tuapaka farm exhibiting udder defects. Potential mastitis was diagnosed by palpation of lumps in the gland, observation of abscesses, or observation of pus in the teat or secretion. Other defects (observed in 11 ewes) included cuts and bites or missing glands and teats.

Udder defects and potential mastitis	Number of cases	Proportion of total sheep	Proportion of total udder defects	Proportion of 91 cases of potential mastitis
Left half only	45	2.5%	44.1%	49.5%
Right half only	34	1.8%	33%	37%
Both halves	12	0.7%	12%	13%
Other defects	11	0.6%	10.8%	-
Total	102	5.6%	100%	100%

lactiferous ducts of three ewes contained large amounts of liquid pus. Pus varied from creamy-soft and pale green to pale tan brown and inspissated.

Samples revealed a consortium of organisms; of 41 ewes, 11 had *S. aureus*, two had *T. (Arcanobacterium) pyogenes* and 22 had mixed taxa.

## Discussion

The prevalence of 5% of ewes with clinical mastitis is higher than the average values of <1% reported earlier (Quinlivan 1968a,b; Clarke 1972) but agrees with the value of 4.1% on one farm reported by Clarke (1972) and with the results of Barber et al. (2016), who reported the incidence of mastitis in Australia to be 1-2% in Merinos, and 3-5% in meat breeds. Menzies (2000) reported widely differing incidences around the world and Cooper et al. (2015) reported clinical mastitis in less than 2% of ewes on 329 farms in England, but the incidence was highly variable among farms. Quinlivan (1968a) reported no difference in susceptibility to mastitis in ewes of different ages while our results, in a much smaller sample, are consistent with higher incidence of mastitis in older ewes. Thus, the incidence of mastitis at Tuapaka farm may be influenced by the particular environment and may not reflect the prevalence in the general New Zealand sheep population.

At post-mortem analysis, hard udder could no longer be identified, probably due to the long intervening period between diagnosis and slaughter. Since hard udder had been detected early in lactation in 5% of ewes (in unrelated trials; Peterson unpublished), but was found in only 0.6% of ewes 1-2 months after weaning and was undetectable at culling 4-5 months after weaning, it is apparent that the diagnosis of hard udder cannot be made at culling time alone. Since the severity of infection changes with stage of lactation, with more of the acute forms occurring shortly after lambing, and more of the chronic forms post-weaning (Quinlivan 1968b), it is necessary to examine ewes at different stages of their lactation cycle to correctly diagnose mastitis.

Pathogens causing mastitis have been studied in many countries in both meat and dairy sheep. *Staphylococcus aureus* (contagious) is the bacterium most frequently responsible for clinical mastitis (20-60%) whilst coagulase-negative staphylococci (environmental) are the principal causative agents of subclinical mastitis (30-95%)

(Bergonier & Berthelot 2003). Gelasakis et al. (2015) noted that internationally the most common mastitis bacteria strain is *S. aureus*, followed by other *Staphylococcus* spp., *Mannheimia* spp., *Streptococcus* spp., as well as a range of other bacterial species. The results of the current study are in line with these overseas findings and with the earlier work of Ekdahl (1972) in New Zealand.

Udders are usually examined briefly once each year when culling decisions are made. Results of this investigation indicate that farmers should examine ewes more closely and more frequently, to detect mastitis, especially hard udder, which is not visible and must be palpated. Further studies are required to determine incidence and severity of ovine mastitis in New Zealand, to confirm the organisms involved, and to identify appropriate times at which ewes should be examined to detect various types of mastitis.

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